Renal Failure: Its Treatment in Current Systems of Medicines

Dr. Mansoor Ahmad
Dr. Farah Saeed
Dr. Mehjabeen
Dr. Noor Jahan

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RENAL FAILURE: ITS TREATMENT IN CURRENT SYSTEMS OF MEDICINES
(A book for Nephrologists, Pharmacists, Doctors, Technologists, Paramedics, Health care providers and Alternative Medicines Practitioners)

Edited by

Dr. Mansoor Ahmad
M.Sc. DHMS, M.D., D. Sc. (ETH- Zurich, Swiss)
Professor of Pharmacy, University of Karachi

Dr. Farah Saeed
B. Pharm, M. Phil. Ph.D.
Department of Pharmacognosy,
Dow College of Pharmacy,
Dow University of Health Sciences, Karachi

Dr. Mehjabeen
B. Pharm, M. Phil. Ph.D.
Department of Pharmacology, Faculty of Pharmacy,
Federal Urdu University of Arts, Science & Technology, Karachi

Dr. Noor Jahan
B. Pharm, M. Phil. Ph.D.
Department of Pharmacology,
Dow College of Pharmacy,
Dow University of Health Sciences, Karachi

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PREFACE

Renal failure is a pathology which is growing all over the world and which affects other organs of the body, apart from kidneys. Renal failure refers to temporary or permanent damage to the kidneys that result in loss of normal kidney function. There are two different types of renal failure - acute and chronic. Acute renal failure has an abrupt onset and is potentially reversible. Chronic failure progresses slowly over at least three months and can lead to permanent renal failure. The causes, symptoms, treatments, and outcomes of acute and chronic are different. Most of the causes of renal disease are due to poor diet and lifestyle. The fatal diseases like hypertension and diabetes are the major causes of the renal failure. The problem with renal failure is that it doesn’t become apparent until the condition is well established and you have already lost part of your kidney function.

The best treatment for renal failure is to avoid kidney problems in the first place. Kidney failure is treated with a special diet, medicines, regular dialysis treatments and, possibly, a kidney transplant. Dialysis treatment can extend their life. However dialysis is not a cure-all. The treatment only partly replaces some functions of the kidney. It does not benefit other health problems and indeed may make some of them worse. Botanical, Homeopathic and Unani medicines could produce tremendous benefit in the patient with renal impairment/failure, if used judiciously, and in conjunction with allopathic medicines.

Renal Failure – Its treatment in current system of medicine is edited to provide a comprehensive and systematic review of the latest available information concerning pathophysiology, epidemiology, clinical presentation, diagnosis and treatment in different systems of medicine for the acute and chronic renal failure.

This book has been edited to elaborate the importance and need of collaboration of nephrologists, naturopaths, pharmacologists, pharmacognosists, herbalists, homeopaths, and other traditional systems practitioner to implement integrated
system of medicine for the treatment and improving the quality of life of renal failure patients and to control the progression of kidney damage and the need of patients for dialysis and transplantation for renal failure.

It is our hope that the readers will become acquainted with the latest researches being carried out in the field of nephrology for the treatment of renal failure in different systems of medicine. Thus the book is designed to be both a reference source and a practical guide to the clinical management of renal failure. The text should prove useful and valuable to clinicians, educators and investigators alike.
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CHAPTER 1
CHAPTER 1

INTRODUCTION

The Kidneys Physiological Principles

Kidney plays an important part in keeping our body healthy. To understand the effects of kidney disease, it is important to first understand the vital functions which the kidney performs, all of which are required for metabolic homeostasis.

These essential for life functions include 1-2
1. Removal of water, urea, creatinine, and other metabolic wastes and toxins from the blood.
2. Regulation of volume, composition, and pH of the body fluids.
3. Regulation of blood pressure.
4. Synthesis of erythropoietin, a hormone involved with red blood cell production.
5. Conversion of vitamin D to its active form, 1, 25 di-hydroxy vitamin D, required for calcium absorption and bone health 1-2

Diabetes with chronic hyperglycemia and its frequent comorbidities hypertension, anemia, hyperlipidemia, bone disease; fluid and electrolyte imbalances, and the buildup of metabolic wastes and toxins, damages the kidney's structural integrity which impairs its functional capacity 1-2.

The most common problems with renal system arise from:

a. Infection
b. Renal obstruction
c. Renal failure
d. Bladder infection

A prompt diagnosis and appropriate therapy are fundamental to prevent or eliminate glomerulonephritis,
pyelonephritis, hydronephrosis, renal failure and bladder infection.

The kidney is both structurally and functionally complex, and plays a central role in homeostasis. There are many possible varieties of renal malfunction which may cause a wide range of clinical conditions. Manifestations of renal disorder include fluid, electrolyte and pH imbalance, hemodynamic imbalance, the accumulation of drugs, toxins and waste metabolic products, loss of essential metabolites, and endocrine abnormalities such as anemia and bone disease.

Structural damage can occur to the glomeruli, the tubules or the urinary tract, such damage resulting from pathological processes such as infection, inflammation, auto-immunity, neoplasia and toxins. Systemic or local circulatory insufficiency can also seriously damage renal function. The most common pathologies are glomerular inflammation, urinary tract infection and drug-induced nephrotoxicity.

Anatomy of Kidney

The kidney is structurally complex and consists mainly of three main regions: the cortex, the medulla, and the pelvis. The cortex contains the glomeruli and the proximal and distal tubules, and the medulla contains the loop of Henle. Glomeruli in different areas have different-length loops of Henle to permit differential control over urine concentration. The loops of the juxta medullary nephrons, nearest the medulla, extend almost to the pelvis, the area in which urine drains from the collecting ducts. Throughout the kidneys there are interstitial cells, probably concerned with endocrine functions.

The kidney is the key organ of overall homeostatic control. Elimination of waste is normally first associated with the kidneys, but their regulatory functions are equally important. The kidneys also are involved in several major endocrine systems.
Types of Kidney Failure

Kidney failure can occur from an acute situation or from chronic problems.

**Two Types of Kidney Failure**

**“ACUTE”**
Kidney failure develops within hours or days, includes chance of kidney function recovery

**Possible causes, e.g.:**
- traumatic (e.g. post surgical)
- acute intoxications
- part of multiorgan failure
- various other diseases (e.g. infections)

**“CHRONIC”**
Kidney failure develops over years, irreversible at the end

**Possible causes, e.g.:**
- secondary to high blood pressure and / or diabetes
- chronic bacterial inflammation of the kidneys
- cystic kidneys
- various autoimmune diseases

Statistics of Incidence, Prevalence and Treatment Strategies of Renal Failure

Chronic kidney disease is a worldwide public health problem, a social calamity and an economic catastrophe. In the year 2000; in US alone 30 million people were diagnosed with chronic kidney disease.

Renal failure is a pathology which is growing all over the world and which affects other organs of the body, apart from kidneys.

More than 26 million Americans have kidney disease or are at risk of developing kidney disease and around 50,000 people die each year due to kidney failure. The number of kidney failure patients is growing at a rate of 6% a year in America.

According to global overview of end-stage renal disease patients, conducted in 2004, about 1,783,000 of the population were suffering from ESRD; 77% were on dialysis treatment and 23% had undergone renal transplantation.

With reference to research conducted in 2004, of the, 1,371,000 patients on dialysis 89% were treated by hemodialysis and 11% had undergone peritoneal dialysis treatment.

Millions of people are affected annually by non-fatal kidney diseases, most notably infections of the kidney or lower urinary tract, kidney stones, and urinary obstruction. 20% of all women suffer from infection of the urinary tract or kidneys at some time.
in their lives, as many as 5% of the US population develop renal stones.

More than 35% of people aged 20yrs or older with diabetes have chronic kidney disease. More than 20% of people aged 20 years or older with hypertension have chronic kidney disease. While chronic kidney disease is more common among women, men with chronic kidney disease are 50% more likely than women to progress to kidney failure. Leading cause of end-stage renal disease are diabetes and hypertension. In 2006, 7 out of 10 new cases of end-stage renal disease in the United States had diabetes or hypertension listed as the primary cause. Less common causes include glomerulonephritis, hereditary kidney disease, and malignancies such as myeloma. Incidence of end-stage renal disease is greater among adults older than 65 years.

It is estimated that by 2030, more than 2 million people in the United States will need dialysis or transplantation for kidney failure. Currently approximately 19 million adults in the United States are in the early stages of the disease, defined by a GFR of less than 60ml/min per 1.73m² of body surface area or the presence of kidney damage, regardless of the cause, for three or more months. Risk factors for chronic kidney disease include an age of more than 60 years, hypertension, diabetes, cardiovascular disease, and a family history of the disease.

Statistics of Causes of Death in Patients on Dialysis

Cardiac disease accounts for half of all deaths in End stage renal disease patients. Infections (usually septicemia) are the next major cause (25%) and cerebrovascular disease is the third largest cause of death (6%). Figures vary slightly across countries and regions. In the UK 35% patients die from cardiac
Introduction

disease, 20% from infection, 13% from stopping dialysis, 9% from malignancies, and 7% from cerebrovascular disease. In Australia 46% of all deaths are from cardiac causes, 12% infection, 10% vascular disease, and 21% from dialysis withdrawal or cessation. Death rate in general is higher in Caucasian patients than African Americans. Other factors associated with early death are increasing age, diabetes, cardiac failure, poor functional status, ischemic heart disease, and cancer. In the US withdrawal from dialysis is a common cause of death (up to 20% of all dialysis patients), because of failure to thrive or medical complications. Withdrawal is common in older, Caucasian patients. Withdrawal rates are lower in Europe, possibly because of the initial acceptance of patients with marginal benefit from dialysis in the US.

References
CHAPTER 2

Blood sample taken

24-hour urine sample collected

Serum creatinine levels are used to measure glomerular filtration rate
CHAPTER 2

MEDICAL TESTS FOR THE DETECTION OF KIDNEY DISEASE

Introduction
A person can have kidney disease without any symptoms; therefore, a doctor may first detect the condition through routine blood and urine tests. The National Kidney Foundation recommends three simple tests to screen for kidney disease: a blood pressure measurement, a spot check for protein or albumin in the urine, and a calculation of glomerular filtration rate (GFR) based on a serum creatinine measurement. Measuring urea nitrogen in the blood provides additional information.

Blood Pressure Measurement
High blood pressure can lead to kidney disease. It can also be a sign that the kidneys are already impaired. The only way to know whether a person’s blood pressure is high is to have a health professional measure it with a blood pressure cuff. The result is expressed as two numbers. The top number, which is called the systolic pressure, represents the pressure in the blood vessels when the heart is beating. The bottom number, which is called the diastolic pressure, shows the pressure when the heart is resting between beats. A person’s blood pressure is considered normal if it stays below 120/80, stated as “120 over 80.” The NHLBI recommends that people with kidney disease use whatever therapy is necessary, including lifestyle changes and medicines, to keep their blood pressure below 130/80.

Microalbuminuria and Proteinuria
Healthy kidneys take wastes out of the blood but leave protein. Impaired kidneys may fail to separate a blood protein called albumin from the wastes. At first, only small amounts of
albumin may leak into the urine, a condition known as microalbuminuria, a sign of deteriorating kidney function. As kidney function worsens, the amount of albumin and other proteins in the urine increases, and the condition is called proteinuria. A doctor may test for protein using a dipstick in a small sample of a person’s urine taken in the doctor’s office. The color of the dipstick indicates the presence or absence of proteinuria.

A more sensitive test for protein or albumin in the urine involves laboratory measurement and calculation of the protein-to-creatinine or albumin-to-creatinine ratio. Creatinine is a waste product in the blood created by the normal breakdown of muscle cells during activity. Healthy kidneys take creatinine out of the blood and put it into the urine to leave the body. When the kidneys are not working well, creatinine builds up in the blood.

The albumin-to-creatinine measurement should be used to detect kidney disease in people at high risk, especially those with diabetes or high blood pressure. If a person’s first laboratory test shows high levels of protein, another test should be done 1 to 2 weeks later. If the second test also shows high levels of protein, the person has persistent proteinuria and should have additional tests to evaluate kidney function.

Glomerular Filtration Rate (GFR) Based on Creatinine Measurement

GFR is a calculation of how efficiently the kidneys are filtering wastes from the blood. A traditional GFR calculation requires an injection into the bloodstream of a substance that is later measured in a 24-hour urine collection. Recently, scientists found they could calculate GFR without an injection or urine collection. The new calculation—the eGFR—requires only a measurement of the creatinine in a blood sample.

In a laboratory, a person’s blood is tested to see how many milligrams of creatinine are in one deciliter of blood (mg/dL). Creatinine levels in the blood can vary, and each laboratory has its own normal range, usually 0.6 to 1.2 mg/dL. A person whose creatinine level is only slightly above this range will probably
not feel sick, but the elevation is a sign that the kidneys are not working at full strength. One formula for estimating kidney function equates a creatinine level of 1.7 mg/dL for most men and 1.4 mg/dL for most women to 50 percent of normal kidney function. But because creatinine values are so variable and can be affected by diet, a GFR calculation is more accurate for determining whether a person has reduced kidney function.

The eGFR calculation uses the patient’s creatinine measurement along with age and values assigned for sex and race. Some medical laboratories may make the eGFR calculation when a creatinine value is measured and include it on the lab report. The National Kidney Foundation has determined different stages of CKD based on the value of the eGFR. Dialysis or transplantation is needed when the eGFR is less than 15 milliliters per minute (mL/min).

**Blood Urea Nitrogen (BUN)**

Blood carries protein to cells throughout the body. After the cells use the protein, the remaining waste product is returned to the blood as urea, a compound that contains nitrogen. Healthy kidneys take urea out of the blood and put it in the urine. If a person’s kidneys are not working well, the urea will stay in the blood.

A deciliter of normal blood contains 7 to 20 milligrams of urea. If a person’s BUN is more than 20 mg/dL, the kidneys may not be working at full strength. Other possible causes of an elevated BUN include dehydration and heart failure.

**Additional Tests for Kidney Disease**

If blood and urine tests indicate reduced kidney function, a doctor may recommend additional tests to help identify the cause of the problem, such as kidney imaging and biopsy.

**Kidney Imaging**

Methods of kidney imaging—taking pictures of the kidneys—include ultrasound, computerized tomography (CT)
scan, and magnetic resonance imaging (MRI). These tools are most helpful in finding unusual growths or blockages to the flow of urine.

**Kidney Biopsy**

A doctor may want to examine a tiny piece of kidney tissue with a microscope. To obtain this tissue sample, the doctor will perform a kidney biopsy—a hospital procedure in which the doctor inserts a needle through the patient’s skin into the back of the kidney. The needle retrieves a strand of tissue less than an inch long. For the procedure, the patient lies face down on a table and receives a local anesthetic to numb the skin. The sample tissue will help the doctor identify problems at the cellular level.

**References**

CHAPTER 3
CHAPTER 3

ACUTE KIDNEY FAILURE

Introduction

Acute renal failure (ARF) is the syndrome arising from a rapid fall in GFR (over hours to days). It is characterized by retention of both nitrogenous (including urea and creatinine) and non-nitrogenous waste products of metabolism, as well as disordered electrolyte, acid-base, and fluid homeostasis.

Until recently, there has been no consensus on a clinical definition of ARF, making it difficult to compare and interpret studies of prevention, incidence and treatment. A survey of 598 participants at a critical care nephrology conference in 2004 revealed 199 different criteria to define ARF, and 90 for initiating RRT.

In 2004, a multilayered definition of ARF was proposed by the Acute Dialysis Quality Initiative (ADQI). In this model, ARF is stratified into 5 stages based on severity and duration of injury: Risk, Injury, Failure, Loss and End-stage disease (RIFLE).

More recently, as a modification of the RIFLE criteria, the concept of Acute Kidney Injury (AKI) has emerged and is likely to be widely adopted as the standard over the next few years.

Acute Kidney Injury is
defined as functional or structural abnormalities, or markers of kidney damage (including abnormalities in blood, urine, tissue tests or imaging studies), present for <3 months.

The major cause of acute renal failure is hypervolemia secondary to dehydration as a result of gastroenteritis (32%). Obstetric blood loss largely due to inadequate obstetric care is also an important cause (15%). Other main causes are nephrolithiasis (10%), acute glomerulonephritis (12%) and sepsis. The precise drug involved is drug-induced acute renal failure because of over the counter sales of drugs.

Acute kidney failure is a sudden and complete loss of kidney function. Many things can cause acute kidney failure, such as accidents, medicines, surgery, low blood pressure from shock, blockages of the bladder or kidney or serious infections. Without enough blood, the kidneys cannot work. The kidneys may start working again with medical treatment. Patients with acute kidney failure may need dialysis therapy until the kidneys start to work again.

Between 5% - 25% of all hospitalized patients develop ARF. A greater prevalence of ARF is found in critically ill patients. Despite improvement in the medical care of individuals with ARF, mortality generally exceeds 50%.

There are typically three categories of ARF: pre-renal, intrinsic, and post-renal ARF. The pathophysiologic mechanisms differ for each of the categories.

**Prerenal Acute Renal Failure**

Pre-renal ARF is characterized by reduced blood delivery to the kidney. Causes of intravascular volume depletion are: hemorrhage, dehydration, or gastrointestinal fluid losses, reduced cardiac output, hypotension, NSAIDs, ACE, ARBs, Reno vascular obstruction, systemic vasoconstriction. Pre-renal ARF occurs in approximately 10% to 25% of patients diagnosed with ARF.
Acute Kidney Failure

Intrinsic Acute Renal Failure
Intrinsic renal failure, also referred to as intra-renal ARF, is caused by diseases that can affect the integrity of the tubules, glomerulus, interstitium, or blood vessels. Damage is within the kidney; changes in kidney structure can be seen on microscopy. Acute tubular necrosis (ATN) represents a pathophysiologic condition that result from toxic or ischemic insult to the kidney. The most common cause of intrinsic renal failure is ATN and it accounts for approximately 50% of all cases of ARF.

Postrenal Acute Renal Failure
Post-renal ARF is due to obstruction of urinary outflow. Causes include benign prostatic hypertrophy, pelvic tumors, and precipitation of renal calculi. Post-renal ARF accounts for less than 10% of cases of ARF.

Clinical Presentation and Diagnosis of Acute Renal Failure
There are some clinical and laboratory findings that assist in the general diagnosis of ARF, others are used to differentiate between pre-renal, intrinsic, and post-renal ARF. For example, patients with pre-renal ARF typically demonstrate enhanced sodium reabsorption, which is reflected by a low urine sodium concentration and a low fractional excretion of sodium. Urine is typically more concentrated with pre-renal ARF and there is a higher urine osmolality and urine: plasma creatinine ratio compared to intrinsic and post-renal.

Sign and Symptoms of Uremia
- Peripheral edema
- Weight gain
- Nausea/vomiting/diarrhea/anorexia
- Mental status changes
- Fatigue
- Shortness of breath
- Pruritus
- Volume depletion
- Weight loss
- Anuria alternating with polyuria
- Colicky abdominal pain radiating from flank to groin
Physical Examination Findings
- Hypertension
- Jugular venous distention
- Pulmonary edema
- Rales
- Asterixis
- Pericardial or pleural friction rubs
- Hypotension/orthostatic hypotension
- Rash
- Bladder distention
- Prostatic enlargement

Laboratory Tests
- Elevated serum creatinine concentration
- Elevated BUN concentration
- Decreased creatinine clearance
- BUN: creatinine ratio (elevated in pre-renal ARF)
- Hyperkalemia
- Metabolic acidosis

Urine Analysis
- Sediment
- Scant or bland (pre-renal or post-renal ARF)
- Brown, muddy granular casts (highly indicative of ATN)
- Proteinuria (glomerulonephritis or allergic interstitial nephritis)
- Eosinophiluria (acute interstitial nephritis)
- Hematuria /red blood cell casts (glomerular disease or bleeding in urinary tract)
- White blood cells or casts (acute interstitial nephritis or severe pyelonephritis)

Common Diagnostic Procedures
- Urinary catheterization (insertion of a catheter into a patient's bladder; an increase in urine output may occur with post renal obstruction)
Acute Kidney Failure

- Renal ultrasound (uses sound waves to assess size, position, and abnormalities of the kidney; dilation of the urinary tract can be seen with post renal ARF)
- Renal angiography (administration of intravenous contrast dye to assess the vasculature of the kidney)
- Retrograde pyelography (injection of contrast dye into the ureters to assess the kidney and collection system)
- Kidney biopsy (collection of a tissue sample of the kidney for the purpose of microscopic evaluation; may aid in the diagnosis of glomerular and interstitial diseases)

Discriminating Between Acute Renal Failure from Chronic Renal Failure

Patients are often found to have ↑Cr or ↓eGFR ±oliguria on presentation with unrelated medical or surgical conditions. Differentiating true ARF from stable (long standing) CKD or even an acute deterioration on pre-existing renal impairment is often very important.

Much is made of the ability to distinguish the two after an initial clinical assessment and blood tests. Many of these features, even when present, are at best suggestive and, at worst, misleading. The only two consistently useful discriminators are¹:

1. Previous measurements of renal function.
2. Ultrasound

References
CHAPTER 4

Classification of Chronic Kidney Disease (CKD)

<table>
<thead>
<tr>
<th>eGFR (mL/min)</th>
<th>CKD Stage 1</th>
<th>CKD Stage 2</th>
<th>CKD Stage 3</th>
<th>CKD Stage 4</th>
<th>Established renal failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 90</td>
<td>Kidney damage with normal or increased eGFR</td>
<td>Kidney damage with mild eGFR fail</td>
<td>Moderate fall in eGFR</td>
<td>Severe fall in eGFR</td>
<td>Established renal failure</td>
</tr>
<tr>
<td>60-89</td>
<td>CKD Stage 2</td>
<td>CKD Stage 3</td>
<td>CKD Stage 4</td>
<td>CKD Stage 5</td>
<td>Established renal failure</td>
</tr>
<tr>
<td>30-59</td>
<td>CKD Stage 3</td>
<td>CKD Stage 4</td>
<td>CKD Stage 5</td>
<td>Established renal failure</td>
<td></td>
</tr>
<tr>
<td>15-29</td>
<td>CKD Stage 4</td>
<td>Established renal failure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;15</td>
<td>Established renal failure</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Stage 1 & 2 must have other evidence of kidney damage which may be one of the following: Persistent microalbuminuria, persistent proteinuria, persistent hematuria (other exclusion of other causes), structural abnormalities of the kidneys demonstrated on ultrasound scanning or other radiological tests or biopsy proven chronic glomerulonephritis.

Patients found to have an eGFR of 60-89 mL/min without one of these markers should not be considered to have CKD and should not be subjected to further investigation (unless there are additional reasons to do so).
CHAPTER 4

CHRONIC KIDNEY DISEASE/END-STAGE RENAL DISEASE

Introduction

Chronic kidney disease, also known as chronic renal insufficiency, progressive kidney disease, or nephropathy, is defined as the presence of kidney damage or decreased glomerular filtration rate for 3 months or more. Generally, CKD is a progressive decline in kidney function (a decline in the number of functioning nephrons) that occurs over a period of several months to years. The decline in kidney function in CKD is often irreversible. Therefore, measures to treat CKD are aimed at slowing the progression to end-stage renal disease (ESRD). 1

Incidence of End-Stage Renal Disease

The incidence of end stage renal disease is estimated to be about 100 patients/million populations. During the last five years the incidence of diabetes mellitus as a cause of end stage renal disease has increased and now diabetes and chronic glomerulonephritis are the leading causes of end stage renal disease, followed by hypertension and renal stone disease 2.

The survey was done in 1997 by the Health Care Finance Administration that more than 850,000 people are being treated worldwide for ESRD; 230,000 are Americans. As the graph to the right illustrates, the incidence of ESRD increases with age, with ages 45-64 are being the largest age group exhibiting
Renal Failure: Its Treatment in Current Systems of Medicines

ESRD. The incidence decreases in advancing years because of mortality associated with ESRD. ESRD is more prevalent in men; 54% of ESRD patients are men whereas 46% are women. African and Native Americans have higher incidence of ESRD. In 1998, the National Kidney Foundation reported 40% of ESRD patients were African American. African Americans also develop kidney failure at an earlier age than Caucasians.3

The Stages of Chronic Kidney Disease

The National Kidney Foundation developed a classification system for CKD. The staging system defines the stages of CKD based on GFR level, but also accounts for evidence of kidney damage in the absence of changes in GFR, as in stage 1 CKD. 1The severity of chronic kidney disease (CKD) is described by five stages;

1. **CKD1** – GFR above 90mL/min/1.73m² with evidence of kidney damage
2. **CKD2** (Mild) – GFR of 60 to 89 mL/min/1.73m² with evidence of kidney damage
3. **CKD3** (Moderate) – GFR of 30 to 59 mL/min/1.73m²
4. **CKD4** (Severe) – GFR of 15 to 29 mL/min/1.73m²
5. **CKD5** Kidney failure - GFR less than 15 mL/min/1.73m²

Some people add CKD5D for those stage 5 patients requiring dialysis; many patients in CKD5 are not yet on dialysis

Stages 1 and 2 CKD

Description

People with Stage 1 CKD have kidney damage with normal or high GFR greater than 90 ml/min. They generally do not experience any symptoms of kidney damage even if the kidneys are no longer functioning at full capacity. Most people are diagnosed with Stage 1 CKD in the process of being tested for another condition such as diabetes or high blood pressure, which are the two leading causes of kidney disease.
Other signs of Stage 1/2 CKD:
- Higher than normal levels of creatinine or urea in the blood
- Blood or protein in the urine
- Evidence of kidney damage in an MRI, CT scan, ultrasound or contrast X-ray
- A family history of polycystic kidney disease

Stage 3
Description
A person with Stage 3 CKD has kidney damage with a moderate decrease in the GFR of 30-59 ml/min. As kidney function declines, waste products and toxins begin to build up in the blood. Once toxins reach a certain level, uremia occurs and complications of kidney disease such as high blood pressure, anemia (a shortage of red blood cells) and/or early bone disease are more likely.

Stage 4
Description
A person with Stage 4 CKD has advanced kidney damage with a severe decrease in GFR to 15-30 ml/min. It is likely someone with Stage 4 CKD will need dialysis or a kidney transplant in the near future.

As kidney function declines, waste products and toxins build up in the blood causing a condition known as "uremia." At Stage 4, complications such as high blood pressure, anemia (a shortage of red blood cells), bone disease, heart disease and other cardiovascular diseases become more likely so it is important that people at Stage 4 CKD pay careful attention to their health.

Stage 5
Description
A person with Stage 5 CKD has end stage renal disease (ESRD) with a GFR of 15 ml/min or less. At Stage 5 kidney disease, your kidneys are no longer able to remove waste and fluids from the body effectively which leads to a build-up of toxins in the blood. Most people at Stage 5 CKD will need dialysis or a kidney transplant.
Patient in this stage of CKD may experience symptoms such as:

- Loss of appetite
- Nausea and/or vomiting
- Headaches
- Fatigue
- Trouble concentrating
- Itching
- Little or no urine
- Swelling, especially around the eyes and ankles
- Muscle cramps
- Tingling in hands or feet
- Changes in skin color
- Increased skin pigmentation

The most common reasons for chronic kidney disease are:

- Damage to kidneys as a result of diabetes and high blood pressure.
- Kidney diseases:
  - Polycystic disease – a hereditary disease which causes a slow destruction of the kidneys
  - Glomerulonephritis – swelling of the filter parts of the kidney (called the glomeruli) that works to remove waste and fluid from the blood.
- Damage to the kidney from heart disease or drug abuse.
- Kidney infections.
- Kidney stones or a blockage present from birth.

With chronic kidney disease, waste builds up in the blood. These wastes must be removed by dialysis treatment. Once the kidneys stop working, they will usually not work again. The patient will need regular dialysis or a kidney transplant.
Chronic Kidney Disease/ End-Stage Renal Disease

The incidence of end stage renal disease is likely to be higher in Pakistan and India than the reported from the developed world, with chronic glomerulonephritis being the most common cause, accounting for more than one-third of patients, while diabetic nephropathy accounts for about one-fourth of all patients in India.

Patients are generally younger (mean age 42 years) at the time of detection of ESRD and two-thirds first see a nephrologist after they have reached end stage.

Increasing awareness of renal disease among the population and general practitioners could result in early diagnosis of chronic renal failure and give preventive strategies to delay the onset of end stage renal disease.

End stage renal disease is a common and rapidly increasing public health problem all over the world, both in developed and developing countries.

Due to lack of exact figure, the incidence of end stage renal disease is estimated to be 100 per million populations (pmp) in South Asian countries Pakistan, India and Bangladesh.

Because of the progressive nature of CKD, determination of risk factors for CKD is difficult. Risk factors identified for CKD are classified into three categories:

Susceptibility Factors, which are associated with an increased risk of developing CKD, but are not directly proven to cause CKD. These factors are generally not modifiable by pharmacologic therapy or lifestyle modifications.

Initiation Factors, which directly cause CKD. These factors are modifiable by pharmacologic therapy.

Progression factors, which result in a faster decline in kidney function and cause worsening of CKD. These factors may also be modified by pharmacologic therapy or lifestyle modifications to slow the progression of CKD.

The Ways to Diagnose Chronic Kidney Disease Earlier

If the patient has any of the following signs, then he should contact the doctor:

- Darkness or redness in the urine
- Urine that has an offensive odor
- Urine that looks very foamy
Frequent urination, sometimes with pain or burning on passing urine
Any definite change in the amount of urine passed
Persistent thirst
Edema – swelling of the legs, hands, face
Raised or high blood pressure
Back pain in the renal area, especially if there is fever
Anemia
Tiredness or feeling unwell without apparent cause
Widespread itchy skin (pruritus)

These signs can indicate other problems besides kidney disease. For those patients who already have high blood pressure or diabetes, it’s important that all instructions from the physician are carefully followed. Poorly controlled blood pressure and diabetes are two major causes of Chronic Kidney Disease.

Other potential causes of Chronic Kidney Disease include large kidney stones and prostate disease. Potentially, injury is caused to the kidneys when the flow of urine out of the body is impeded or blocked.

Glomerulonephritis (a family of different forms of kidney inflammation) can lead to Chronic Kidney Disease. These conditions affect the glomeruli, the fine filters in the kidneys. At one time, these diseases were collectively known as Bright’s disease – before modern research identified them as separate conditions.

A fresh urine sample can be checked for visual changes, like the presence of blood or a very dark coloring, cloudiness or a very frothy appearance.

If there is a bad odor, an infection may be suspected. Most infections are short lived (acute), but can become chronic. Chronic Pyelonephritis is a persistent infection of the kidneys – damage can be caused that contributes to Chronic Kidney disease and eventual kidney failure.

Clinical Presentation of Chronic Kidney Disease

General

The development of CKD is usually subtle in onset, often with no noticeable symptoms.
Symptoms:
Stages 1 and 2 CKD and generally asymptomatic.
Stages 3 and 4 CKD may be associated with minimal symptoms.
Typical symptoms associated with stage 5 CKD include pruritus, dyspepsia, nausea, vomiting, constipation, muscle pain, fatigue, and bleeding abnormalities.

Signs
Cardiovascular: Worsening hypertension, edema, dyslipidemia, left ventricular hypertrophy, electrocardiographic changes and chronic heart failure.
Musculoskeletal: Cramping
Neuropsychiatric: Depression, anxiety, impaired mental cognition.
Gastrointestinal: Gastro esophageal reflux disease, gastrointestinal bleeding, and abdominal distention.
Genitourinary: Changes in urine volume and consistency, foaming of urine, and sexual dysfunction

Laboratory Tests
Stage 1 and 2 CKD: Increased blood urea nitrogen (BUN) and serum creatinine (SCr) and decreased GFR.
Stages 3, 4, and 5 CKD: Increased BUN and SCr; decreased GFR.

Advanced Stages
Increased potassium, phosphorus, and magnesium; decreased bicarbonate; calcium levels are generally low in earlier stages of CKD, and maybe elevated in stage 5 CKD, secondary to the use of calcium-containing phosphate binders.
Decreased albumin, if inadequate nutrition intake in advanced stages.
Decreased red blood cell
count, hemoglobin and hematocrit; iron metabolism may also be altered. Erythropoietin levels are not routinely monitored and are generally normal to low. Urine positive for albumin or protein. Increased parathyroid hormone level; decreased vitamin D levels. Stool may be Hem occult –positive if GI bleeding occurs from uremia.

Other Diagnostic Tests

Structural abnormalities of kidney may be present on diagnostic exams

References:
CHAPTER 5
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DRUG-INDUCED KIDNEY DISEASE

Introduction
The kidney maintains the vital functions of clearing excess body fluid and removing metabolic and exogenous toxins from the blood. The kidney is particularly vulnerable to drugs and other agents that cause renal damage. The heart pumps approximately 25% of cardiac output into the kidneys. Any drug in the blood will eventually reach the highly vascularized kidneys. May potentially cause drug-induced renal failure. If the drug is primarily cleared by the kidney, the drug will become increasingly concentrated as it moves from the renal artery into the smaller vasculature of the kidney. The drug may be filtered or secreted into the lumen of the renal tubules. The concentrated drug exposes the kidney tissue to far greater drug concentration per surface area. Drug-induced kidney failure is a major adverse event associated with multiple medication classes. Medications as diverse as OTC analgesics (ibuprofen, acetaminophen), antibiotics and chemotherapy agents can cause kidney damage. Medication use accounts for 2% of hospital admissions for acute renal failure and up to 15% of admissions into intensive care.

Clinical Presentations
Patients who experience acute-onset renal failure often complain of increased shortness of breath, ankle swelling and weight gain. The reduced ability of the kidney to clear extra fluid from the body. Stopping the medication may allow the kidney to
If the kidney has extensive damage, the kidney may reduce or even stop producing urine. Hemodialysis may be necessary for a short-term bridge until the kidney can recover. In some cases, the damage is irreversible and the patient will require life-long dialysis or a kidney transplant.

Drug-induced renal disease can mimic renal disease from other causes, such as autoimmune disease and infection. A thorough physical examination and medical history should be performed. Increase in serum creatinine and BUN. Additional urine tests: protein excretion, creatinine concentration, osmolality, or sodium excretion. A thorough and accurate review of all medications, including all prescription, over-the-counter and herbal medications. Importance of dose and duration of exposure rule out all other causes of kidney failure.

**Prevention**

Following are the principles of pharmacotherapy in order to avoid drug-induced kidney failure.

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**TABLE 1. DRUG CLASSES ASSOCIATED WITH RENAL FAILURE/DYSFUNCTION**

<table>
<thead>
<tr>
<th>Drug Class</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotics</td>
</tr>
<tr>
<td>Analgesics</td>
</tr>
<tr>
<td>Anticonvulsants</td>
</tr>
<tr>
<td>Antifungals</td>
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<tr>
<td>Amphotericin B</td>
</tr>
<tr>
<td>Antineoplastics</td>
</tr>
<tr>
<td>Anti-hypertensives</td>
</tr>
<tr>
<td>Drugs of abuse</td>
</tr>
<tr>
<td>Diagnostic agents</td>
</tr>
<tr>
<td>Herbal supplements</td>
</tr>
<tr>
<td>HMG-CoA reductase inhibitors</td>
</tr>
<tr>
<td>Immune globulin</td>
</tr>
<tr>
<td>H₂-antagonists</td>
</tr>
<tr>
<td>Lithium</td>
</tr>
<tr>
<td>Proton pump inhibitors</td>
</tr>
<tr>
<td>Others</td>
</tr>
</tbody>
</table>

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Drug-Induced Kidney Disease

- Knows the potential nephrotoxicity of diagnostic and therapeutic pharmacologic agents.
- Compare the potential risks and expected benefits for each course of treatment.
- Consider alternative diagnostic and therapeutic approaches.
- Use the lowest dose and shortest course of therapy that is efficacious.
- Monitor appropriately for potential toxicity.
- Monitor therapy if toxicity is occurs.
- If possible, nephrotoxic agents should be avoided inpatients at highest risk of developing ATN, including elderly patients and those with heart failure, liver disease, existing renal disease, renal-artery stenosis and diabetes mellitus.
- Maintenance of adequate hydration and therapeutic drug monitoring may minimize the risk of ATN.¹

Treatment

The offending drug should be withdrawn, if possible. Supportive measures should be implemented, with careful monitoring of fluid balance, hydration status and electrolytes, and institution of renal replacement therapy if indicated. Intravenous dopamine 1-3μg per kg per minute appears to cause selective vasodilatation of renal vessels, but prospective controlled trials and meta-analyses have concluded that it does not reduce mortality or accelerate recovery of renal function.²

References

CHAPTER 6
CHAPTER 6

RENAL FAILURE IN CHILDREN

Introduction
Renal failure in childhood is very different from renal failure in adults. While kidney disease is a complicated chronic illness affecting many aspects of a child’s life, it is a manageable condition. The goal of treatment is to have the patient lead a normal life.

The causes of kidney disease are also different for children than adults. Among the children under the age of 12, the most common causes of kidney failure are problems with the structure of the kidney, bladder, or anywhere along the urinary tract. Most children are born with these problems. In many cases of structural kidney disease, surgery is required either to preserve kidney function, to manage urinary problems, or to prepare patients for renal transplantation. In children over the age of 12, glomerulonephritis is the most frequent cause of kidney failure.

Glomerulonephritis can also occur in younger children under 12. Not all kinds of glomerulonephritis are focal segmental glomerulosclerosis, which can lead to renal failure by causing scarring of the kidneys. In contrast to adults, high blood pressure and diabetes mellitus are extremely rare cause of kidney failure in children. High blood pressure does not usually cause kidney failure in children, but often is caused by the renal failure.
Renal Failure: Its Treatment in Current Systems of Medicines

Treatment
The treatment provided to the child must meet the medical and social needs of each child. The goal of treatment is to provide most normal lifestyle possible to allow the child to grow and mature.

Medication
Failure to grow has been a common problem among children with chronic kidney disease, but improved medications such as recombinant growth hormone, nutritional supplements, and human recombinant erythropoietin have allowed for normal growth in many patients.

Hemodialysis
Children with renal failure receive dialysis by having a permanent catheter, similar to a large IV placed in their neck, chest or leg. Other patients have a graft or fistula which is a connection under the skin which is used for dialysis by placing a needle into it. Although needle insertion, a necessary part of hemodialysis, may be uncomfortable, the treatment itself is not painful. Many children rest, do homework, sleep, or play during the hemodialysis treatment. Some small children may require more intensive monitoring during dialysis.

Peritoneal Dialysis
There are several ways to do peritoneal dialysis. All types of peritoneal dialysis used in adults are available for children, including CAPD and CCPD. First, a PD catheter appropriate to the size of the patient is placed surgically into the patient’s abdomen.
Transplantation

Transplantation is the treatment of choice for nearly all children with ESRD. A renal transplant may allow the child an opportunity for better growth than dialysis treatments and for more normal and active life as a child. In addition, there may not be as many diet restrictions once the patient has a new kidney.

Many parts of renal transplantation in children are no different from those in adults. The patient and the donor must have blood types that match and must be able to undergo surgery to have the kidney removed from the donor and put into the patient. The donor for a kidney transplant to a child may be a living relative, cadaver, or a living unrelated adult. The chances of successful kidney transplantation are more when a child receives a kidney from a donor who is at least six years older. Usually adult kidneys are transplanted in to small children. If possible, a living related kidney transplant is preferred because there is a lower chance of rejection and better long-term kidney survival.

Preparation for a kidney transplant is complex and requires a full range of medical services. The child is tested to assure that he is healthy and prepared for the procedure. Immunizations are updated. The child is tested for infections. In some cases, surgery to correct bladder problems may be necessary before transplantation.

References
CHAPTER 7

Figure 1 - Kidney biopsy. Panel A shows extensive fibrosis, appearing in trichromes stain (1x25) as green areas. Panel B (Masson stain-466x) shows global hyalinosis, mostly of mesangial cells and a cellular crescent formation on the right side (arrow). Panel C shows a silver stain exhibiting occasional double contours of the glomerular basement membrane (arrow). Panel D reveals a high power electron micrograph abundant tubulomucoial inclusions in the cytoplasm of glomerular endothelial cells.
CHAPTER 7

HIV AND KIDNEY DISEASE

Introduction

Almost 40 million people worldwide are infected with the human immunodeficiency virus-1 (HIV). HIV is associated with almost every described renal lesion, but HIV-associated nephropathy (HIVAN) has been the most common renal lesion identified in such patients in the developed world.

HIVAN

HIVAN result from direct HIV infection of renal proximal tubular cells and podocytes, causing the so-called collapsing variant of focal and segmental glomerulosclerosis with cystic tubular dilation. As HIV infection has burgeoned globally, HIVAN has become a significant cause of ESRD, particularly in black adults.

Symptoms and Signs

Oedema, nephrotic-range proteinuria and renal impairment is common. Blood pressure is often normal¹.
Management of HIVAN

Highly active anti-retroviral therapy (HAART), a three drug regimen of 2 reverse transcriptase inhibitors and a protease inhibitor, has completely transformed the outlook in HIV-infected individuals. ACEI may reduce proteinuria and delay progression.

Corticosteroid treatment can be useful in patients who do not respond to the above treatment. There is some evidence that cyclosporine might be helpful in selective cases, however further trials are required on both steroids and cyclosporine before these drugs can become standardized treatment if at all\textsuperscript{2,3}.

Standard Universal Precautions For HIV-Infected Patients With ESRD

They do not require isolation on hemodialysis. Survival on either HD or CAPD seems comparable. Renal transplantation is increasingly thought to improve patients survival compared to dialysis, but patient selection is important. Those with stable disease, compliant on medication, with low viral load and preserved CD4 counts can be referred for consideration – drug interaction and toxicities are a major challenge\textsuperscript{1}.
HIV and Kidney Disease

References
Hepatitis B vaccine protects against serious disease causing inflammation and damage to the liver
CHAPTER 8

HEPATITIS B AND KIDNEY DISEASE

Introduction

Hepatitis B (HBV) has affected 350 million people world-wide, especially in Africa and South east Asia. It is a DNA virus transmitted from mother to children, or between close interaction and sexual contacts.

When HBV invade into the body, they will duplicate in the liver cells, leading to Hepatitis B. Besides, these viruses will combine with antibodies, producing immune complex. More and more immune complexes will circulate with the blood circulation and deposit in the renal tissue, causing pathological changes of glomerular basement membrane. With gradual damage of the renal tissues, kidney function declines gradually and symptoms will appear.

In mild cases, patients may present swelling on eyelids, lower back pain, fatigue, decreased urine output, etc. In severe cases, symptoms like high blood pressure, blood in the urine, swelling of limbs can be caused. Individuals with serious kidney damage will experience oliguria or anuria. In the end, uremia can be caused.

Pathogenesis of Hepatitis B and Kidney Disease

The occurrence of Hepatitis B and Kidney Disease is confirmed to be associated with the immune injury caused by deposition of immune complex in the glomerular capillary loops. About 5 months after patient's affected by HBV, there may be HBCAb, HBeAb, and HBsAb in the blood serum.

Part of the polypeptide products is tested to be the antigenic determinant of HBsAb. These components will combine with antibodies, forming immune complex. With massive immune complex depositions in the glomerular capillary loops,
complement can be activated, resulting in immune injury to the kidneys. Three common nephropathies are described:

**Membrane Glomerulonephritis (MN)**

It is common among children between 2-12 years old, presenting with proteinuria. Usually HBeAg positive. Tends to remit spontaneously with clearance of HBeAg and development of anti-HBe antibodies. Adult present with a chronic nephrotic syndrome, viral liver disease and progressive renal impairment.

**Mesangiocapillary Glomerulonephritis (MCGN)**

This nephropathy is common in adults. Clinical presentations include high blood pressure, heavy proteinuria, haematuria and progressive increased creatinine. HBsAg and anti-HB antibodies are found while cryoglobulins are not present.

**Classical Polyarteritis Nodosa (PAN)**

Occurs within 4 months of infection in adults, presenting as a medium vessel vasculitis.

**Treatment of HBV-Associated GN in Adults**

The purpose of treatment is to eradicate the virus (HBsAg negative, undetectable viral DNA) and promote sero-conversion to immune status (anti-HBs antibody positive). Significant renal disease tends not to remit without treatment. Liver disease may require treatment on its own merits. Response rates are higher in membranous glomerulonephritis than mesangiocapillary
glomerulonephritis. Remission tends to follow clearance of HBeAg and the appearance of anti-HBe antibodies.

The best means of controlling HBV-related renal disease is to prevent it. Vaccination against HBV should be given to high risk individuals. The medication used for treatment includes Interferone -α₂b and Lamivudine 100mg. Corticosteroids should be avoided.

**Standard Universal Precautions for Hepatitis B Infected Patients with Esrd**

Hepatitis B infected patients with ESRD require isolation on hemodialysis units. Dedicated machines should be used, and dialyzer re-use avoided. There is no contraindication to CAPD in well patients. Transplantation is thought to offer a survival benefit over remaining on dialysis, but selecting appropriate patients requires full HBV serology testing, viral load, and may require liver biopsy to predict risk. Yearly screening for hepatocellular carcinoma, with serum α-fetoprotein and USS liver should be performed in high risk patients.

**References**

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HEPATITIS C AND KIDNEY DISEASE

Introduction
HCV is a RNA virus with 6 different genotypes, causing hepatitis, cirrhosis and hepatocellular carcinoma. It is transmitted parentally, affecting IV drug users and those accidentally infected through needle-sharing or blood products. Infection is mild and often subclinical after a long incubation period. 70-85% fails to clear acute infection and become chronic carriers. Chronic HCV and its associated mixed cryoglobulinaemia are known to be the principal cause of type 1 MCGN.

HCV-Related Type 1 MCGN
Cryoglobulins deposited in medium and small vessels in the skin, joints and glomeruli, fixing complement and causing local inflammation and injury leading to glomerulonephritis.

Symptoms and Signs
Fatigue, weakness, weight loss, episodic palpable purpuric rash on legs, arthralgia, myalgia, and mono-neuritis multiplex. Hepato-splenomegaly and Raynaud’s phenomenon. May present as an acute and disseminated vasculitis. Renal manifestations include hematuria, proteinuria, increased blood pressure, renal impairment.

Management
Most HCV-infected patients have evidence of liver disease, and seemingly normal hepatic function does not exclude the presence of HCV. Patients with HCV infection are generally treated with pegylated interferon-α and the oral anti-viral agent...
ribavirin. Of the 6 known HCV genotypes, types 2 and 3 are more responsive to therapy than types 1 or 4, with sustained virological response rates of 80% versus 45%. Hemolysis related to ribavirin is common.

No known therapy specifically alters renal outcomes in Mesangiocapillary glomerulonephritis (MCGN), but the following therapy can be considered:

- EPO to maintain Hb > 11g/dL
- ACEI to limit proteinuria
- HMG-CoA-reductase inhibitors if LFT normal.

Also recommend:
- Avoid alcohol
- Avoid sharing blood-contaminated items
- Use condoms

**Standard Universal Precautions for Hepatitis C Infected Patients with ESRD**

Hepatitis C infected patients with ESRD do not require isolation on hemodialysis. Check ALT- 6 monthly. Yearly screening for hepatocellular carcinoma ± cirrhosis by USS liver and serum α-fetoprotein should be performed. Renal transplantation offers superior survival to maintenance HD or CAPD. HCV positive transplant recipients do less well over the long-term than HCV patients. A sub-group of HCV + ESRD patients may benefit from anti-HCV treatment with pegylated-IFN prior to transplantation.

**References**

Kidney Failure and Cancer

CHAPTER 10

KIDNEY FAILURE AND CANCER

Kidney Failure Associated with Cancer

Introduction

Kidney disease frequently complicates malignancy and its treatment. The spectrum of disease in this setting includes acute renal failure (ARF), chronic renal failure, and tubular disorders.

Renal failure remains an important complication of cancer and its treatment. The spectrum of cancer-associated renal disease has changed in the past 20 years, in large part as a result of the use of newer chemo radiotherapy regimens. Nevertheless, a simple and systematic approach to assess and treat potential pre-renal, intra-renal, and post-renal causes is indicated in all patients. Early diagnosis and treatment of renal failure is vital—both to improve renal outcomes and to ensure that patients are best prepared for further oncologic treatment. Close cooperation with oncology colleagues is essential to improve outcomes in these complex patients.

According to the definition and classification of chronic kidney disease by the National Kidney Foundation and Kidney Disease Outcomes Quality Initiative, it is associated with other diseases including cancer. Chronic kidney disease and cancer are connected in a number of ways in both directions: cancer can cause chronic kidney disease either directly or indirectly through the adverse effects of therapies; chronic kidney disease may, conversely, be a risk factor for cancer; and both maybe associated because they share common risk factor, most often toxins. It is also well established that patients on renal replacement therapy for end-stage kidney disease, either dialysis or transplantation are at high risk of cancer.

A wide variety of cancers can occur at many sites in patients with chronic kidney disease before and after renal replacement therapy. Kidney transplant recipients are at very high risk of cancers, most, but not all, of which with likely viral etiology. Patients on dialysis and individuals with early stage chronic
kidney disease also experience, but to lower extent, an excess risk for a number of tumors².

The researchers discovered that men with moderate kidney dysfunction had a 39% increased risk of developing cancer over the risk seen in men with normal kidney function. Risk increased as kidney function declined, and men with significant kidney dysfunction had a three-fold increased risk above normal. The risk for lung and urinary tract cancers, but not prostate cancer, was higher among men with kidney disease³.

**Causes of Renal Failure in Cancer Patients**

**Pre-Renal**
- extracellular fluid depletion (poor intake, vomiting, diarrhea, hypercalcemia)
- hepatorenal syndrome (veno-occlusive disease, hepatic resection)
- drugs (calcineurin inhibitors, nonsteroidals)

**Intrinsic**
- glomerular
- membranous nephropathy
- amyloidosis (multiple myeloma)
- pamidronate-associated collapsing
- glomerulopathy (incidence unknown)
- light-chain deposition disease
- tubulointerstitial
- acute tubular necrosis (toxic/ischemic)
- lymphomatous infiltration of the kidney
- light-chain deposition disease
- drugs (cisplatin, ifosfamide)
- intravenous contrast
- cast nephropathy (multiple myeloma)
- vascular
- thrombotic-thrombocytopenic purpura/hemolyticuremic
Kidney Failure and Cancer

- syndrome (post-HCT, gemcitabine, mitomycin C)
- tumor infiltration (renal cell carcinoma with renal vein thrombosis)

Post-Renal
- intra tubular obstruction
- uric acid nephropathy
- methotrexate
- cast nephropathy (multiple myeloma)
- extra renal obstruction
- bladder outlet, ureteral (primary disease, retroperitoneal lymphadenopathy, retroperitoneal fibrosis)

Cancer of the Kidney
Most renal cancers have no symptoms early in their course. The most common presenting sign of a renal tumor is blood in the urine (hematuria). When a renal tumor is more advanced, there may be other signs such as weight loss, back or flank pain, fever, or loss or energy. A mass or lump in the abdomen may also be noted by a physician when the tumor is large. Several types of cancer can develop in the kidney, but the most common form of kidney cancer in adults is renal cell cancer (also called renal cell adenocarcinoma).

About 8300 adults die each year from kidney cancer. An estimated 18100 new cases are diagnosed each year, comprising 2% of all cancers. The average age at diagnosis is 55-60 years. Kidney cancer is the 8th most common cancer in men and the 10th most common cancer in women. Approximately 85% of
renal cell cancers are adenocarcinomas, for the most part of proximal tubular origin. Most of the remainder is transitional cell carcinomas of the renal pelvis

**Risk Factors**

While it is not known exactly what causes kidney cancer, researchers study patterns of cancer in the population to look for factors that are more common among people who develop cancer and those who do not. Following are some of the risk factors for kidney cancer:

- **Age**
  Risk increases with age, and most cases occur in people age 50-70.

- **Male Gender**
  Almost twice as many men develop kidney cancer than do women.

- **Race**
  More common among African American men than in white men.

- **Tobacco Use**
  Smokers are twice more likely to develop kidney cancer than nonsmokers. Risk increases the longer a person smokes.

- **Obesity**
  Obesity has been associated with a higher risk of kidney cancer among women. Reasons for possible link are unclear at this time.

- **Radiation**
  Women who have been treated with radiation therapy for disorders of the uterus have a slightly elevated risk of developing kidney cancer.

- **Dialysis**
  Patients on long-term use of dialysis to treat chronic kidney failure also experience an elevated risk.
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- **Von Hippel-Landau Disease (VHL)**
  Genetic disorder seems to predispose some people to developing kidney, as well as other types of, cancer.

**References**

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DIET FOR RENAL PATIENTS

Introduction

Diet is an important consideration for those with impaired kidney function. A kidney diet is one that is low in sodium, phosphorous and protein. A kidney diet stresses the importance of consuming high-quality protein and limiting fluids. Some kidney diets may also call for limited potassium and calcium. Every person is different, and therefore, a dietician will work with each patient to come up with a kidney diet that is tailored to his or her needs.1-3

Fluid & Fluid Control

Kidneys help control the amount of fluid that leaves your body. If your kidney disease progresses, your kidneys may be unable to regulate the removal of fluid from your body and as a result your doctor may ask you to limit your fluid intake. Too much fluid may cause swelling, shortness of breath, or high blood pressure.

What exactly is a fluid? Fluids are any food that is liquid or anything that melts into a liquid. Examples of fluids include the following:

- Coffee • Tea • Sodas • Soups • Popsicles • Ice cream, sherbet
- Ice cubes • Gelatin
- Milk, liquid creamer • Water • Wine and beer

If your doctor advises you to decrease the amount of fluids you eat and drink each day these tips may help you.

- Drink only when thirsty. Do not drink out of habit or to be social
- Eat less salt so you will feel less thirsty
- Suck on ice chips. (Measure small units into a cup)
• Brush your teeth three to four times a day; this is to prevent your mouth from drying out
• Suck on a lemon wedge
• If you have diabetes, control your blood sugar
• Chew sugarless gum or suck on sugarless hard candy
• Take your medications with sips of fluid
• When dining out, ask your beverage to be served in a child-size glass
• Measure how much fluid your favorite cup or glass holds so you will be better able to monitor the amount of fluid you drink
• After measuring out the total amount of fluid you can drink for the day, place the water in a container. During the day drink only from this container so you can keep an eye on the amount of fluid you have consumed.

Phosphorus

Phosphorus is a mineral that works with calcium to keep your bones healthy and strong. Phosphorus is needed by the body for building and maintaining bones and teeth and for normal nerve and muscle function. When kidney function declines, the body has a difficult time keeping phosphorus and calcium in balance. As a result of this imbalance, the body cannot get rid of excess phosphorus (phosphorus levels increase) and the body cannot take in enough calcium (calcium levels decrease). To try and correct this imbalance the body will “steal” calcium from the bones, which makes the bones weak. Problems associated with high phosphorus levels include itchy skin, bone and joint pain, and brittle bones.

Foods that are high in phosphorus include:
• Cola Drinks • Peanut Butter • Cheese • Sardines
• Chicken/beef liver • Nuts • Caramels • Beer • Ice Cream

Lower Phosphorus Food Substitutes Include
• Broccoli • Non-dairy milk substitute • Sherbet • Non-cola soda
• Zucchini squash • Hard Candy

A large serving size of a low phosphorus food can become a high phosphorus food.
If your phosphorus level remains high your doctor may prescribe a phosphate binder for you to take. This medication will bind with the phosphorus in the food you eat and prevent phosphorus from being absorbed in the body.

It is important that you take this medication exactly as instructed by your doctor.

**Potassium**

Potassium helps to keep your nerves and muscles, especially your heart, working properly. Potassium is a mineral and can be found in many foods. The kidneys are responsible for helping to keep the correct amount of potassium in your body. It can be very dangerous if your potassium level is too high. Too much potassium can make your heart beat irregularly or even stop without warning.

**Foods that are High in Potassium Include the Following**

**Fruits:** Bananas, Oranges, Apricots

**Vegetables:** Broccoli, Potatoes, Tomatoes, Mushrooms

**Other Food:** Chocolate, Coffee (limit to 2 cups per day), Salt Substitute, Bran & bran products, Nuts & dried fruit

**Low-Potassium Foods Include the Following**

**Fruits:** Apples, Grapes, Pears, Watermelon, Cranberries, Cherries

**Vegetables:** Beans (green or wax), Cucumber, Onions, Lettuce, Carrots

**Other Food:** Rice, Noodles, Cake, Cereal, Bread & bread products

It is important to remember that almost all foods contain potassium. Serving size will determine whether foods are a low, moderate, or high potassium level. A large serving size of a low potassium food can become a high potassium food.
Protein
Diet plays an important role in the management of kidney disease. The diet your physician will ask you to follow will be based upon your level of kidney function, your body size, and any other medical conditions you may have. Your diet may be helpful in delaying the need for dialysis.
Protein is needed to maintain muscles, aid in building resistance to infections, and repair and replace body tissue.
As your body breaks down protein foods, waste products called urea are formed. As kidney function declines, urea builds up in the bloodstream.
Eating too much protein may cause urea to build up more quickly. This will make you feel sick.
Eating less protein may be helpful in reducing your blood urea levels.
Reducing protein intake must be monitored by your doctor and dietician.

Examples of Foods High in Protein are
• Meat • Poultry • Milk Products • Eggs

Foods Low in Protein Includes the Following
• Fresh beans (pinto, kidney, navy) • Grains • Vegetables
You need both high quality and low quality protein in your diet. Your physician will determine how much protein should be in your diet.

Sodium
Before making any changes to your diet, make sure you discuss them with your doctor or dietician.
Sodium is needed by the body for many functions such as controlling muscle contractions, balancing fluids, and controlling blood pressure. Healthy kidneys remove excess sodium in the urine. As kidney function declines, sodium and fluids may accumulate in your body. Fluid retention may cause swelling in your eyes, hands, and/or ankles. To keep your sodium level in balance, your doctor may ask you to limit the sodium in your diet.
Foods high in sodium include the following:
Diet for Renal Patients

- Table salt • Bouillon cubes • Potato chips • Nuts • Bacon • Cold Cuts • Cheese • Canned, dehydrated, or instant soup • Canned vegetables • Processed dinner mixes (such as Hamburger Helper)

Low Sodium Alternatives
- Season with a variety of spices like garlic and oregano
- Use lemon

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Introduction

Regular exercise can improve the heart function, increase the hematocrit, and hemoglobin levels, improve the glucose control, and decrease the blood pressure. Improved exercise tolerance can improve the overall physical well-being and lower the level of anxiety and stress. These changes occur at different rates in different individuals, depending on their age, health, type and frequency of exercise and fitness level.\(^1\)

Exercise Benefits for People with Chronic Kidney Disease

Proper exercise can help the patients with chronic kidney disease a lot and provide them many health benefits.

Increased Energy

Fatigue is commonly seen in people with chronic kidney disease. If the patients can exercise regularly, over a period, they will develop greater resistance to fatigue and their energy will increase.

Improve Immunity

Some chronic kidney disease patients have to take immunosuppressive drugs over time, which can weaken their immunity significantly over time. Proper exercise has an effective function in enhancing immunity so your body is better to equipped to fight off diseases.

Help Deal with Stress

Long-term chronic kidney disease can cause much psychological pressure on patients. Exercise can relieve tension, which enables the patients to relax and feel less tense.
Lower High Blood Pressure
As exercise can improve oxygen transportation and circulation around the body, the heart can become stronger and blood pressure is lowered. Therefore, it can lower the risk of cardiovascular diseases.

Types and Intensity of Exercises to Help Kidney Disease
There are basically three types or classes of exercises: resistance, skills-based, and aerobics. Of these three, the best type of exercise to help kidney disease is probably aerobic exercise. Let us look at the main benefits of each type.

Resistance exercise increases muscular strength and endurance. It requires resistance through the use of body weight, free weights, or exercise machines. While this is not considered a primary exercise to help kidney disease, it is important in maintaining a strong and erect posture. Resistance exercise not only increases muscular strength but it also tones muscles and helps to maintain bone density (especially in elderly persons).

You do not necessarily need free weights or an exercise machine to get started with resistance exercises. Simple push-ups, pull-ups, sit-ups, and squats are good forms of resistance exercises. Start with five (5) to fifteen (15) minutes, two to three times per week. Over time, you will experience increased muscular strength and endurance, and your overall physical appearance will improve.

Skills-based exercise deals with improving coordination, balance, flexibility, speed, etc. This is probably the least needed exercise to help kidney disease. It is not, however, entirely useless. Flexibility, in particular, can be incorporated as a part of your exercise program. Any exercise program should start with a warm-up and end with a warm-down. Stretching (which facilitates flexibility) is a good means of warming-up and warming-down. This keeps muscles slender and toned, and helps to prevent damage to the muscles. You only need to stretch for about five minutes or so, before and after each exercise routine.
Exercise For Renal Failure Patients

Aerobic exercise is continuous and incorporates the use large muscle groups that keep the heart rate elevated. This is the best type of exercise to help kidney disease and includes walking, jogging, cycling, dancing, swimming, etc. Aerobic exercise helps to improve overall quality of life. Some benefits of aerobic exercise include:

- Weight control
- Lowers resting heart rate thereby, reducing the work load of the heart
- Lowers blood pressure and therefore minimizing the risk of kidney failure
- Reduces cholesterol
- Improves immune system functioning
- Increases insulin sensitivity to help prevent type II diabetes, an underlying cause of kidney failure.
- Significantly improves cardiovascular disease conditions

These are just some of the major benefits of aerobic exercise. It is important to note that aerobic exercises should be done in moderation, especially if you are just getting started. If you do too much, too intensely, you can overwork your system and organs leading to serious problems, including heart failure.

Frequency of Exercise

Exercise should be a minimum of three days per week. Many hemodialysis patients prefer to exercise on the days they are off dialysis.

Duration of Exercise

During the first week, exercises five minutes each session, then add one or two minutes per session each week until the patient gradually work up to one half hour. At the beginning, a short walk twice a day might be very effective. For weight control and increased benefits, the duration of walk has to be increased.
Intensity of Exercise
The guidelines for intensity of exercise to be done are as follows:
- The breathing should not be so labored that it becomes difficult to talk.
- The person should feel completely recovered within an hour.
- The muscles movement will be felt, but there should be no soreness.
- Exercise should be done at a comfortable push level.
- Exercise should be done slowly at the beginning of each session to warm up, and then increase the pace. Decrease the pace to cool down towards the end of the session.

When to Exercise
- Schedule the exercise into the regular daily routine
- Exercise after one hour of taking a large meal
- There should be someone supportive to help during exercise if needed
- During hot weather, exercise should be done in the morning or evening.

When Not to Exercise
- Fever
- Changes in dialysis schedule
- Hot and humid weather
- Any orthopaedic problem that might worsen on exercise

When to Stop Exercise
- Excessive fatigue
- Shortness of breath
- Chest pains
- Irregular or rapid heartbeats
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ALLOPATHIC DRUGS FOR RENAL FAILURE PATIENTS

Introduction

Dialysis cannot replace all the bodily functions of the healthy kidneys. By staying on the renal diet and following fluid restrictions the dialysis treatment is facilitated in removing wastes and keeping the normal body's water content.

Besides going for dialysis treatments and following the dialysis diet, there are medications that help maintain the best quality of health for the longest possible time. The following are some of the medical problems that may occur when the kidneys no longer function and the medicines commonly prescribed for the particular problem. Patients responding positively to the medical treatment were found to have better survival rate.

1. Medical Problem: Anemia

Anemia occurs when a person has a low red blood cell count. Anemia causes: fatigue or tiredness, lack of energy for exercise, difficulty in concentrating and strain on the heart. Most people with chronic kidney disease and nearly all patients with end stage renal failure, who are on dialysis, have anemia, since the kidneys make and secrete the hormone erythropoietin. This hormone is responsible for keeping a normal red blood cell count. Most patients with anemia due to chronic kidney disease who are not yet on dialysis will receive it as an injection directly under the skin. Most patients with renal failure on hemodialysis will get the hormone during each treatment by intravenous
injection into the return dialysis tubing. Most peritoneal dialysis (PD) patients will get erythropoietin by injection directly under the skin. The erythropoietin hormone given to patients goes under the names of: Procrit®, Epogen®, Aranesp® or epoietin alpha.

**Treatment of Anemia: Iron**

For proper working of erythropoietin, iron needs to be present to make red blood cells. Without iron fewer red blood cells are made, they are smaller in size and cannot carry as much oxygen. Small amounts of red blood cell with their iron are lost during each dialysis session. If the iron is not replaced, eventually dialysis patients lack enough iron and erythropoietin does not work as well. Because of this, most dialysis patients need to receive iron.

Oral iron can be used, but frequently is not effective, because many people find it causes stomach pains and constipation. Sometimes the iron losses are too great to be replaced by oral iron. Many dialysis units now give small amounts of intravenous iron during hemodialysis. Regular blood tests will tell your doctor if you need iron therapy. There are three different types of intravenous iron and they go under the names of InFeD®, Ferrlecit® and Venofer®. Because InFeD® can cause severe (although very rare) allergic reactions, most dialysis units today will use either Ferrlecit® or Venofer® for iron replacement. With proper iron management and the use of erythropoietin, over 90% of patients can enjoy energy levels that come from having a normal red blood cell count.

2. **Medical Problem: Bone disease and calcifications**

People with chronic kidney disease and those on dialysis can experience loss of bone minerals, including calcium and phosphorus. The calcium and phosphorus can also mix together, get hard and buildup (form calcifications) in the small blood vessels of the feet, intestines and heart. This condition can lead
to amputations, abdominal pain, gangrene of the intestines and heart failure. The cause of bone disease and calcifications come about due to the mix of dietary calcium, phosphorus, vitamin D and a hormone called PTH (parathyroid hormone). PTH is secreted by four small glands located on the surface of the thyroid gland in the neck. With renal failure, the body gets from sunlight and food is inactive. When PTH levels rise, there is inflammation in the bones, plus calcium and phosphorus are lost out of the bones. Because of kidney failure, the kidneys can no longer get rid of the extra phosphorus that is in the blood. Dialysis removes only a little bit of phosphorus. High phosphorus levels plus calcium become solid in small blood vessels.

**Phosphorus Binders**

Preventing or reversing this process can be done through diet and medicines such as phosphorus binders. Even when patients limit foods that are high in phosphorus, they would still have a high phosphorus level if they didn’t take their phosphorus binders. The binders prevent the body from absorbing the phosphorus from the foods eaten.

**Calcium-Containing Binders**

Calcium-containing binders are effective in preventing phosphorus absorption by combining with the phosphorus in the intestinal tract. Calcium acetate, also called PhosLo®, is one commonly used phosphorus binder. There are many others, usually containing calcium carbonate. Even Tums®, which is a form of calcium carbonate, can be effective. Because most patients will require 3 to 6 pills/capsules with every meal, calcium absorption from these medicines can be significant enough to cause concern. Some of the calcium from these binders is absorbed into the bloodstream and might deposit in small blood vessels, causing organ damage. Over the past five
years another medicine called Renagel® (sevelamer) has been used as a phosphate binder. This medicine mixes with phosphorus in the intestinal tract, but does not contain calcium.

Both medicines are effective in lowering phosphorous levels, but they need to be taken with every meal and with snacks. An even newer medicine, Fosrenol® (lanthanum carbonate) has been approved for use. Like the other two medicines, it binds phosphorus in the intestinal tract and needs to be taken with every meal. Unlike the other two medicines it is designed to be a chewable tablet.

Active Vitamin D

Although limiting foods with phosphorus from the diet is very important, active vitamin D is necessary in maintaining normal PTH levels and in bone health. High PTH levels cause inflammation of bones, muscles and tendons, loss of bone calcium and phosphorus and may be the reason for severe itching in some dialysis patients.

The oral form of active vitamin D may be effective in preventing high PTH levels in patients with chronic kidney disease. Currently, the two most available medicines are Rocaltrol® (calcitriol) and Hectorol® (doxercalciferol). These oral medicines work better for those with chronic kidney disease who are not yet on dialysis than they do for dialysis patients. Therefore, these medicines are usually given to dialysis patients intravenously during hemodialysis.

A third, and more common medicine used intravenously is another form of active vitamin D called Zemplar® (paricalcitol). In many ways it is similar to the other two, but may decrease the tendency to cause high blood calcium levels when compared to calcitriol (called Calcijex® when given intravenously).

A new class of medicines called "calcimemetics" has been developed. One called Sensipar® (cinacalcet) is given orally and is highly effective in lowering PTH levels.

Vitamins and Minerals

The dialysis procedure removes large amounts of water-soluble vitamins, such as vitamin C, B-complex vitamins and
folic acid. While a good diet can usually keep up with these losses, many dialysis patients don’t always have an appetite. Most nephrologists feel that the use of a B-complex vitamin, along with folic acid is a good protection for when patients don’t have a good appetite. A few vitamins include: Nephro-Vite®, Nephrocaps® and Nephroplex®. These are commonly used, since they have been designed to replace losses specific to dialysis therapy.

3. Medical Problem: Itching
   Many dialysis patients have itching and dry skin. It is important to learn why and correct the cause.

   **Topical Hydrating Agents/ Topical Cortisone & Oral Antihistamines**
   The itching can frequently be treated with topical hydrating agents or topical cortisone along with oral antihistamines, such as Benadryl® (diphenhydramine), Atarax® or Vistaril® (hydroxyzine) or Zyrtec® (loratadine).

4. Medical Problem: Cramps
   Some patients are prone to leg cramps, not only on dialysis, but during the nighttime as well. This probably has to do with the rapid fluid and electrolyte shifts in and out of muscle cells from the hemodialysis treatment.

   **Quinine sulfate**
   Quinine sulfate has helped many patients as a preventative measure for cramps when taken either before dialysis or at bedtime.

5. Other Medical Problems & Their Treatment
   There are many other medications in use, which are not for dialysis itself but are related to the most common causes of kidney disease.
Patients with diabetes mellitus represent 40% of patients on dialysis. Their diabetes needs to be carefully controlled, not only with diet, but with either pills or insulin shots designed to maintain normal blood glucose.

Patients with high blood pressure (hypertension) need to be treated by establishing at an appropriate dry weight, following a low sodium diet and the use of high blood pressure medicines.

Since heart disease is common in dialysis patients many studies are now looking at medicines that can decrease the rate of heart disease. High homocysteine levels are commonly found in patients with heart disease and are also found in patients on dialysis. High doses of folic acid can lower homocysteine levels and high dose folic acid therapy is being evaluated as a possible preventative treatment in dialysis patients.

Likewise the HMG co-a reductase inhibitors, more commonly known as "statins" (Lipitor®, Zocor®, Pravachol®, and others) are being studied, even for patients on dialysis who don’t have high cholesterol, because these drugs seem to lower the rate of heart disease.1

References:
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RENAL REPLACEMENT THERAPY

Introduction

Patients who progress to ESRD require renal replacement therapy (RRT). The modalities that are used for RRT are dialysis, including HD and peritoneal dialysis (PD), and kidney transplantation. The United States Renal Data Service (USRDS) reported that the number of patients with ESRD was 452,957, with 102,567 new cases being diagnosed in 2003. The most common form of RRT is dialysis, accounting for 72% of all patients with ESRD 1.

Dialysis

Thomas Graham in 1854 coined the word “dialysis” for the observation that urea passed through parchment. This was followed, in 1913, by John Jacob Abel’s demonstration that salicylate could be removed from blood by using his hollow-fiber artificial kidney.22

Dialysis is the artificial process of getting rid of waste and unwanted water from the blood. This process is naturally done by our kidneys. Some people, however, may have failed or damaged kidneys which cannot carry out the function properly; they may need dialysis. In other words, dialysis is the artificial replacement for lost kidney functions (renal replacement therapy).

When to Start Dialysis

Current guidelines recommend commencing dialysis when:

• GFR < 15 ml/min with uremic symptoms (persistent nausea and vomiting, anorexia, malnutrition, volume overload, restless legs).
• GFR < 10 ml/min whether symptomatic or not.
• Diabetic patients should initiate dialysis at an earlier stage than patients with another cause of end-stage renal failure, i.e. GFR 10-15 ml/min, or creatinine > 500µmol/L, as diabetics are more susceptible to uremic symptoms, fluid retention and hyperkalemia.
• Refractory hypokalemia, acidosis, pulmonary edema, pericarditis, encephalopathy, and neuropathy are all (urgent) indications for dialysis (the aim should be to start dialysis before any of these are present).
• There is no clear evidence that an early start to dialysis confers a survival benefit.
• Pre-emptive transplantation is the treatment of choice of ESRD. Consider when GFR <20 ml/min\(^2\).

Dialysis may be used for people who have become ill and have acute kidney failure, or for fairly stable patients who have permanently lost kidney function.

When a person is healthy the kidneys regulates the body levels of water and minerals, and remove waste. The kidneys also produce erythropoietin and 1, 25 – di hydroxy cholecalciferol (calcitriol) as part of the endocrine system. Dialysis does not correct the endocrine functions of failed kidneys; it only replaces some kidney functions such as water and fluid removal\(^3\).

**Dialysis is not a Cure**

Hemodialysis and peritoneal dialysis are treatments that help to replace the work of the kidneys. These treatments help the patient feel better and live longer, but they don’t cure kidney failure. Although patients with kidney failure are now living longer than ever, over the years kidney disease can cause problems such as heart disease, bone disease, arthritis, nerve damage, infertility, and malnutrition. These problems won’t go away with dialysis, but doctors now have new and better ways to prevent or treat them.

There are two types of dialysis:
• Hemodialysis
• Peritoneal dialysis

Initiation of dialysis is dependent on the patient's clinical status. Symptoms that may indicate the need for dialysis include persistent anorexia, nausea, vomiting, fatigue, and pruritus. Other criteria that indicate the need for dialysis include declining nutritional status, declining serum albumin levels, uncontrolled hypertension, and volume overload, which may manifest as chronic heart failure, and electrolyte abnormalities, particularly
hyperkalemia. Blood urea nitrogen (BUN) and serum creatinine (SCr) levels may be used as a guide for the initiation of dialysis, but should not be the absolute indicator. Dialysis is initiated in most patients when the GFR falls below 15 ml/minute/1.73m². Patients should determine which modality of dialysis to use based on their own preferences.

**Hemodialysis**

Hemodialysis is the most common method used to treat advanced and permanent kidney failure. Since the 1960s, when hemodialysis first became a practical treatment for kidney failure, many researches have been carried out to make hemodialysis treatments more effective and minimize side effects.

In hemodialysis, an artificial kidney (hemodialyzer) is used to remove waste and extra chemicals and fluid from the blood. To get the blood into the artificial kidney, the doctor needs to make access into the blood vessels. This is done by minor surgery to the arm or leg.
Sometimes, an access is made by joining an artery to a vein under the skin to make a bigger blood vessel called a fistula. However, if blood vessels are not adequate for a fistula, then a soft plastic tube is used to join an artery and a vein under the skin. This is called a graft.

Occasionally, an access is made by means of a narrow plastic tube, called a catheter, which is inserted into a large vein in the neck. This type of access may be temporary, but is sometimes used for long-term treatment. Usually, each hemodialysis treatment lasts about four hours and is done three times per week. A type of hemodialysis called high-flux dialysis may take less time.

**Principles of Haemodialysis in Practice**

At its simplest, a dialysis machine simply pumps blood and dialysate through a dialyser.

- The dialysate is a solution of purified water, sodium, potassium, magnesium, calcium, chloride, dextrose, and bicarbonate or acetate.
- The blood and dialysate are kept separate within the dialyser by a semipermeable membrane. As the dialysate contains no waste products of metabolism (urea, creatinine, etc.), these will diffuse from blood into dialysate.
- Diffusion is maximized by maintaining high flow rates (of blood and dialysate), and by pumping the two solutions in opposite directions (countercurrent flow).
- Convective clearance can be added by generating a TMP within the dialyser. CRRT rely mainly on convective mechanisms for solute removal, and often do not have any dialysate at all. In conventional HD, small MW molecules are not removed to any great extent by convection, but almost entirely by diffusion. In contrast large MW molecules (e.g. β2-microglobulin or vitamin B12) are removed more effectively by convection than diffusion. This has led to an increasing use of UF methods in HD to increase removal of
larger MW molecules [hemodiafiltration (HDF) or high volume hemo filtration].

A HD machine is made more complex by the addition of a number of safety devices, pump controllers, pressure and flow monitors, air leak detectors, patient BP monitors, the ability to change the composition of the dialysate, and increasingly systems to monitor blood chemistry, access flow, and delivered dialysis dose, and provide data to remote controllers and databases.

Effects of Hemodialysis on Lifestyle

The following are the generally observed effects in patients undergoing hemodialysis.

- **Flexibility:** Difficult to fit in with school; work especially if unit is far from home. Home HD offers more flexibility
- **Travel:** Necessity to book in advance with HD unit of places of travel
- **Responsibility & Independence:** Home HD allows the greatest degree of independence
- **Sexual Activity:** Anxiety of living with renal failure affects relationship with partner
- **Sport & Exercise:** Can exercise and participate in most sports
- **Body Image:** Especially with fistula; patient can be very self-conscious about it

Problems with Hemodialysis

Problems associated with hemodialysis are listed below:

- **Rapid changes in BP:** fainting, vomiting, cramps, chest pain, irritability, fatigue, temporary loss of vision
- **Fluid overload:** especially in between sessions
- **Fluid restrictions:** more stringent with HD than PD
- **Hyperkalaemia:** especially in between sessions
- **Loss of independence**
- **Problems with access:** poor quality, blockage etc. Infection (vascular access catheters)
- **Pain with needles**
- **Bleeding:** from the fistula during or after dialysis
• **Infections:** during sessions; exit site infections; blood-borne viruses e.g. Hepatitis, HIV

**Peritoneal Dialysis**

In this type of dialysis, the blood is cleaned inside the body. Minor surgery will have to be performed to place a plastic tube called a catheter into the abdomen to make an access. During the treatment, the abdominal area (called the peritoneal cavity) is slowly filled with dialysate through the catheter. The blood stays in the arteries and veins that line the peritoneal cavity. Extra fluid and waste products are drawn out of the blood and into the dialysate.

There are several kinds of peritoneal dialysis but two major kinds of peritoneal dialysis are:

- Continuous Ambulatory Peritoneal Dialysis (CAPD)
- Continuous Cycling Peritoneal Dialysis (CCPD)

**Principles of Peritoneal Dialysis**

In PD, solute and fluid exchange occur between peritoneal capillary blood and dialysis solution in the peritoneal cavity. The membrane lining this cavity consists of a vascular wall, interstitium, mesothelium, and adjacent fluid films. Small molecular weight solute transfer occurs by diffusion, i.e. down a concentration gradient. The longer dialysate is allowed to dwell in the peritoneum, the more a given solute will pass from blood into dialysate, thereby reducing the concentration.
gradient and hence its rate of passage. The concentration gradient can also be from dialysate to blood.

**Fluid Movement**

In PD this is determined by osmosis. Fluid will move across the peritoneal membrane from the compartment with the lower to that with the higher osmotic pressure. Fluid removal is achieved by increasing the osmotic pressure within dialysate, usually by increasing the dialysate dextrose concentration. Movement of fluid induces a movement of solutes by convection or a solvent drag in the absence of a concentration gradient. This is important for middle-sized molecular weight solute transfer.

**Continuous Ambulatory Peritoneal Dialysis (CAPD)**

It is the only type of peritoneal dialysis that is done without machines. A bag of dialysate has to be inserted in peritoneal cavity through the catheter. The dialysate stays there for about 4-5 hours before it is drained back into the bag and thrown away. This is called an exchange. While the dialysate is in the peritoneal cavity, a person can carry out usual activities at work and home.

**CAPD Exchange**

![CAPD Exchange Diagram]

**Continuous Cycling Peritoneal Dialysis (CCPD)**

It is usually done at home using a special machine called a cycler. Each cycle usually lasts 1-1/2 hours and exchanges are done throughout the night while you sleep.³
Lifestyle Changes with Peritoneal Dialysis

- **Flexibility**
  Peritoneal dialysis can be performed almost anywhere and have least impact on work / school life (especially APD).

- **Travel**
  Dialysis supplies can be delivered to most parts of the world; travel more flexible. APD machines are portable; will fit into a car boot, can be carried by train/air.

- **Responsibility**
  Peritoneal dialysis requires more responsibility from patient but more independence.

- **Sports/Exercise**
  Most of the sport and exercise activities can be done. Advice on swimming, lifting, contact sports.

- **Sexual Activity**
  Peritoneal dialysis may affect relations based on patient anxiety.

- **Delivery & Storage of Supplies**
  Space is required for storing boxes, containing peritoneal dialysis supplies; as well as specially recruited and trained delivery staff is also mandatory.

### Problems with Peritoneal Dialysis Treatment

**Monotomy of treatment**
- The treatment never goes away against days off with HD

**Body Image Problems**
- Especially with a permanent catheter
- Abdominal stretching

**Fluid Overload**
- Much less a problem than with HD

**Dehydration**
- Less common than fluid overload

**Abdominal Discomfort**
- Bloated feeling

**Poor drainage**
- Common problem esp with new patients
- Fibrin plug
- Catheter displacement
Leakage
- Fluid may leak around catheter exit site. (May leak into scrotum)
- Stop PD temporarily
- Resite catheter (use new one)

Infections
- Exit site infections
- Tunnel infection
- Peritonitis

Hernia
- Aggravation of pre-existing herniae (repair)
- Evolution of new herniae

Declining effectiveness of the peritoneum
- Example is repeated infection
- Effect of glucose in the dialysis fluid

Complications of Dialysis
Dialysis cannot correct all the side effects of kidney failure. Some of these must be considered in the precautions of exercise.
- Osteodystrophy
- Muscle Weakness
- Autonomic Neuropathy
- Hypertension
- Hyperlipidemia

Clinical Outcomes of Haemodialysis and Peritoneal Dialysis—Choosing the Right Modality for Individual Patient
The overall mortality rate of peritoneal dialysis (PD) and hemodialysis (HD) has considerably improved during the past two decades. The rate of PD mortality, in particular, has fallen more rapidly compared to HD due to major improvements in PD delivery, efficacy, and safety resulting in a similar adjusted five-year survival in HD and PD patients at 33.5% and 33.9%, respectively. While both modalities have the same five-year survival, several recent studies seem to indicate that PD is associated with better survival during the first 1-2 years of dialysis, whereas HD is associated with better survival thereafter. In addition to these general findings, individual patient outcome is determined by patient characteristics that affects PD and HD
mortality. Age, gender, ethnicity, race, body mass index, educational background, health literacy, and the patient’s comorbid conditions are patient-specific variables that influence outcome on HD or PD treatment. Choosing the right patient for the right modality has been shown to have a great impact not only on health-related outcomes but on quality of life. Thus, it is important to consider the social implications of each modality on the patient’s life. Independent from patient characteristics and preference, the quality of general and nephrological care will affect the outcomes of dialysis. Clinical knowledge and training have historically focused more on center HD than PD. Many nephrology training programs do not offer trainees sufficient exposure to home therapies. Accordingly, center experience has shown to significantly impact the relative risk of death and technique failure in PD. Thus, adequate training and education of patients and health care professionals, together with thoroughly considering the impact of patient characteristics and patient preference is the key to choosing dialysis modality.

References
Kidney Transplantation

CHAPTER 15

KIDNEY TRANSPLANTATION

Introduction

Kidney transplantation is a procedure that places a healthy kidney from another person into the renal failure patient’s body. Usually the failed kidneys are left in place, but sometimes removed. The transplanted kidneys take over the work of two failed kidneys and a person no longer need dialysis.

During a transplant, the surgeon places the new kidney in the lower abdomen and connects the artery and vein of the new kidney with the artery and vein of the person, who is undergoing transplantation.

The renal failure patient may receive a kidney from a member of their family. This kind of donor is called a living-related donor. A patient may receive a kidney from a person who has recently died. This type of donor is called a cadaver donor. Sometimes a spouse or very close friend may donate a kidney. This kind of donor is called a living-unrelated donor.

The time it takes to get a kidney varies. There are not enough cadaver donors for every person who needs a transplant. Because of this, the patient must be placed on a waiting list to receive a cadaver donor kidney. However, if a patient’s relative gives him/her a kidney; the transplant operation can be done sooner.

The transplantation process has many steps. First, patient should talk with the doctor because transplantation isn’t for everyone. The patient could have a condition that would make transplantation dangerous or unlikely to succeed.
It is very important for the donor's blood and tissues to closely match that of renal failure patient. This match will help prevent patient's body's immune system from fighting off, or rejecting, the new kidney. The transplant team considers three factors in matching kidneys with potential recipients.

- **Blood type.** The patient’s blood type (A, B, AB, or O) must be compatible with the donor's. Blood type is the most important matching factor.

- **Human leukocyte antigens (HLAs).** The patient’s cells carry six important HLAs, three inherited from each parent. Family members are most likely to have a complete match. He/she may still receive a kidney if the HLAs aren't a complete match as long as your blood type is compatible with the organ donor's and other tests show no problems with matching.

- **Cross-matching antigens.** The last test before implanting an organ is the cross-match. A small sample of the patient’s blood will be mixed with a sample of the organ donor's blood in a tube to see if there's a reaction. If no reaction occurs, the result is called a negative cross-match, and the transplant operation can proceed.

The surgery takes from 3 to 6 hours. The usual hospital stay may last from 10 to 14 days. After that patient may leave the hospital, then he/she will go to the clinic for regular followup visits.

In a living donation, the donor will probably stay in the hospital about the same amount of time. However, a new technique for removing a kidney for donation uses a smaller incision and may make it possible for the donor to leave the hospital in 2 to 3 days.

Between 85 and 90 percent of transplants from deceased donors are working 1 year after surgery. Transplants from living relatives often work better than transplants from unrelated or deceased donors because they’re usually a closer match.

**Possible Complications**

Transplantation is the closest thing to a cure. But no matter how good the match, the renal failure patient’s body may reject the new kidney. The chance of his/her body accepting the new
Kidney Transplantation

Kidney depends on their age, race, and medical condition. A common cause of rejection is not taking medication as prescribed.

Normally, 75 to 80 percent of transplants from cadaver donors are working one year after surgery. However, transplants from living relatives often work better than transplants from cadaver donors. This fact is because they are usually a closer match.

Diet for transplant patients is less limiting than it is for dialysis patients. The patient may still have to cut back on some foods, though. The patient’s diet probably will change as their medicines, blood values, weight, and blood pressure change.

- The renal failure patient may need to count calories. The medicine may give him/her a bigger appetite and cause them to gain weight.
- The renal failure patient may have to limit eating salty foods. Their medications may cause salt to be held in their body, leading to high blood pressure.
- The renal failure patient may need to eat less protein. Some medications cause a higher level of wastes to build up in his/her bloodstream.

The doctor will give the patient with kidney transplant medicines called immune-suppressants to help prevent their body's immune system from attacking the kidney, a process called rejection. The patient needs to take immune-suppressants every day for as long as the transplanted kidney is functioning. Sometimes, however, even these medicines can't stop their body from rejecting the new kidney. If this happens, the patient goes back to some form of dialysis and possibly waits for another transplant.

Immuno-suppressants weaken the immune system, which can lead to infections. Some medicines may also change the appearance. The face may get fuller; the patient may gain weight or develop acne or facial hair. Not all patients have these problems, though, and diet and makeup can help.

Immuno-suppressants work by diminishing the ability of immune cells to function. In some patients, over long periods of time, this diminished immunity can increase the risk of developing cancer. Some immune-suppressants can cause
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cataracts, diabetes, extra stomach acid, high blood pressure, and bone disease. When used over time, these drugs may also cause liver or kidney damage in a few patients.

Advantages and Disadvantages of Kidney Transplantation

A discussion of the advantages and disadvantages of transplantation is an important part of the decision whether or not to consider a transplant.

Advantages

Some advantages that have been found by persons who have had a successful transplant are:
- Freedom from dialysis and freedom from the time commitment that it requires.
- Increased energy.
- Fewer, if any, diet and fluid restrictions.
- Feeling better physically.
- A better quality of life.
- No longer seeing themselves as chronically ill.

Disadvantages

Some disadvantages to a kidney transplant have been found to be:
- Needing to take medicines for the life of the kidney.
- Needing to undergo a surgical procedure under general anesthesia.
- Side effects of the medicines (see section on medications).
- Potential for problems related to the surgery and/or anesthetic.

Evaluation of Potential Recipients

After thinking about the advantages and disadvantages, the next step is the decision by the physicians as to whether or not a transplant is a good choice for the renal failure patient. The decision will be based upon a thorough medical history and physical examination. It will include a chest X-ray, an EKG or ECG, abdominal ultrasound, and echocardiogram. Bladder tests may be done in order to see how well the patient’s bladder functions. Additional testing will be scheduled based on the
Kidney Transplantation

patient’s individual medical needs. The patient’s own kidneys may need to be removed only if medically necessary.

A psychiatric evaluation may be done in order to determine the patient’s understanding of the benefits and risks as they were explained to him/her. It also helps to look at how he/she might react to the transplanted kidney or the medications.

Tissue typing is done during the evaluation. This involves taking a sample of blood and analyzing it for markers on the white blood cells (antigens) that are used to match a donated kidney with a potential recipient.

Evaluation for transplant includes a discussion about the patient's family and whether or not there is a person (related or not) who could donate a kidney. This is called a living donor kidney transplant. The other source of a kidney is a person who has recently died and whose family has consented to kidney donation. This is called a cadaver kidney transplant. Tissue typing and matching for both types of transplants will be discussed in a later section.

Evaluation of the patient's support system is also done. It is important that transplant patients have reliable transportation at the time of transplantation, as well as for clinic follow-ups. Also, family or other support systems may be necessary to ensure the proper taking of medications, home testing, or other situations which may arise post-operatively.

Tissue Typing

Tissue typing, also called HLA typing, is a process of identifying genetic markers (antigens) on white blood cells. In the laboratory, these markers can be specifically identified. Each person inherits his antigens from his or her parents. Certain antigens are used in choosing potential recipients and donated kidneys. In order to carry out tissue typing, a blood sample is drawn and the genetic markers on the blood cells are identified. Each marker has a letter and number in its name. For example, two common antigens are known as HLA-A1 and HLA-B8. Over 100 of these antigens have been identified. Some of the antigens are more common than others.

In a family with the same mother and father, children can inherit various combinations of antigens from the parents. Each
child inherits 1/2 of his/her antigens from each parent. Four combinations of inherited antigens are possible. This means there is a one out of four, or a 25%, chance for an identical match between brothers and sisters as well as a 25% chance for no match at all. There is a two out of four, or 50%, chance that there will be a 3 antigen match between brothers and sisters.

A 6 antigen match is the best possible match for kidney transplants, but lesser matches are used and have successful outcomes.

If a potential transplant candidate has someone interested in donating a kidney, tissue typing is done to see how closely his/her antigens match the recipient. Other blood tests (including the cross-match) will be done to determine if the pair is compatible. If the transplant candidate does not have someone able to donate a kidney, the antigens identified in the recipient are used to match with a donated cadaver kidney.

However, it must be clearly understood that drawing blood for tissue typing and obtaining blood samples routinely for antibody screens does not mean that someone is automatically placed on the list for a cadaver kidney. No one's name is placed on the list in the computer unless it is known that he/she is ready to become an active candidate and have his/her name placed on the list. At that time, it is necessary to talk with the transplant coordinator and give her the necessary phone numbers.

When on the list, it is necessary to let the coordinators know if donor is treated for any infections or are hospitalized for any reason. A transplant cannot be performed under these conditions and it is necessary to put that donor’s name on hold until he/she is well again.

**Blood Type Compatibility**

In addition to matching donated kidneys to recipients by tissue typing, blood types must also be compatible. Blood types are identified as A, B, AB, and O. The most common type is O. Fifty percent of the population has blood type O. The next most common is blood type A with 40% of the population having this type. Only a small percent of the population has blood types B or AB.

It can be seen from the table below of blood type compatibility that a kidney from an O donor can be transplanted
into a person with any blood type. A kidney from an AB donor can only go to an AB recipient. The person with AB blood can receive a kidney from a person with any blood type. Various other combinations are possible for persons with A or B blood. It is not necessary to match the positive and negative signs that are a part of a person's blood type. However, there are federal regulations that govern matching for cadaver kidneys that outline the combinations that can be used. These regulations do not apply to living donor transplants.

### BLOOD TYPE COMPATIBILITY

<table>
<thead>
<tr>
<th>Donor can be type:</th>
<th>If the blood type of the recipient is:</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>O or A or B or AB</td>
</tr>
<tr>
<td>A</td>
<td>A or AB</td>
</tr>
<tr>
<td>B</td>
<td>B or AB</td>
</tr>
<tr>
<td>AB</td>
<td>AB</td>
</tr>
</tbody>
</table>

### Matching Kidneys to Recipients

Matching donated kidneys to recipients is based entirely upon blood group compatibility and antigen matching, not upon age, race, or sex. Because of many variables involved in receiving a kidney, it is possible that some people may wait for a kidney a short time after going on the list, while others may wait a long time.

Because the time it takes to obtain a cadaver kidney for a recipient is unpredictable, hemodialysis or peritoneal dialysis may be necessary during the waiting time. Even though a living donor transplant may be planned, dialysis may still be needed. Transplant and dialysis are options that work together to treat end-stage kidney disease.

There are three ways that you can receive a kidney for transplant.

- A living related kidney can come from a brother, sister, parent, aunt, uncle, or cousin.
- A living non-related kidney can come from a husband, wife, friend, or extended family.
- A (cadaver) deceased donor kidney come from a person who has recently died and had asked to donate the kidney after death.
The healthy kidney is transported in cool salt water (saline) that preserves the organ for 48 hours. This gives the health care providers time to perform tests that match the donor's and recipient's blood and tissue before the operation.

Procedure for a living kidney donor

If you are donating a kidney, you will be placed under general anesthesia before surgery. The procedure used to require a long surgical cut. However, nowadays short surgical cut (mini-nephrectomy) or laparoscopic techniques are available to perform kidney transplantation.

Procedure for kidney recipient

People receiving a kidney transplant are given general anesthesia before surgery. The surgeon makes a cut in the lower belly area. Kidney transplant surgery takes about 3 hours.

Between 85 and 90 percent of transplants from deceased donors are working 1 year after surgery. Transplants from living relatives often work better than transplants from unrelated or deceased donors because they’re usually a closer match.

To minimize the risk of kidney rejection, the patients with transplanted kidney needs to take immune suppressive drugs.

The immune suppressants help prevent your body’s immune system from attacking the kidney, a process called rejection. A patient will need to take immune suppressants every day for as long as the transplanted kidney is functioning.

Immuno suppressants weaken the immune system, which can lead to infections. Some medicines may also change the appearance. The face may get fuller; the patient may gain weight or develop acne or facial hair. Not all patients have these problems, though, and diet and makeup can help.

Immuno suppressants work by diminishing the ability of immune cells to function. In some patients, over long periods of time, this diminished immunity can increase the risk of developing cancer. Some immune suppressants can cause cataracts, diabetes, extra stomach acid, high blood pressure, and bone disease. When used over time, these drugs may also cause liver or kidney damage in a few patients.

The following three factors have to be considered to predict that whether the recipient's immune system will either accept or reject the new kidney.
Kidney Transplantation

1. **Blood type.** The blood type (A, B, AB, or O) must be compatible with the donor’s. Blood type is the most important matching factor.

2. **Human leukocyte antigens (HLAs).** The cells carry six important HLAs, three inherited from each parent. Family members are most likely to have a complete match. The patient may still receive a kidney if the HLAs aren’t a complete match as long as your blood type is compatible with the organ donor’s and other tests show no problems with matching.

3. **Cross-matching antigens.** The last test before implanting an organ is the cross-match. A small sample of the blood will be mixed with a sample of the organ donor’s blood in a tube to see if there’s a reaction. If no reaction occurs, the result is called a negative cross-match, and the transplant operation can proceed.\(^2,3\)

### Advantages of Kidney Transplantation Over Dialysis in End-Stage Renal Disease Patients

- A transplant diet is less limited than a dialysis diet.
- Many medical problems like anemia, high blood pressure, heart problems, and nerve damage often improve after a transplant.\(^4\)

### References

CHAPTER 16

HRQOL Outcomes of CKD Support

Physical functional
- Physical Activity
- Fatigue/Sleep
- Pain/Symptoms
- Diet and appetite

Psychological
- Stress, anxiety, and depression
- Subclinical distress
- Cognitive functioning

Social
- Family relationships
- Vocational role
- Relationship stability
- Sexual activity/satisfaction

Global QOL
- Perceived health
- Daily functioning
- Happiness
- Satisfaction
- Spirituality

CKD treatment

Physical functional

Psychological

Social

Global QOL
HEALTH-RELATED QUALITY OF LIFE INSTRUMENT

Introduction

HRQOL refers to the subjective perception of the effect of a disease or its treatment on one’s health and overall QOL. It includes physical, psychological, and social dimensions of health as assessed by the patient. It is clearly influenced by the individual’s beliefs, life experiences, personality, and expectations. Emphasizing the inherently subjective nature of HRQOL is important. The physical dimensions of health (e.g., disabilities, impaired physical strength) can be assessed “objectively” through either healthcare personnel or different instruments. These measurements provide information about the patient’s “health status” or “functioning.” HRQOL, on the other hand, assesses how the presence of the disease’s physical symptoms, such as impairment of physical functioning and reduced stamina, affect one’s overall well-being, life satisfaction, or QOL. This means that two individuals with either similar physical health or equal severity of the disease could have vastly different HRQOL.

Evidence accumulated over the last 10–15 years has clearly demonstrated that HRQOL measurements correlate with “objective” measures of physical health and predict traditional “hard outcomes” (i.e., hospitalization and mortality). They also add additional information to the assessment of the overall well-being of patients with chronic medical conditions.

In order to understand the relationship among the disease, its treatment, and HRQOL, the concept of illness intrusiveness must be understood. Illness intrusiveness was introduced to represent illness-induced disruptions to lifestyles, activities, and interests that compromise QOL. Conceptualized as a facet of the chronic
disease experience common across conditions, illness intrusiveness is a fundamental determinant of HRQOL. The central hypothesis is that disease (ie, pain, fatigue, disability) and treatment factors (ie, time required for treatment, untoward side effects) indirectly influence subjective well-being and HRQOL through their effects on illness intrusiveness. For example, depriving the individual of the gratifying consequences of psychologically meaningful activities could affect the patient’s HRQOL. Psychological and social factors act as moderator variables that influence both the magnitude of illness intrusiveness, which is occasioned by disease and treatment factors, and the degree to which illness intrusiveness compromises QOL.\(^6\)\(^7\)

The Kidney Disease Quality of Life Questionnaire–Short Form (KDQOL-SF) has become the most widely used QOL measure for CKD patients. It is a self-report tool that includes the Medical Outcomes Study Short Form-36 generic core and several multi-item scales targeted at QOL concerns of special relevance for patients with CKD.

**Significance and Utilization of Health-Related Quality of Life Data**

In addition to the traditional “hard outcome measures” (ie, mortality, morbidity, hospitalization), HRQOL has been recognized as both an equally important aspect of healthcare delivery and a measure of treatment effectiveness and quality in chronic medical conditions, CKD in particular. ESRD is a life-threatening disease that leads to numerous and severe symptoms and complications. These severe comorbid conditions will have a major impact on the affected patients’ HRQOL. RRTs are able to alleviate, but they are very intrusive and cure neither the disease nor its symptoms. Patients suffering from ESRD need RRTs to survive, but they also expect to achieve a certain level of well-being. In industrialized countries achieving survival is not enough for a treatment to be considered “successful” unless it also yields an appreciable gain in HRQOL.\(^8\)\(^9\)

An increasing number of professionals feel that HRQOL assessment is essential to evaluating quality and effectiveness of CKD patient care, comparing alternative treatments and RRT
modalities, improving clinical outcomes, facilitating complex rehabilitation of CKD patients, and enhancing patient satisfaction. Several authors have suggested that regular HRQOL monitoring become part of regular CKD patient assessment and incorporated into the continuous quality assurance and quality improvement systems.  

### Potential Clinical Application of Health-Related Quality of Life Assessment

Currently, HRQOL is used almost exclusively in clinical studies. At the same time, the nephrology community is increasingly realizing the potential importance of HRQOL assessment in the clinical care of its patients. As discussed above, HRQOL scores have consistently predicted morbidity, hospitalization, and mortality independent of other clinical parameters. In addition, HRQOL scores provide additional information on the individual’s well-being beyond the information gained from the patient’s clinical and laboratory assessments. In this respect, these scores are similar to serum albumin, an important predictor of both morbidity and mortality that is widely used to assess and characterize the patient’s overall condition. A declining serum albumin level is viewed as an alarming signal that could trigger a decision to start RRT in a patient with marginal renal function. HRQOL scores are steadily being interpreted in a similar way, and they could similarly serve to inform clinical decisions. Overall, HRQOL measurements should be part of the quality culture in the management of CKD patients.

### Clinical Practice Guidelines for Renal Patients

National and International standards or clinical practice guidelines outlining the expected level care for renal patients have been set out by the Renal Association (in the UK), The Kidney Foundation Dialysis Outcomes Quality Initiative (K/DOQI) in the US, and the European Best Practice Guidelines. These guidelines cover (among other things):

- Pre-dialysis care
- Dialysis prescription and monitoring
- Vascular access preparation and care
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- Anemia management
- Nutrition
- Management of renal bone disease
- Cardiovascular risk factor management
- Management of infection in dialysis patients

Research Recommendation

Therefore, large-scale longitudinal studies are needed to evaluate the relationship between GFR and all domains of functional status and well-being throughout the course of progression of kidney disease. More research should be undertaken using the recommended standardized instruments and their outcomes compared. Whenever specific medications could affect outcomes, usage should be assessed. Because conditions such as anemia, bone disease, cardiovascular, disease, and diabetes can affect functioning and well-being, researchers need to study whether appropriate management of these conditions improves functioning and well-being. Finally, researchers need to examine the effectiveness of rehabilitation interventions in earlier stages of chronic kidney disease. Doing so could provide further scientific evidence for the relationship of kidney function and treatment on patients' risk of dysfunction, hospitalization, and death and increase understanding of what interventions improve functioning and well-being and reduce the burden of chronic kidney disease on the patient, his or her family, and society.

References

CHAPTER 17

HOMEOPATHIC SYSTEM OF TREATMENT

Introduction

Homeopathy does not recognize kidneys as a mere organ of excretion or selective filtration but always recognizes it in relation to the individual as a whole. Kidneys have a generalized function – the fluid coming to it and going from it influence every organ, tissue and cell of our body. Kidney function influences the complete vital economy of the body. Homeopathy has medicines that treat damaged kidneys. The medicines stimulate the body’s immune system to repair the kidneys.

All stages of kidney failure can benefit from homeopathy, even end stage kidney failure when dialysis is imminent. Homeopathic medicine has been found to be beneficial in kidney transplant patients too by decreasing the chance of donor rejection.

Depending on the severity of the kidney failure, homeopathic medicines can promote kidney preparation. This means that successful treatment of kidney failure with homeopathy can eliminate the need for the life-long treatment that conventional medicine requires.

Homeopathic Medication for the Treatment of Acute Renal Failure

Some of the remedies which prove to be very helpful in cases of acute renal failure are: Similimum, Ars.Alb, Apis mel, Carbo. Veg, Cuprum ars, Digitalis, Helleborous, Opium and Vereterum album.

Homeopathic Medication for the Treatment of End-Stage Renal Disease

The most useful homeopathic remedies in these cases include: Apis mel, Ars alb, Cuprum ars, Colchicum, Eel serum, Glonoine, Helleborous, Morphinum, Opium, Plumbum met, Apocynum can, Solidago virga and Tribulus terrestris were found to be most useful organ remedies in severe cases.
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*Natrum muriaticum*, Silica and *Calcarea carbonica* have been found to act satisfactorily in cases with poor symptomatology and therefore they have been recommended for use in such cases.

**Homeopathic Remedies**

**Aconite**

Incipient stage of post scarlatinal nephritis, pain in loins, scanty urine without blood.

**Apis Mellifica**

Apis is not so much a remedy for chronic Bright’s disease as for the acuter forms. There are oedematous swellings of the face and extremities, paleness, ascites, edema pulmonum, pains in the head, back and limbs. Albuminuria following scarlatina. It may be of use in any form of Bright’s disease when there are dull pains in the kidneys, scanty urine and frequent Micturition. The urine is heavily charged with albumen and contains blood corpuscles. The edema appears quickly, there is general dropsy and suppression of urine and perhaps an eruption of the skin like a nettle rash. The patient is drowsy, apathetic and has a bruised feeling all over. Apis in such cases acts best in trituration; do not depend on the tincture or dilutions. Hepar is recommended by Kafka in Bright’s disease following scarlatina. A valuable symptom for Apis is the feeling of suffocation. He does not see how he is get another breath.

**Apocynum**

Palliative in dropsical conditions where the urine is scanty. Also useful for coma & convulsions in the nephritis of pregnancy.

**Arsenicum**

This remedy corresponds to all stages of Bright’s disease, bearing a closer resemblance than any other remedy. It
comes in later in the disease where there is dropsy, pale skin, waxen appearance, watery diarrhoea and great thirst. The urine is dark, casts are abundant, and it contains much albumen. There are attacks of dyspnoea when lying down in the evening and after midnight, relieved by an expectoration of mucus. It may come in immediately after Aconite in many cases. “Blood boils” make a special indication for this remedy. Baehr, Millard and Hale question the usefulness of Arsenicum in kidney affections. However, it seems a simile to the large white kidney; in fact, one could hardly wish for a closer correspondence. Hughes considers it a favorite with anxiety and sinking of vital forces will call for Arsenicum. Calcarea arsenica has been used in the anemia, progressive emaciation and debility of this disease with success.

**Aurum Muriaticum**

Aurum muriaticum is a homeopathic remedy that works well for those with acute renal failure who also suffer from gout or syphilis. Those who will benefit from this homeopathic treatment often suffer from vertigo as well as digestive problems. These digestive problems can include gas, bloating and heart burn.

**Belladonna**

Belladonna is recommended for those with acute renal failure who also have inflammation of the kidneys. Belladonna is of the greatest service in inflammation of the kidneys with piercing burning pains in the lumbar region, returning periodically with increased severity. Many people who will benefit from this homeopathic treatment have blood in the urine on occasion.

**Cantharis**

This remedy pictures nephritis with cutting pains in the lumbar region; the
urine is passed in drops and is mixed with blood, with much urging. Post scarlatinal and post diphtheric kidney diseases with dropsy may indicate Cantharis.

**Convallaria**

Convallaria is a homeopathic medicinethat works well for those with acute renal failure who also have heart palpitation. This homeopathic treatment is often suggested to sufferers who have inflammation of the kidneys that is associated with any type of heart disorder. It affords relief when there is extreme rapid & irregular action of the heart, and in general anasarca & ascites from mitral insufficiency.

**Cuprum Arsenicum**

Cuprum arsenicum is also useful in uremic conditions and is praised highly by Good no. Cuprum is a valuable remedy for uremic eclampsia.

**Digitalis**

This remedy has an irritant action on the kidneys. It is homoeopathic to granular degeneration. Heart symptoms, feeble pulse, scanty, dark, turbid urine, faintness at the stomach, rheumatic pains will indicate it. It is especially useful when the circulation is weak. Rheumatic pains, pulmonary catarrh with profuse expectoration are marked symptoms.

**Glonoine**

Glonoine has albuminous urine and will sometimes be found useful in acute and haemorrhagic nephritis.

**Kali Chloricum**

This remedy is said to be the most homoeopathic of all remedies in Bright’s disease. It has scanty, dark, albuminous urine containing casts. It excites a violent nephritis.
**Mercurius Corrosivus**

This remedy corresponds to the large white kidney. There is albuminous, scanty and red urine; pale waxen color of the body; there are lumbar pains, great dyspnœa and excessive strangury. It takes the first rank among all the mercurials for nephritis, and it comes in the later stages. Syphilitic complication further indicates it. There is an expression of uneasiness on the face. Dr. Ludlam considers it our best remedy for the albuminous nephritis of pregnancy and Baehr lauds it in suppurative nephritis.

**Plumbum**

Granular degenerations of the kidneys, with tendency to uremic convulsion. Dropsy, sallow face, emaciation, edema about the ankles. It seems to correspond to the contracted or cirrhotic form of nephritis, holding the same relation here that Arsenic and Mercury do in chronic nephritis. Royal emphasizes this remedy saying that it arrested the progress in many cases and permanently cured not few for him.

**Phosphorus**

Phosphorus produces as marked nephritis as any drug. It is one of the most important remedies in Bright’s disease; the characteristic symptoms are: lassitude of the whole body, hands and feet icy cold, sleepiness. The fatigue is greatest in the morning, and there is heat in the body without thirst, especially in the evening. The patient is indisposed to work, is giddy, forgetful and has a heavy headache, particularly in the forehead; there is edema of the upper eyelids, a mist before the eye, a yellowish fray complexion, a sickly edema of the face, want of appetite, pressure and burning in the stomach, and a light colored painless diarrhea which is very weakening. It suits well fatty or waxy casts, is dark brown, scanty and albuminous, or covered with an iridescent film. Pulmonary complications will call for Phosphorus; and inability to lie on the left side is a prominent symptom in these cases. Vomiting and gastric symptoms are...
usually present. A small dose of Phosphorus will act much safer and better in eclampsia than a large dose of Morphine.

**Terebinth**

One of our reliable and most frequently indicated remedies in the early stages of renal diseases when congestion is prominent, when there is much pain in the back of a dull character extending along the ureters. The great characteristic of dark smoky urine will be present. There is anasarca, and of course, the urine is bloody and albuminous. It is recommended in post scarlatinal renal affections. The prostration is not accompanied by the restlessness of Arsenicum.

**References**

3. Homeopathic Medicines & Treatment for diseases of kidney like Urinary Tract Infection, Renal Calculi, Kidney Failure, Glomerulonephritis, Nephritis etc.
CHAPTER 18
HERBAL REMEDIES FOR KIDNEY PROBLEMS

Introduction
In recent times, focus on plant research has increased all over the world and a large body of evidence has collected to show immense potential of medicinal plants used in various traditional systems. More than 13,000 plants have been studied during the last 5 year period.

Chronic kidney failure is a significant problem in modern society. Botanical medicine can be used to help forestall the need for dialysis by treating the causes and effects of renal failure, as well as reducing the many adverse effects of dialysis itself.

There are many natural and herbal ways to keep kidneys healthy. One of the best ways to help your kidneys stay healthy is to simply drink more water. As drinking plenty of fluid each day will help in continuously flushing out toxins, wastes and bacteria from your body which can cause disease, infections, and kidney stones.

(Java tea), *Cordyceps chinensis* (cordyceps, Chinese caterpillar fungus), *Centella asiatica* (gotu kola), and *Capsicum* spp. (cayenne).

**Home Remedies for Kidney**

1. Pomegranate is commonly used home remedy for kidney stones. Take its juice or grind its seed into fine paste. Cook horse gram in water and mix 1-2 tablespoon of this paste to it. Drink this concoction 2-3 times a day.
2. Drink one glass of fresh tomato juice the first obsession in the morning. Add a pinch of salt and pepper to this.
3. Radish and lady finger are very helpful in treating kidney stones. Take radish extract 3-4 times a day. For lady finger extract, cut 3-4 raw lady fingers in to small pieces and soak it in bowl of water. After keeping it overnight, press the lady fingers and take out all its extract. Drain the combination and drink it 2-3 times a day.
4. Fruits like apple, water melon and figs are extremely beneficial for kidney stones. Eat 3-4 apples every day. Similarly, raw watermelon or watermelon extract is also very good for health.
5. Take 2-3 figs and boil them in a glass of water. Take this in an unfilled stomach. Repeat this for 1-2 months.

**Effective Natural Herbs in Use of Kidney Failure**

Natural herbs which are particularly effective in kidney problems are: Kelp, Parsley, Juniper berries, Ginger, Uva ursi, dandelion, Garlic, Golden seal, Cayenne.

**Kelp**

Kelp is one of the most known herbal diuretics. This herb is quite high in natural potassium, iodine, iron, and various vitamins and minerals your body needs.

**Parsley**

It is a natural diuretic; it contains high content of potassium and many other vitamins and minerals.
**Juniper Berries**

It is a herbal diuretic. It not only helps increase the urine flow from your kidneys, but it also helps to clear congestion from the kidneys and urinary tract system too.

**Ginger**

It is an excellent herb to use for cleansing the kidneys.

**Uva Ursi**

It is an excellent natural herb which tends to be particularly effective in treating kidney problems. This herb stimulates the functionality of the kidneys while also strengthening and toning the urinary tract passage ways. This herb is also known for helping to clear out stones too.

**Dandelion**

Dandelion has strong diuretic properties.

**Garlic**

Garlic has a wide variety of uses and is a common herbal remedy. It is an important antiseptic. It is also used to treat hypertension.

**Golden Seal:**

Golden seal is anti-microbial; a bitter herb whose root is an anti-catarrhal, tonic and used as an antibiotic.

**Cayenne**

It is possibly the purest and best stimulant in herbal medicine. It produces natural warmth and helps the blood circulation, and eases weakness of the
stomach and intestines. Cayenne is added to tonics and is said to ward off diseases and can prevent development of cold and fevers.

**Researches on Herbal Drugs Efficacious for Kidney Patients**

Herbal drugs, like Corn silk (*Zea mays*) and Marshmallow (*Althea officinalis*), are used as demulcents. The Gravel root (*Eupatorium purpureum*), Stone root (*Collinsonia canadensis*), as and Elijory of the wall (*Parietaria officinalis*) which are reported to have "anti-lithic" and diuretic properties, have long been employed in the treatment of renal stones. These may have tremendous potential in the treatment of these disorders.

In a rat model, *Verbena officinalis*, *Lithospermum officianale*, *Taraxacum officinale*, *Equisetum arvense*, *Arctostaphylos uva-ursi* and *Arctium lappa* were found to have mild antibacterial action and the ability to alkalinize the urine, which resulted in solvent action on uric acid stones. For patients with stone disease accompanied by renal insufficiency, it would probably be wise to avoid herbal therapies because of uncertainty regarding the pharmaco-compounds which undergo renal excretion.

For hemodialysis patients, botanical approaches may turn out to be excellent substitute therapies in this population, which requires multiple pharmaceutical agents with frequent side effects. Adjunctive therapy for blood pressure control (both for hypo and hypertension), improvement of uremic bruising via improvement of capillary fragility using bilberry (*Vaccinium myrtillus*) or other herbs rich in bioflavonoids, improvement or maintenance of energy levels with adaptogens or identification of herbs which enhance iron or calcium absorption might be fruitful areas for research.

In renal transplant recipients, the use of bilberry extract for the treatment of prednisone-associated bruising. Cyclosporine-treated patients are at increased risk for gout, which is extremely resistant to treatment. As NSAIDs are typically contraindicated in this population because of the potential for decreasing renal function, we have utilized botanical products rich in linolenic acid (e.g. oil of evening primrose or flaxseed oil), without untoward effects. Again, the "adaptogenic" potential of the
ginsengs might be of use in these patients who undergo chronic corticosteroid therapy, although the potential for immune stimulation must be borne in mind, and such therapy should probably be reserved for stable patients in the late post-transplant period.

An interesting and potentially beneficial herb for renal transplant recipients with hepatitis B or C is milk thistle (*Silybum marianum*). Renal transplant recipients commonly develop urinary tract infection, and may require chronic antibiotic prophylaxis. Use of herbs which have antibacterial or demulcent activity (e.g. cranberry, corn silk) may be helpful in this regard.

Finally, the drugs which we use as primary immunosuppressants, cyclosporine and tacrolimus, have nephrotoxic effects. An isolate of ginkgolides from Ginkgo biloba, BN52063, has been shown in a rat model to protect against acute cyclosporine nephrotoxicity. In addition, the same isolate has been reported to decrease the incidence of early delayed graft function in a human trial. It is not known whether a standardized ginkgo extract would be as effective.

Finally, several herbs have been shown to be renoprotective in animal models of drug toxicity. After administration of a tubulotoxin such as cisplatinum or cyclosporine, it is believed that tubule cells die and the medulla becomes ischemic, resulting in the generation of free radicals by the remaining viable cells. Herbs such as the ones mentioned above, and other with potent antioxidant properties, may be able to prevent damage by oxygen radicals in known situations where nephrotoxicity may result (e.g. administration of nephrotoxic drugs, exposure to radio contrast agents).

**Conclusion**

Obviously, botanical medicine use in the patient with kidney disease is a potentially fruitful area for research. As the potential for patient benefit and the interest on the part both of patients and nephrologists alike are both tremendous, ideally, nephrologists, nurses, naturopaths, pharmacognosists, herbalists and others with an interest in both herbs and kidney patients will form a collaboration to judiciously use herbs in the renal failure patients.
Renal Failure: Its Treatment in Current Systems of Medicines

References
CHAPTER 19

UNANI TREATMENT FOR KIDNEY PROBLEMS

Introduction

The kidneys are the principle functional unit of the renal system. Other components of the renal system include the bladder, the ureter and the urethra. The main functions of the kidneys are: Excretory, Regulatory, Endocrine and Metabolic.

The functions of the kidney with respect to the temperament and structure are in line with the inherent wisdom of needs. It also operates under the sub-faculties of attraction, retention, alternative (metabolism), as well as, repulsive (excretory/elimination).

Plant remedies traditionally used as diuretics are Zeamays, Taraxacum officinale, Apium graveolens. The diuretics are indicated in following conditions:
• Dysuria and oliguria linked to urinary infection or stones
• Heart failure
• Ascitis
• Nocturnal enuresis and other functional disturbances of micturation
• Urinary stones

Plant remedies traditionally used as urinary antiseptics are: Arctostaphlos uva ursi, Barosma butulina, Berberis vulgaris. Urinary antiseptics are indicated in UTI or stones, prostatitis, and cystitis.

Unani Drugs Used in the Treatment of Kidney Diseases

*Achyranthes Aspera:* (Tibbi Name: Chirchita; English Name: Prickly chaff flower)

Decoction of the plant is an effective diuretic and prescribed for the treatment of renal dropsies.

*Alpinia Galanga:* (Tibbi Name: Kholanjan; English Name: The greater galangal)

The drug is used as a diuretic and in diseases of the kidney.
**Althaea Officinalis**: (Tibbi Name: Gul khairo; English Name: Marshmallow)

The drug is utilized as diuretic and for inflammation of kidneys.

**Amomum Subulatum**: (Tibbi Name: Ilaichi kalan; English Name: Greater cardamom)

The drug is used with the seeds of melon as diuretic to treat graves of kidneys.

**Aneilema Scapiflorum**: (Tibbi Name: Musli siyah; English Name: Curciligo)

The drug in combination with the juice of tulsi leaves is administered for relieving pain in the kidneys.

**Anemone Obtusiloba**: (Tibbi Name: Ratanjot; English Name: Al – kanet)

The drug is useful for the treatment of kidney disorders.

**Anethum Graveolens**: (Tibbi Name: Soya; English Name: Dill)

The drug has diuretic properties and is used in unani medicines for kidney problems.

**Asparagus Officinalis**: (Tibbi Name: Halyun; English Name: Asparagus)

The drug is a good diuretic. It stimulates the kidneys and is useful to excrete calculi.

**Asparagus Racemosus**: (Tibbi Name: Satavar; English Name: Asparagus)

Asparagus is diuretic and is useful in kidney disorders.

**Atropa Acuminata**: (Tibbi Name: Luffah; English Name: Belladonna/ Deadly Nightshade)

The drug is diuretic. It is prescribed in kidney problems.

**Bergenia Ciliata**: (Tibbi Name: Pakhanbed; English Name: Bergenia)

The plant is regarded as a diuretic. Removes the kidney and bladder stones.
Bombax Ceiba: (Tibbi Name: Simbal; English Name: Silk cotton tree)

- Dry young fruits of the drug are beneficial in calculous affections and chronic inflammation including ulceration of the bladders and kidneys.

Cassia Absus: (Tibbi Name: Chaksu; English Name: Cassia abus seeds)

- The seeds of the drug are pulverized with Santalum album Linn (Burada Sandal) in mortar and moistened with water for some time in an unused earthen ware pot and administered orally to patients suffering from uremia, consequently it stop blood in urine and corrects the functions of kidneys.

Cichorium Intybus: (Tibbi Name: Kasni; English Name: Chicory)

- Seeds are beneficial for kidney diseases, especially for renal stones.

Daucus Carota: (Tibbi Name: Gajar; English Name: Carrot)

- The seeds of the drug possess diuretic properties and useful in diseases of kidney.

Digitalis Purpurea: (Tibbi Name: Digitalis; English Name: Foxglove)

- The drug has diuretic properties and is useful in treating renal obstructions.

Ficus Carica: (Tibbi Name: Anjeer, English Name: Fig)

- The drug has diuretic properties. It is prescribed for the removal of calculi of the kidney and bladder.

Foeniculum Vulgare: (Tibbi Name: Saunf/Badyan, English Name: Fennel)

- The drug has diuretic properties. It is useful to clear the obstruction of kidney.

Ipomea Reniformis: (Tibbi Name: Chohakani, English Name: Mouse ear)

- The plant is useful in diseases of kidney.
Linum Usitatissimum: (Tibbi Name: Alsi , English Name: Linseed, Flax)
The bark and leaves of the drug are useful for kidney ailments.

Olea Europaea: (Tibbi Name: Zaitun, English Name: Olive)
It has diuretic properties and long-term use resolves the obstructions/ fatty depositions/ stones in kidney.

Portulaca Oleracea: (Tibbi Name: Kulfa, English Name: Purslane)
The drug is diuretic and is prescribed for the treatment of kidney.

Prunus Amygdalus: (Tibbi Name: Badam, English Name: Almond tree)
Bitter almonds are diuretic. Almonds are useful for the treatment of renal calculi.

Punica Granatum: (Tibbi Name: Anar, English Name: Pomegranate)
It is diuretic. The seeds are prescribed for kidney disorders.

Raphanus Sativus: (Tibbi Name: Muli/Turb, English Name: Radish)
The radish is useful in diseases of kidneys. The juice of the fresh leaves is used as a diuretic.

Rosa Damascena: (Tibbi Name: Gulab, English Name: Rose)
It is beneficial for the kidneys.

Rubia Cordifolia: (Tibbi Name: Majith, English Name: Indian madder)
The root is used for the treatment of kidney diseases. It is used as a diuretic.

Tephrosia Purpurea: (Tibbi Name: Sarphoka, English Name: Purple Tephrosia)
The whole plant is regarded to cure kidney disorders. The drug is also used as a diuretic.
Unani Treatment For Kidney Problems

References


CHAPTER 20
CHAPTER 20

VITAMINS REQUIREMENT FOR RENAL PATIENTS

Introduction

Dialysis patients have very specific vitamin needs. It is important that these patients only take multivitamins and supplements recommended by their health care team at the dialysis clinic. Many renal multivitamins are available for people who are dialysis dependent, with or without a prescription. A higher dose of folic acid (1.0 milligram [mg]/tablet or higher) usually are available by prescription only.

The renal multivitamin is primarily composed of water-soluble vitamins. Their composition usually follows the recommendations of the National Kidney Foundation. Some have fat-soluble vitamins and/or minerals added, which usually is what distinguishes them.

Patients with end-stage renal disease (ESRD) have larger vitamin needs for four different reasons:

- The dialysis patients’ meal plan is restricted in fruits, vegetables, and dairy products. Therefore, their diet becomes deficient in many vitamins. Their frequent lack of appetite and poor food intake also contributes to an insufficient intake of vitamins.
- The dialysis treatment itself directly promotes the loss of vitamins from the body. The treatment cleans the blood from extra fluid and toxins, as well as important vitamins.
- People with ESRD also require more protection from heart disease, access problems, inflammation, and anemia, which increases their vitamin needs.
- The body uses vitamins differently for those who are in a uremic state.

Water-soluble vitamins do not accumulate in the body and replacement is necessary, especially because they are lost through the treatment. Folic acid, B6, and B12 are known to decrease the risk of heart disease by decreasing homocysteine levels. Homocysteine is an amino acid produced by the breakdown of protein. Its high level in the dialysis population is known to lead to heart disease. Renal vitamins contain vitamins
Fat-soluble vitamins are not removed by the dialysis treatment and can easily accumulate to toxic levels, if supplemented in excess. Vitamin A usually is not added because of its toxicity. Vitamin D usually is given as the active form by prescription. Serum levels of parathyroid hormone (PTH), phosphorus, and calcium will help determine the appropriate dosage. Some renal multivitamins now include vitamin D in the form of cholecalciferol or D3. Vitamin E, an antioxidant is offered in some multivitamins, but supplementation usually is not needed. Vitamin K is not added because of its known effect on blood clotting.

The vitamin and mineral recommendations for peritoneal dialysis patients are similar to those of patients on hemodialysis. The water-soluble vitamins are lost to the peritoneal dialysate and replacement is required. Vitamin D is prescribed based on the serum levels of calcium, phosphorus, and parathyroid hormone in the same fashion as hemodialysis patients, but it is taken orally.

**Water Soluble Vitamins**

Most B vitamins are supplied in a combined tablet form of 3-6 different vitamins. As they are water soluble, and when in excess easily cleared from the body, even in severe renal failure, supplementation is a safe way to ensuring deficiency of this group of vitamin is avoided.

**Vitamin B1 (Thiamine)**

Dietary sources of thiamine include fresh green vegetables, whole meal grains and some meats. Potassium-restricted or protein-restricted diets may result in thiamine deficiency. It may take 12 months or more for the deficiency to develop. For patients following a prolonged protein-restricted diet, supplementary thiamine (1.0-1.5 mg/day is adequate maintenance) should be added to their medication profile.

**Vitamin B2 (Riboflavin)**

Meat is the rich source of Vitamin B2. As commonly 40% patients become vitamin B2 deficient on a protein-restricted
Vitamins Requirement for Renal Patients

diet. CKD patients following a prolonged protein-restricted diet, chronic kidney disease patients following a prolonged protein-restricted diet should have their diet supplemented with vitamin B2 by 1.0-2.0 mg/day.

**Vitamin B6 (Pyridoxine)**

Meat is a natural dietary source rich in pyridoxine. Predialysis patients on erythropoietin and patients on protein-restricted diets can develop pyridoxine deficiency. Such at-risk patients should have supplementary pyridoxine (5 mg/day is adequate maintenance) added to their medication profile. The relevance of reports of mega-dosing with vitamin B6 (300 mg/dl) being associated with a lowering of serum cholesterol is unknown in CKD patients.

**Vitamin B12 (Cobalmin)**

Vitamin B12 is lonely plentiful in meat and meat product foodstuffs. B12 requirements are low and deficiency is rare, and can take several years to develop after the introduction of a diet deficient in B12. Annual serum B12 levels can be monitored in high-risk patients, especially vegetarians.

**Folic acid**

Fresh green vegetables are the dietary source of folic acid, but prolonged cooking destroys folic acid present in vegetables. Folic acid deficiency results in megaloblastic anaemia. Predialysis patients on supplementation erythropoietin may need folic acid supplementation with 200μg per day, due to increased use of folate.

**Vitamin C**

Low potassium diets are also low in vitamin. Patients on low potassium diets can become vitamin C deficient. Serum ascorbic acid levels are low in most pre-dialysis patients. Supplementary vitamin C of > 60mg per day may increase the risk of hyperoxalosis and associated nephrolithiasis.

A high intake of vitamin C is associated with hyperoxalosis, which may contribute to the vascular disease of renal failure patients or obstructive uropathy. Care should be taken not to
exacerbate the chronic kidney disease with oxalosis/urine crystal formation from the excessive administration of supplementary vitamin C. Vitamin C supplementation may be given to assist the absorption of oral iron.

**Fat-Soluble Vitamins**

**Vitamin D**

Administration of vitamin D and dose adjustment should be prescribed initially at low doses with careful monitoring of serum calcium, phosphorus and PTH. Vitamin D is potentially valuable for patients at high risk of developing secondary hyperparathyroidism.

Choosing a renal multivitamin sometimes is difficult and confusing. The decision is made by personal preference.
### Comparison of Some of the Main Renal Multivitamins and Their Contents

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References
CHAPTER 21
The purpose of current research in nephrology is to develop and implement strategic plans that will enable to:

1. Prevent or delay the need for dialysis,
2. Broaden appropriate chronic kidney disease patient care options, and;
3. Improve the quality of all stages of chronic kidney disease care.

The Current Evidence for Slowing the Progress of Chronic Kidney Disease

The current evidence would favor the following treatment goals in order to both slow the progression of CKD and reduce CVD risk:

- BP <130/80 mmHg (<125/75 mmHg if >1 g/day proteinuria) to reduce proteinuria, slow CKD progression and reduce cardiovascular risk.
- ACEIs and ARBs to reduce proteinuria and slow CKD progression.
- Non-dihydropyridine calcium channel blockers to reduce proteinuria.
- HbA1c <6.5% in diabetics to reduce micro vascular complications.
- Total cholesterol <4 mmol/l.
- Smoking cessation.
- Dietary modification (weight reduction, alcohol and salt limitation, and avoidance of excess protein intake).
- Encourage exercise.¹
Interventions to Delay CKD  During the past 20 years, human and animal research has developed our understanding of CKD and led to preventive measures. The notion of renoprotection has resulted in a dual approach to renal diseases based on effective and sustained pharmacological control of blood pressure and reduction of proteinuria. Lowering blood lipids, stopping smoking, and maintaining tight glucose control for diabetes form part of the multimodal protocol for managing renal patients monitored by specific biological markers.

Abnormal urinary excretion of protein is strongly associated with the progression of CKD in both diabetic and non-diabetic renal diseases. Clinical studies have established that a reduction in proteinuria is associated with a decreased rate of kidney function loss. A specific category of drugs that lower blood pressure, the angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers, appear to be more effective than other antihypertensive drugs in slowing the progression of both diabetic and non-diabetic CKDs. The administration of an ACE inhibitor (or of an angiotensin receptor blocker) is an important treatment for controlling blood pressure and slowing the rate of progression of chronic kidney failure. Other drugs to
lower blood pressure are added as necessary to achieve current targets of 120/80 to 130/80 millimeters of mercury. Concurrent diuretic therapy is often necessary in patients with renal insufficiency, because fluid overload is an important determinant of hypertension in such cases.

Dyslipidemia accelerates atherosclerosis and may promote the progression of renal disease. Careful control of the blood glucose level in diabetic patients can be beneficial and may limit other complications. Obesity has not been directly linked to the progression of CKD but is an important risk factor for diabetes and cardiovascular morbidity and mortality. Many patients and health care professionals do not appreciate the benefits of smoking cessation, an important measure in protecting the kidneys from progressive disease resulting from cardiovascular disease (CVD). Additional elements of secondary prevention measures include the treatment of anemia and of abnormal calcium and phosphorus metabolism.

Additionally, acidosis, anemia and disturbances of calcium and phosphate metabolism should be looked for and treated in patients with CKD stage 3 or above.

To extend the evidence base further, more interventional studies specifically in CKD patients are required particularly in the area of CVD risk reduction. Additionally, clarification of the benefits of the interventions discussed for non-proteinuric compared with proteinuric CKD is required. Finally, most of the trials reviewed have age restrictions in the recruitment criteria (generally <70 years). There is currently no evidence to say if the benefit of the interventions discussed persists at higher ages, and given the increasing incidence of CKD with age, trials in older age groups would be of value.

Control Strategies Implementation

Measures for primary and secondary prevention of CKD are now well documented and will eventually reduce the number of patients requiring dialysis. Until recently, the focus has been on RRT to save lives, and considerable efforts are being made to improve the quality of dialysis. In the United States, guidelines derived from the Kidney Disease Outcomes Quality Initiative have added greatly to the quality of dialysis in terms of access (graft or fistula), adequacy, treatment of anemia, treatment of
secondary hyperparathyroidism, and more recently greater emphasis on CVD, all of which contribute to quality-of-life outcomes, but at an increased cost.

The high mortality rate of dialysis approximates 10 percent per year and has changed little over the past; however, new approaches are emerging for dealing with CVD in RRT facilities. More patients with kidney disease die before they get to the point at which they need treatment for renal failure, because early kidney disease is a major marker for CVD and reinfarction, congestive heart failure, and stroke.

The acknowledgment by the World Bank and the World Health Organization that chronic conditions, particularly those resulting from diabetes and hypertension, will increase to become a leading cause of death by 2028 has intensified the need for prevention and RRT programs. The need to increase awareness, launch targeted screening and intervention studies, provide training for staff, maintain education for physicians in kidney and urological disease, and assist centers for RRT is urgent.

Developed nations have well-established nephrology and urology centers attached to academic medical institutions and regional public and private secondary and tertiary referral hospitals. They have training programs to meet national requirements for health professionals including renal physicians, primary care physicians, and nurses specializing in kidney and urological disorders. Their centers incorporate the results of up-to-date research developments pertaining to kidney disease and clinical applications of the latest advances in care and technology. Numerous publications arise from academic endeavors, and a close association exists between health care delivery and pharmaceutical industries. Each country and region has societies of nephrology and urology for adults and children.
### Strategies of Proven Efficacy for Retarding Progression of Chronic Kidney Disease

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<td>Target proteinuria &lt;500 mg/d</td>
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<td>ACE Inhibitor + ARB</td>
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<td>Greater anti-hypertensive and anti-protein uric effect with reduced progression</td>
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<td>Blood sugar control</td>
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Other Anti-Proteinuric Therapies which May Retard the Progression of Chronic Kidney Disease in Humans

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<td>Avoid DHCEB</td>
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<td>β-blocker</td>
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<td>NDH CEB anti-proteinuric</td>
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<td>Restrict NaCl</td>
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<td>Increase anti-hypertensive and therefore anti-proteinuric effects of ACE inhibitors and ARBs.</td>
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<td>Control fluids</td>
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<td>Aldosterone antagonist</td>
<td>Sato 19</td>
<td>Particularly HMG CoA reductase inhibitors</td>
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<td>Lipid control</td>
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<td>Cease smoking</td>
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AASK: African American Study of Kidney Disease and Hypertension; ABCD: Appropriate Blood Pressure Control in Diabetes; ACE: angiotensin-converting enzyme; DHCEB: dihydropyridine; DM: diabetes mellitus; HMG CoA: 3-hydroxy-3-methylglutaryl coenzyme A; MDRD: Modification of Diet in Renal Disease; NDH CEB: non-dihydropyridine calcium entry blocker

Conclusion - Hope Through Research

Kidney disease and kidney failure, especially as a complication of type 2 diabetes mellitus and hypertension, are rising globally and are rising faster in developing countries. The following guidelines for diseases of the kidney and urinary system are recommended:

- Expand surveillance of the prevalence of various kidney and urological diseases in developing countries. Provide support for further epidemiological studies in selected countries for assessing the prevalence of kidney disease and interventions.
to address it and for establishing an international kidney disease data center.

- Promote public awareness in developing countries about the nature and early signs of kidney disease along with knowledge of prevention measures and therapies.

- Focus more attention on the increasing prevalence of diabetes and hypertension, and develop kidney disease programs in that context. Measures of kidney function and protein excretion should be taken. The implementation of primary and secondary prevention to reduce the prevalence of ESRD should be expanded.

- Increase coordination and resources for efficient and timely distribution of supplies and equipment, assessment of patients, and frequent dialysis for acute renal failure patients caused by crush injuries during such major disasters as earthquakes. Countries in earthquake-prone regions should develop emergency policies and practices and be linked with the appropriate international agencies.

- Have the World Bank and the World Health Organization establish a policy advisory group with relevant international groups, such as the International Society of Nephrology, to address and advise national and regional health ministries on kidney and urological strategies as requested.

- Make major health and medical education programs available on an annual basis through existing societies and agencies to train and update physicians, nurses, technicians, and other relevant health professionals.

- Develop selected centers of excellence for education, training, clinical care, and prevention of kidney and urological disease and clinical care of renal failure. At least 10 such centers should be developed in the next decade and located in the countries of the former Soviet Union, Africa, Asia, Eastern Europe, and Latin America. Funds should be provided by international and national agencies and national government organizations and be sustained for up to 10 years.

As understanding of the causes of kidney failure increases, so does the ability to predict and prevent these diseases. Recent
studies have shown that intensive control of diabetes and high blood pressure can prevent or delay the onset of kidney disease.

In the area of transplantation, new drugs to help the body accept foreign tissue increase the likelihood that a transplanted kidney will survive and function properly. Scientists at the NIDDK are also developing new techniques to induce a person’s tolerance for foreign tissue before receiving a transplanted organ. This technique will eliminate or reduce the need for immunosuppressive drugs and thereby reduce expense and complications. In the future, scientists may develop an artificial kidney for implantation.

Participants in clinical trials can play a more active role in their own health care, gain access to new research treatments before they are widely available, and help others by contributing to medical research.

An estimated 11.5% of adults have evidence of chronic kidney disease (CKD), and each year in the United States, more than 100,000 people are diagnosed with kidney failure, the final stage of CKD. The Healthy People 2020 initiative was launched in December 2010, and it includes new kidney-related objectives that focus on monitoring and tracking: Improvements in heart-related care in patients with CKD Increases in the proportion of patients with CKD and diabetes who receive recommended exams and treatments Reductions in the incidence and death rate of CKD Increases in CKD awareness in people with poor kidney function It’s hoped that these new objectives will help reduce new cases of CKD and its progression, complications, disability, death, and economic costs. 23-24
Experimental Approaches to Retard Renal Failure Progression

| Exogenous recombinant growth factor proteins (e.g., HGF, BMP-7, VEGF) |
| Anti-fibrotic drugs and recombinant proteins (e.g., relaxin, pirfenidone) |
| Anti-inflammatory drugs (e.g., immunosuppressants) Cytokine and chemokine antagonists and modulators Endothelin receptor antagonists |
| Aldosterone antagonists Retinoids and vitamin D Anti-proteinuric agents (e.g., pentosan polysulfate, neutralising antibody against heparanase, C5b-9) Modulators of cell cycle regulatory proteins Modulators of intracellular signaling pathways (e.g., protein kinase C inhibitor, ruboxistaurin; MAPK) |

BMP-7: bone morphogenetic protein-7; HGF: hepatocyte growth factor; MAPK: mitogen activated protein kinase; VEGF: vascular endothelial growth factor

References


Recent Research Based Strategies for Reducing the Burden of Renal Failure

CHAPTER 22

![Diagram showing the progression from At Increased Risk to End-Stage in CKD and CVD](image)

- CKD: Kidney Failure, Decreased GFR, Albuminuria
- CVD: Heart Failure, CVD Events, CAD, LVH

**Initiation**
**Progression**
**End-Stage**
**At Increased Risk**

**DIABETES**
HTN, Age, Family History
Case History 1 (Diabetes and renal impairment)

CM is a 27-years old white woman with type 1 diabetes diagnosed at age 14 when she presented with diabetic ketoacidosis. Her initial insulin treatment was complicated by poor glycaemic control, frequent hypoglycaemia and weight gain.

Two years ago she developed hypertension, which was treated with benzdroflumethiazide, 5 mg daily. At that time, her blood urea level was 8.2 mmol/L, serum creatinine was 80 µmol/L, and dipstick urinalysis was negative for protein. She was also noted to have non-proliferative diabetic retinopathy, and given a course of laser treatment.

She has been admitted via A & E complaining of nausea and vomiting. On examination, she was dehydrated and her breath smelled of ketones. She was conscious and alert. Her finger-prick blood glucose was 25.4 mmol/L and the urine dipstick was strongly positive for glucose, ketones and proteins. She was diagnosed as being in diabetic ketoacidosis and was transferred to the intensive care unit for further management.

On admission to the intensive care unit her laboratory results were as follows:

- Na+ 127 mmol/L (135-150 mmol/L)
- K+ 4.5 mmol/L (3.5-5.2 mmol/L)
- Blood Ph 7.15 (7.36-7.44)
- Base excess -20.9 mmol/L
- Bicarbonate 5.8 mmol/L (22-31 mmol/L)
- Urea 18.3 mmol/L (3.5-5.2 mmol/L)
- Creatinine 546 micromol/L (60-110 micromol/L)
- Glucose 40.1 mmol/L
HbA1c \((3.9-6.1\%)\)

Her weight is 54 kg, and she is 160 cm tall.

CM was started on intravenous insulin, fluids, and electrolyte replenishment. Her nausea and vomiting resolved and, although initially, she required 60-70 units of insulin intravenously per day to attain glycaemic control, her blood glucose dropped to 7.4 mmol/L after 4 days of intensive care. However, despite treatment of her diabetes ketoacidosis, including significant rehydration therapy, CM was still found to have an elevated but stable serum creatinine of 246 micromol/L, and so she was transferred from the intensive care unit to the renal unit for further management.

As a result of the urethral catheter she had inserted in the intensive care unit, CM develops a urinary tract infection, with an E.coli that is resistant to trimethoprim and amoxicillin, but is sensitive to gentamycin. She is prescribed a dose of gentamycin, 7mg/kg intravenously once daily for 5 days.

**Questions**

1. What is diabetic ketoacidosis?

Diabetic ketoacidosis is one consequence of untreated diabetes mellitus and is linked to an impaired glucose cycle. In a diabetic patient, DKA begins with deficiency in insulin. This is most commonly due to undiagnosed diabetes mellitus or, in patients who have been diagnosed with diabetes, failure to take prescribed insulin. DKA has a 100% mortality rate if left untreated.

2. Calculate CM's renal function using both the MDRD equation and the Cockcroft-Gault formula.

The standard equations used to calculate renal function may be applied here, as although the serum creatinine is elevated, it is stable. Were it still changing rapidly, the calculations would not be accurate.

**MDRD**

\[ \text{eGFR} = 22 \text{ml/min/1.73m}^2 \]

**Cockcroft and Gault**

Ideal body weight = 45.5+(2.3*3) = 52.4kg. Since CM's actual body weight is not more than 15% greater than her IBW, use CM's actual body weight:
Case Histories

GFR = 1.04* (140-Age)* weight/serum creatinine
   = 1.04*113*54/246
   = 25.8 ml/min

3. What is the likely cause of CM's renal impairment?
CM appears to have developed diabetic nephropathy, although a renal biopsy would be required to establish the definitive diagnosis. Approximately 40% of people with long-standing type 1 diabetes develop diabetic nephropathy. Essentially all patients with diabetic nephropathy also have diabetic retinopathy.
In type 1 diabetes, diabetic nephropathy follows a predictable course from onset of diabetes to the onset of microalbuminuria to nephropathy to end-stage renal disease or death. Hypertension develops in association with microalbuminuria and progresses with diabetic nephropathy, further damaging the kidneys. Once 'end-stage renal disease' is reached, the toxins in the body can no longer be cleared by the kidneys and, unless treated by dialysis, can build up to fatal levels.

4. What are the factors that increase the likelihood of getting diabetic nephropathy?
Some of the factors that are known to increase the likelihood of getting diabetic nephropathy include:
- Poor blood sugar control
- High blood pressure
- Smoking
- Relatives with kidney disease or hypertension
- Onset of diabetes in teen years
- Male
- Indo-Asian or Afro-Caribbean background.

5. What pharmacological and other interventions could be employed to reduce the risk of problems?
Once the process of diabetic nephropathy has begun, nothing can be done to stop it, and in most cases, eventually the patient will progress to end-stage renal failure. However, a number of clinical management points have been shown to slow the rate of progression of diabetic nephropathy, helping the patients to maintain their residual renal function for longer, and delaying the need to instigate dialysis.
<table>
<thead>
<tr>
<th>Stage</th>
<th>Assessment</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>No proteinuria</td>
<td>Monitor blood pressure&lt;br&gt;Monitor blood glucose&lt;br&gt;Screen for microalbuminuria&lt;br&gt;If type 1 diabetes for &gt;5 years or type 2 diabetes</td>
<td>Aim for &lt;130/80 mmHg&lt;br&gt;Aim for &lt;120/70 mmHg if type 1 diabetes&lt;br&gt;Aim for HbA1c &lt;7%&lt;br&gt;Dietary advice for sugar and fat&lt;br&gt;Stop Smoking</td>
</tr>
<tr>
<td>Microalbuminuria</td>
<td>Close monitoring of blood pressure, blood glucose and blood lipid levels&lt;br&gt;Monitor urinary protein</td>
<td>Aim for BP &lt;125/75 mmHg. Add further anti-hypertensive drugs if necessary&lt;br&gt;Add ACE1/ARB if possible&lt;br&gt;Aim for total cholesterol &lt;3.5 mmol/L</td>
</tr>
<tr>
<td>Declining kidney function</td>
<td>Close monitoring of blood pressure, blood glucose and blood lipid levels&lt;br&gt;Monitor urinary protein</td>
<td>As for microalbuminuria</td>
</tr>
</tbody>
</table>

6. Comment on the dose of gentamicin
   Gentamicin is a drug with a narrow therapeutic index, which is highly nephrotoxic and ototoxic. In order to minimize this toxicity, it is necessary to monitor plasma levels of the drug. The usual practice is to reduce the dose in patients with renal impairment and monitor levels very carefully.

7. What dose should be recommended?
   Depending on the equation used, CM’s GFR has been calculated as being between 22 and 26 ml/min. Given this level of renal function, it would be prudent to recommend giving a dose of 4 mg/kg body weight, and then monitoring plasma levels until a suitable trough level of less than 1.5-2.0 mg/L has been reached. Depending on how well the patient clears the drug, daily dosing maybe required, or she may only need a dose every 48 hours.
CASE HISTORY 2 (Hypertension – associated kidney disease)

Mr WD, 42 years old Afro-Caribbean man, presents to his GP with six week history of headaches and lethargy. On examination the following is noted:

- Dipstick – proteinuria
- Blood pressure 180/105 mmHg (120/80 mmHg)
- Serum creatinine 365 micromol/L (60-110 micromol/L)
- Serum urea 15.8 mmol/L (3.2-6.6 mmol/L)
- Weight 98 kg
- Height 180 cm

Mr WD was prescribed nifedipine LA 30mg once daily and enalapril 10 mg twice daily to treat his hypertension. After one week’s treatment, his blood pressure was still only 150/85 mmHg, but the patient was complaining of very swollen ankles. He also mentions that he has developed a persistent cough.

Questions

1. Comment on Mr WD's laboratory results.

   Mr WD has severe hypertension with a blood pressure of 180/105 mmHg. The proteinuria on dipstick is also indicative of renal damage. His serum creatinine is well above the normal range of 65-115 micromol/L, and his serum urea is also elevated (normal range 3.0 – 6.5 mmol/L), indicating that he has moderate to severe renal impairment.

2. Calculate Mr WD's renal function using MDRD equation and the Cockcroft-Gault formula.

   MDRD
   \[ e\ GFR = 21 \text{ ml/min/1.73m}^2 \]

   Cockcroft and Gault
   Ideal body weight = 50 + (2.3*11) = 75.3kg
   \[ \text{GFR} = 1.23 \times (140 – \text{Age}) \times \frac{\text{Weight}}{\text{Serum creatinine}} \]
   \[ \text{GFR} = 1.23 \times 98 \times \frac{75.3}{365} \]
   \[ \text{GFR} = 24.8 \text{ ml/min} \]
3. What do you think may have caused Mr WD's renal impairment?
Hypertension can be a consequence as well as a frequent cause of renal dysfunction. Among 332,544 men screened for the Multiple Risk Factor Intervention Trial (MRFIT), there was a graded relationship between levels of both systolic and diastolic blood pressure at baseline and risk of subsequent ESRD, even after considering other predictors of ESRD risk (i.e. age, race, smoking, cholesterol, diabetes, myocardial infarction and income). Almost half of the incident cases of ESRD (49%) were attributable to hypertension.
In patients with renal disease, hypertension can increase the rate of progression to ESRD and therefore the use of anti-hypertensive drugs can be important in delaying the progression to ESRD. This is the same for all causes of renal impairment. In some cases, initiation of dialysis is needed to control hypertension.
It should be remembered that hypertension in patients with renal dysfunction can be particularly difficult to treat, and many patients require more than one anti-hypertensive agent in order to control their blood pressure.

4. Which drugs are used to treat hypertension?
The drugs commonly used in the treatment of hypertension are:
- Thiazide diuretics
- Beta-blockers
- Alpha-adrenoceptor blockers
- Calcium channel blockers
- Angiotensin-converting enzyme (ACE) inhibitors
- Angiotensin II receptor blockers
- Centrally acting drugs
- Direct acting vasodilators

5. What therapy would you recommend for the treatment of Mr WD's hypertension?
Following the guidelines of the British Hypertension Society, Mr WD should be prescribed a thiazide diuretic and a calcium channel blocker, so a good combination might be nifedipine LA 20 mg or 30 mg once daily or a tomlodipine 5
mg once daily, plus bendroflumethiazide 2.5mg once daily. However, we know that his renal function is poor, with a calculated GFR of approximately 25 ml/min, so he is borderline for thiazides to be clinically effective. In this instance, it might be prudent to prescribe an ACE inhibitor instead, for example, enalapril 5-10 mg twice daily.

6. What could be the cause of Mr WD's new symptoms? Calcium channel blockers are well known to cause peripheral oedema, in particular ankle oedema. This condition is unresponsive to diuretic therapy, and so the only way to reverse it to discontinue the drug. ACE inhibitors apart from converting angiotensin I to angiotensin II, is also responsible for assisting in the breakdown of bradykinins and other inflammatory mediators within the body. These bradykinins manifest their higher blood levels by inducing a coughing reaction. This may vary from a light tickly cough that does not cause too much inconvenience, to a severe hacking cough that necessitates discontinuation of the drug.

7. What treatment would you suggest now for Mr WD's hypertension? In view of his cardiovascular status and proteinuria, WD should be on 'anti-ACE' therapy of some kind. If he is unable to tolerate an ACE inhibitor, an alternative might be to try an ARB. This has the advantage of blocking rennin-angiotensin system, thereby achieving the required therapeutic effect but since ACE itself is not affected, the patient should not develop a cough.

If the patient is finding the ankle oedema caused by the calcium channel blockers distressing, then the drug should be discontinued. Some patients find the symptoms may be reduced by switching to a longer-acting drug within the class, for example, amlodipine. Failing this the drug needs to be discontinued, and an alternative class of drug used, for example, a beta-blocker or an alpha receptor blocker

References

CHAPTER 23
What are the functions of kidneys?
The normal kidney helps in maintaining regulatory, excretory, synthetic and metabolic functions of the body.

What is kidney failure?
Kidney failure refers to temporary or permanent damage to the kidneys that result in loss of normal kidney function.

Is Kidney failure permanent?
Not always. Some kinds of acute kidney failure get better after treatment. In some cases of acute kidney failure, dialysis may only be needed for a short time until the kidneys get better.

In chronic or end stage kidney failure, your kidneys do not get better and you will need dialysis for the rest of your life or get kidney transplantation.

What are the causes of kidney failure?
The causes of kidney disease are: diabetes, hypertension, and inflammation of the kidney, malaria, long-term exposure to lead, solvents and fuels, systemic lupus erythematosus, polycystic kidney disease, physical injury, kidney infection, jaundice, yellow fever.

What are the generalized symptoms of kidney failure?
The symptom of kidney failure are as follows: fatigue, frequency of urination, itchy skin, erectile dysfunction, nausea, shortness of breath, water retention, blood in urine, protein in urine.

What is the major cause of death in renal failure patients?
Nearly half of the deaths in dialysis and post transplantation patients are due to cardiac causes.
Discuss the role of ultrasonography in the diagnosis and management of renal diseases. This is the method of choice for assessing overall renal size and distinguishing solid tumors from cysts. Renal ultrasound can demonstrate dilatation of the pelvicalyceal system, which may be due to obstruction. Perinephric abscess, hematomas or calculi may be demonstrated. Renal biopsy and cyst puncture can be done under ultrasound screening. It is an excellent screening test for polycystic kidney disease. In malignant renal tumours, renal ultrasound can give additional information regarding extension of the tumour to renal veins, vena cava, lymph node or liver. The prostate size and bladder capacity can also be assessed.

What is acute kidney failure?
Acute kidney failure is a sudden and complete loss of kidney function due to accidents, medicines, surgery, and low blood pressure from shock, blockages of the bladder or kidney or serious infection.

What are the symptoms of acute kidney failure?
Symptoms of acute renal failure depend largely on the underlying cause:
- hemorrhage
- fever
- weakness
- fatigue
- rash
- diarrhea
- poor appetite
- severe vomiting
- abdominal pain
- back pain
- muscle cramps
- no urine output or high urine output
- history of recent infection (a risk factor for acute renal failure)
- pale skin
- nosebleeds
- history of taking certain medications (a risk factor for acute renal failure)
- history of trauma (a risk factor for acute renal failure)
- swelling of the tissues
- inflammation of the eye
- detectable abdominal mass
- exposure to heavy metals or toxic solvents (a risk factor for acute renal failure)

What is chronic kidney disease?
Chronic kidney disease or end-stage renal disease is a gradual decrease of kidney function of both kidneys over a
period of time due to damage to kidneys as a result of diabetes, high blood pressure, heart disease or drug abuse, kidney diseases, kidney infections, kidney stones.

- What do you know about prevalence of chronic kidney disease?
The incidence of chronic kidney disease increases with age and is greater in females and some ethnic populations.

- What are the risk factors for developing chronic kidney disease?
The risk factors for developing chronic kidney disease are:
- Increased susceptibility to kidney damage – older age, family history of chronic kidney disease, low birth weight, low income or educational level.
- Worsening kidney damage – higher level of proteinuria, higher blood pressure, poor glycaemic control in diabetes, smoking.
- Increased morbidity and mortality in kidney failure – Temporary vascular access, anaemia, low serum albumin level, late referral to dialysis.

- What are the symptoms of chronic kidney failure?
- vomiting • bone pain • headache • insomnia • itching • dry skin • malaise • fatigue with light activity • muscle cramps • high urine output or no urine output • recurrent urinary tract infections • urinary incontinence • pale skin • bad breath • hearing deficit • detectable abdominal mass • tissue swelling • irritability • poor muscle tone • change in mental alertness • metallic taste in mouth Poor appetite

- Which organs are affected in chronic kidney disease?
In severe, chronic kidney disease virtually all body systems are adversely affected.
Renal Failure: Its Treatment in Current Systems of Medicines

- Which grade of chronic kidney disease is an important risk factor for cardiovascular disease?
  All grades of chronic kidney disease are important risk factors for cardiovascular disease.

- What are the clinical signs and symptoms of severe chronic kidney disease?
  Clinical signs and symptoms of severe chronic kidney disease include nocturia, oedema, anaemia, hypertension, bone pain, neurological changes and disordered muscles function.

- What is the aim of treatment of chronic kidney disease?
  The aims of treatment are to reverse or arrest the process responsible for chronic kidney disease and relieve symptoms.

- What is the most important preventive measure to avoid further renal damage in cases of chronic kidney disease?
  To prevent further renal damage, adequate control of blood pressure is essential.

- What is the last treatment for ESRD patients?
  - Renal transplantation remains the treatment of choice for end stage renal disease. However, up to 60% of patients on dialysis programmes are not fit enough to be put on the transplant list.

- What are the treatment protocols for kidney failure?
  Kidney failure is treated with a special diet, medicines, regular dialysis treatments and, possibly, a kidney transplant.

- Which medicines are commonly prescribed in renal failure patients on dialysis?
  The few commonly prescribed medicines are:
  Multivitamins
  Phosphate binders
  Calcium supplements
  Synthetic erythropoietin

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Frequently Asked Questions

Other common medications: iron, antihypertensives, heart stimulants, and stool softeners.

- Which procedures are used in allopathic system for the treatment of chronic renal failure patients?
  There are three procedures in allopathic system for the treatment of chronic renal failure patients:
  Symptomatic medicines with some specific medicines to control the causative disease such as diabetes mellitus, hypertension, etc.
  Dialysis (Hemodialysis or peritoneal dialysis)
  Kidney transplantation

- What is the significance of dialysis?
  Dialysis is a treatment that does some of the things done by healthy kidneys. It is needed when your own kidneys can no longer take care of your body's needs.

- When is dialysis needed?
  You need dialysis when you develop end stage kidney failure, usually by the time you lose about 85-90% of your kidney function.

- What does dialysis do?
  Like healthy kidneys, dialysis keeps your body in balance.
  Dialysis does the following:
  Removes waste, salt and extra water to prevent them from building up in the body.
  Keeps a safe level of certain chemicals in your blood, such as potassium, sodium and bicarbonate.
  Helps to control blood pressure.

- What are the types of dialysis?
  There are two principal types of dialysis: hemodialysis and peritoneal dialysis. In both, toxic metabolites are transferred from the patient's blood across a semi-permeable membrane to a dialysis solution.
What is hemodialysis?
In hemodialysis, an artificial kidney is used to remove waste and extra chemicals and fluid from the blood. To get the blood in to the artificial kidney, the doctor needs to make a fistula, graft or a catheter.

In general, what is the duration and frequency of hemodialysis?
Each hemodialysis treatment lasts about four hours and is done three times per week.

What is peritoneal dialysis?
In this type of dialysis, the blood is cleaned inside the body. The surgery has to be performed to place a plastic tube called a catheter into the abdomen to make an access. During the treatment, the abdominal area is slowly filled with dialysate through the catheter. The blood stays in the arteries and veins that line the peritoneal cavity. Extra fluid and waste products are drawn out of the blood and into the dialysate.

What are the different kinds of peritoneal dialysis?
There are several kinds of peritoneal dialysis but two major ones are:
Continuous Ambulatory Peritoneal Dialysis
Continuous Cyclic Peritoneal Dialysis

Do dialysis help cure the kidney disease?
No. Dialysis does some of the work of healthy kidneys, but it does not cure the kidney disease.

Is dialysis uncomfortable?
The patients may have some discomfort when the needles are put into the fistula or graft. However, some patients may have a drop in their blood pressure.

Do dialysis patients have to control their diets?
Yes
Frequently Asked Questions

- What are the problems associated with dialysis?
The problems associated with dialysis are: headaches, nausea/vomiting, fluid overload, low blood pressure, muscle cramping, anemia, high blood pressure, infection, bone disease, itching, nervous movement and blood leaks.

- What is the frequency of increase in dialysis in chronic kidney disease patients?
The need of dialysis therapy continues to increase at about 10% per annum.

- What is kidney transplantation?
Kidney transplantation is a major treatment option for many patients with kidney failure. It involves placing a healthy kidney into the body where it can perform all of the functions that a failing kidney cannot.
Kidney transplantation involves the placement of new kidney on the lower right or left side of your abdomen where it is surgically connected to nearby blood vessels. Placing the kidney in this position allows it to be easily connected to blood vessels and the bladder. The vein and artery of your new kidney are attached to your vein and artery. The new kidney's ureter is attached to your bladder to allow urine to pass out of your body.

- What are the sources for obtaining kidney for transplantation?
Kidneys for transplantation come from three sources:
Living related family donors,
Living, non-related donors,
Cadaver or non-living donors.

- What is done with the diseased kidneys when transplanting a new one?
In most cases, the diseased kidneys are not removed. There are three conditions that might require your diseased kidneys to be removed:
Repeated infection that could spread to the transplanted kidney
Uncontrollable hypertension caused by your original kidneys
Backup of urine into your kidneys (a condition called reflux)

- What are the side effects associated with the use of complementary/alternative medicines in renal diseases? Complementary/alternative medicines are increasingly used to diagnose or treat renal diseases, and numerous studies have reported benefit of this type of medicine. But have its own risks. The potential sensitizing capacity of numerous herbal remedies may lead to allergic contact dermatitis and more rarely to IgE mediated clinical symptoms. Organ toxicity has been observed associated with various herbal preparations involving the liver, kidneys and the heart. Some herbs may have carcinogenic properties.

- Which type of kidney patients should take homeopathic treatment?
  - Healthy persons who have strong family history of renal failure should take classical homeopathic treatment to avoid the risk of renal failure.
  - Diabetic patients who are suffering from hypertension also must take homeopathic treatment.
  - Persons who are having symptoms of early renal failure of unknown etiology.
  - Patients suffering from recurrent renal stone formation or having stone lodged somewhere in the ureter.
  - Kidney patients of any stage should start homeopathic treatment earliest possible for avoiding passing into the complete renal damage.
  - Patients who are taking some urine forming medicines such as lasix, Dytor etc should start homeopathic treatment otherwise their kidneys will have to suffer badly.
  - Patients who are on dialysis can get rid off the frequent dialysis by taking classical homeopathic treatment.
  - Patients who are waiting for renal transplantation should start homeopathic treatment which can stop the urgent need of kidney donor.
Frequently Asked Questions

- What are the important homeopathic medicines used in chronic renal failure?
  Important homeopathic medicines used in chronic renal failure are ammonium carb., arsenic carb, aurum met, cicuta, cuprum met, cuprum ars, glonine, opium, phosphorus, picric acid, platina, terbinthina.

- How do homeopathic medicines treat end-stage renal failure?
  In homeopathy, medicines developed immunizations in damage nephrons in kidney, so kidney function is not damage further and become strong. After initiation of homeopathy treatment, decrease in serum creatinine and BUN takes place gradually.

- How can we correct renal anemia?
  Renal anemia is common when the GFR falls below 30ml/min but can be corrected by epoetin in 90-95% of cases.

- What is meant by azotemia?
  Azotaemia is a term used to denote an increase in the concentration of urea and creatinine in the blood, which occurs as a result of a fall in glomerular filtration rate.

- What is meant by oliguria?
  Oliguria refers to the production of insufficient urine to enable solute to be excreted in adequate amounts, and is usually less than 400ml of urine/day.

- What is anuria?
  Anuria refers to complete cessation of urine flow. It more commonly indicates obstruction of the outflow from both kidneys.

- What is meant by polyuria?
  The term polyuria denotes persistent increase in urine volume of more than 3L/day. But this should be qualified to
exclude normal individuals who desire a large fluid intake, and therefore, form large volumes of urine.

- **What is proteinuria?**  
  Pathologically proteinuria is the urinary excretion of more than 150 mg of protein/day.

- **What is microalbuminuria?**  
  Normal urine contains albumin in a concentration of less than 30 mg/L. Elevation of albumin in the urine from >30 to <300 mg/day is called microalbuminuria; which is known to be an early indicator of diabetic glomerular disease.
ANNEXURES
**Annexures**

**ANNEXURE 1**

**LABORATORY VALUES**

<table>
<thead>
<tr>
<th>Laboratory Value</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine (CR)</td>
<td>Creatinine is a waste product of muscle breakdown. Healthy kidneys remove creatinine from the blood. With kidney failure, creatinine builds in the blood until it is removed by dialysis. A high level of creatinine may cause itching and damage to nerve endings. High creatinine levels may cause numbness and tingling of toes.</td>
</tr>
<tr>
<td>Blood Urea Nitrogen (BUN)</td>
<td>The BUN is a waste product of protein breakdown. The more protein a person eats, the more urea is produced. The BUN will go up if a person eats large amounts of protein. Eating smaller quantities of higher quality protein will provide adequate nutrition required while cutting down on protein waste build-up. Other factors can also raise BUN such as infection.</td>
</tr>
<tr>
<td>Potassium (K)</td>
<td>Potassium regulates muscle action. The heart is a muscle which is sensitive to potassium. Too much or too little potassium can cause the heart to stop. Salt substitutes are to be avoided because they are made from potassium.</td>
</tr>
<tr>
<td>Chloride (Cl)</td>
<td>Chloride is necessary for nerves and muscle to work together.</td>
</tr>
<tr>
<td>Sodium (Na)</td>
<td>Sodium is necessary for maintaining the body's cells and water. The higher the level of sodium in the blood, the more water is retained by the body. Too much sodium will increase thirst, enhance swelling (called edema), and cause shortness of breath. Extra sodium and water in the body may cause the blood pressure to rise.</td>
</tr>
<tr>
<td>Calcium (Ca)</td>
<td>Calcium is stored in the bones and is needed to keep them strong. If the calcium level is too low, the body will steal calcium from the bones, causing them to be weak. High calcium levels may be a sign of a problem but are not generally associated with dialysis.</td>
</tr>
<tr>
<td>Test</td>
<td>Description</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Phosphorus (P)</strong></td>
<td>Phosphorus is made by protein breakdown and is also found in many foods. It is a chemical that works with calcium to keep bones strong. Often a high phosphorus level will cause low calcium level and vice versa. Dialysis alone may not be enough to lower the phosphorus level in the blood. A medicine called a phosphorus binder may be taken with meals to control the phosphorus level. Constant high phosphorus levels lead to bone disease, itching, and other skin problems.</td>
</tr>
<tr>
<td><strong>Alkaline Phosphatase (Alk.Phos.)</strong></td>
<td>This is an enzyme that comes from the liver and bones. High levels may indicate liver or bone disease.</td>
</tr>
<tr>
<td><strong>Albumin &amp; Total Protein</strong></td>
<td>These levels are measured because they relate to nutritional or protein status.</td>
</tr>
<tr>
<td><strong>Glucose</strong></td>
<td>Glucose, or blood sugar, is necessary for energy and maintaining normal body tissue.</td>
</tr>
<tr>
<td><strong>Hematocrit (HCT)</strong></td>
<td>The hematocrit is the amount of red blood cells compared to water (plasma) in the blood. Chronic renal failure causes the hematocrit to go down because the red blood cells do not live as long, and the kidneys no longer make the hormone that helps to make red blood cells. If the hematocrit becomes too low, a blood transfusion may be necessary. Most patients receive synthetic erythropoietin to help with this problem.</td>
</tr>
<tr>
<td><strong>Hemoglobin (HB)</strong></td>
<td>It is a protein, carried by red blood cells, which transport oxygen through the body.</td>
</tr>
</tbody>
</table>
### BLOOD LEVELS FOR DIALYSIS PATIENTS

<table>
<thead>
<tr>
<th>Blood Parameter</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Urea Nitrogen (BUN)</td>
<td>60 – 110 mg/dl</td>
</tr>
<tr>
<td>Creatinine</td>
<td>8.0 – 20.0 mg/dl</td>
</tr>
<tr>
<td>Potassium (K)</td>
<td>3.5 – 5.0 mEq/L</td>
</tr>
<tr>
<td>Calcium (Ca)</td>
<td>8.5 – 10.5 mg/dl</td>
</tr>
<tr>
<td>Blood Sugar (fasting)</td>
<td>60 – 100 mg/dl</td>
</tr>
<tr>
<td>Blood Sugar</td>
<td>less than 140 mg/dl</td>
</tr>
<tr>
<td>Alkaline Phosphatase</td>
<td>25 – 100 units/L</td>
</tr>
<tr>
<td>Phosphorus (P)</td>
<td>2.3 – 4.7 mg/dl</td>
</tr>
<tr>
<td>Sodium</td>
<td>135 – 145 mEq/L</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.8 – 5.5 gm/dl</td>
</tr>
<tr>
<td>Total Protein</td>
<td>6.0 – 8.0 gm/dl</td>
</tr>
<tr>
<td>Hematocrit (HCT)</td>
<td>33% - 36%</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>11 -12</td>
</tr>
</tbody>
</table>
## ANNEXURE 3

### LABORATORY TESTS RECOMMENDED FOR DIALYSIS PATIENTS

<table>
<thead>
<tr>
<th>Laboratory Test</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine Analysis</td>
<td>Includes urine glucose, urine protein, white blood cells, red blood cells, and microscopic evaluation. Normal reference range: negative.</td>
</tr>
<tr>
<td>Urine Albumin Excretion Rate (UAER)</td>
<td>The amount of albumin that is excreted in the urine over a given period. An increase in the albumin excretion rate indicates the glomeruli are functioning abnormally. Normal UAER reference range: &lt;30mg/24h or 20 microgram/min.</td>
</tr>
<tr>
<td>Urinary Albumin: Creatinine Ratio (UACR)</td>
<td>Measurement of the albumin to creatinine ratio in a random, clean catch urine sample. Normal UACR reference ranges: &lt;17mg/g (men) or &lt;25 mg/g (women).</td>
</tr>
<tr>
<td>Urine Protein: Creatinine (UPC)</td>
<td>This test is performed on a random, clean catch urine sample. Macroalbuminuria, also called clinical proteinuria, is defined by a UPC of ( \geq 200 \text{ mg} / \text{g} ).</td>
</tr>
<tr>
<td>24-Hour Creatinine Clearance</td>
<td>(Requires a 24 hour urine collection plus a serum sample). This was historically the 'golden standard' to detect renal dysfunction and to monitor renal function. However, due to difficulty of collection, the estimated GFR is now recommended as the best estimate of kidney function, and to monitor progression of CKD. Normal creatinine clearance reference range: 85-135ml/min.</td>
</tr>
<tr>
<td>Blood Urea Nitrogen (BUN)</td>
<td>The blood level of urea, which is the end product of protein metabolism. BUN increases as kidney function deteriorates. However, increased levels are also</td>
</tr>
</tbody>
</table>

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Annexures

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum Creatinine (Scr)</td>
<td>Creatinine is a nitrogenous waste product formed from the metabolism of muscle and dietary protein. Levels rise as kidney functions deteriorate, but eventually stabilize. A serum creatinine level should be measured at least annually for the estimation of GFR in all adults with diabetes regardless of the degree of albumin excretion. Serum creatinine alone should not be used as a measure of kidney function, but used to estimate GFR and stage the level of CKD. Normal reference range: 0.5 – 1.4 mg/dl.</td>
</tr>
<tr>
<td>Estimated Glomerular Filtration Rate (GFR)</td>
<td>A serum creatinine level should be measured at least annually for the estimation of GFR in all adults with diabetes regardless of the degree of albumin excretion. Normal reference range: 110 – 130 mg/ml/1.73m²</td>
</tr>
</tbody>
</table>
# ANNEXURE 4

## RECOMMENDED DIETARY NUTRIENT INTAKE FOR PATIENTS WITH CHRONIC RENAL FAILURE NOT UNDERGOING DIALYSIS

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>GFR &lt; 25ml/min: 0.6 g protein/kg/day; if patient unable to maintain adequate intake of calories or will not accept this diet, 0.75g protein/kg/day.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (g/kg/day)</td>
<td>GFR &lt; 25ml/min: 35 kcalories/kg/day if &lt; 60 years of age; 30-35 kcalories/kg/day if ≥ 60 years of age.</td>
</tr>
<tr>
<td>Fat (Percentage of total energy intake)</td>
<td>30</td>
</tr>
<tr>
<td>Polynsaturated/saturated fatty acid ratio</td>
<td>1.0:1.0</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>Remainder of non-protein calories</td>
</tr>
<tr>
<td>Total fiber intake (g/day)</td>
<td>20-25</td>
</tr>
<tr>
<td>Minerals (range of intake)</td>
<td>1000-3000g</td>
</tr>
<tr>
<td>Sodium (mg/day)</td>
<td>40-70</td>
</tr>
<tr>
<td>Potassium (mEq/day)</td>
<td>5-10</td>
</tr>
<tr>
<td>Phosphorus (mg/kg/day)</td>
<td>1400-1600</td>
</tr>
<tr>
<td>Calcium (mg/kg/day)</td>
<td>200-300</td>
</tr>
<tr>
<td>Magnesium (mg/day)</td>
<td>≥ 10-18</td>
</tr>
<tr>
<td>Iron (mg/day)</td>
<td>15</td>
</tr>
<tr>
<td>Zinc (mg/day)</td>
<td>Up to 3000 as tolerated</td>
</tr>
<tr>
<td>Water (mg/day)</td>
<td>Renal formulated multivitamin supplement</td>
</tr>
</tbody>
</table>

Ref: Independent study module series, 2006; Diabetes and Kidney diseases. Revised by: Diane Deutsch-Keaby PhD, RD, MT; Carolyn Jennings, MPH, KD, CDE. Reviewed by: Maurie Ferriter, Jean Chickering, RN, MSN; Paula Ackerman, MS, RD, CDE)
## ANNEXURE 5

### NUTRITIONAL RECOMMENDATIONS FOR PATIENTS ON HEMODIALYSIS AND PERITONEAL DIALYSIS THERAPY

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Consultation (g/kg)*</th>
<th>Consultation (g/kg)*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Protein (g/kg)</strong></td>
<td>1.2-1.3; at least 50% high biologic value</td>
<td>1.2-1.3; at least 50% high biologic value</td>
</tr>
<tr>
<td><strong>Energy (kcal/kg or KJ/kg)</strong>*</td>
<td>30-35 (125-145) if ≥ 60 years of age and 35 (145) if &lt;60 years of age</td>
<td>30-35 (125-145) if ≥ 60 years of age and 35 (145) if &lt;60 years of age</td>
</tr>
<tr>
<td><strong>Phosphorus</strong></td>
<td>800-1200 mg/day or 17 mg/kg*</td>
<td>1200 mg/day or 17 mg/kg*</td>
</tr>
<tr>
<td><strong>Sodium</strong></td>
<td>2000-3000 mg/day (88 – 130 mmol/day)</td>
<td>Individualized based on blood pressure and weight</td>
</tr>
<tr>
<td><strong>Potassium</strong></td>
<td>40 mg/kg* or approximately 2000-3000 mg/day (50-80 mmol/day)</td>
<td>Generally unrestricted with CAPD and APD: 3000-4000 mg/day (130-175 mmol/day) unless serum level is increased or decreased.</td>
</tr>
<tr>
<td><strong>Fluid</strong></td>
<td>500-1000 ml/day + daily urine output.</td>
<td>CAPD and APD approx. 2000-3000 ml/day base on daily wt fluctuations, urine output, ultrafiltration and blood pressure; unrestricted if wt. and BP are controlled and residual renal function = 2-3L/day.</td>
</tr>
<tr>
<td><strong>Calcium</strong></td>
<td>1000-1800 mg/day</td>
<td>1000-1800 mg/day</td>
</tr>
</tbody>
</table>

* If patient is < 90% or >115% of median standard weight, use adjusted body weight.

Ref: Independent study module series, 2006; Diabetes and Kidney diseases. Revised by: Diane Deutsch-Keaby PhD, RD, MT; Carolyn Jennings, MPH, KD, CDE. Reviewed by: Maurie Ferriter, Jean Chickering, RN, MSN; Paula Ackerman, MS, RD, CDE.

---

Annexures
## NUTRITION GUIDELINES FOR ADULT KIDNEY TRANSPLANT RECIPIENTS

<table>
<thead>
<tr>
<th>NUTRIENT</th>
<th>PRESCRIPTION</th>
<th>PURPOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First 6-8 weeks after transplantation and during treatment for acute rejection</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>1.3-1.5 g/kg</td>
<td>Counteract protein catabolism; promote wound healing. Meet post surgery energy demands and allows protein to be used for anabolism</td>
</tr>
<tr>
<td>Calories</td>
<td>30-35k calories (125 – 145 KJ), Kg</td>
<td></td>
</tr>
<tr>
<td><strong>After 6-8 weeks</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>1.0g/kg</td>
<td>Minimize muscle protein wasting. Achieve/maintain optimal weight</td>
</tr>
<tr>
<td>Calories</td>
<td>Sufficient to achieve optimal weight for height</td>
<td></td>
</tr>
<tr>
<td><strong>At all times</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>Consistent carbohydrate intake; increase fiber content</td>
<td>Meet energy demands; promote bowel regularity</td>
</tr>
<tr>
<td>Fats</td>
<td>No more than 30% of calories;</td>
<td>Reduce post transplant hyperlipidemia; helps prevent progression of atherosclerosis</td>
</tr>
<tr>
<td>Potassium</td>
<td>Variable; restrict or supplement as necessary based on serum level</td>
<td>Maintain acceptable potassium level</td>
</tr>
<tr>
<td>Sodium</td>
<td>2-4g (87-175 mmol) must be necessary</td>
<td>Maintain blood pressure; minimize edema</td>
</tr>
<tr>
<td>Calcium</td>
<td>may be necessary 1000-1500 mg</td>
<td>Minimize further bone demineralization; correct calcium/phosphorus imbalance</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>1200-1500mg; some patients may require supplements</td>
<td>Minimize further bone demineralization; correct calcium/phosphorus imbalance</td>
</tr>
<tr>
<td>Fluids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ad libitum</td>
<td>demineralization; correct calcium/phosphorus imbalance Maintain adequate hydration</td>
<td></td>
</tr>
</tbody>
</table>

Ref: Independent study module series, 2006; Diabetes and Kidney diseases. Revised by: Diane Deutsch-Keaby PhD, RD, MT; Carolyn Jennings, MPH, KD, CDE. Reviewed by: Maurie Ferrier, Jean Chickering, RN, MSN; Paula Ackerman, MS, RD, CDE)
### ANNEXURE 7

**RECOMMENDED DAILY INTAKE OF SELECTED VITAMINS**

| VITAMIN         | MALES          | FEMALES*       | COMMENTS                                                        |
|-----------------|----------------|----------------|*****************************************************************|
| Vitamin A       | 750 µg retinol equivalents | 750 µg retinol equivalents |                                                                |
| (retinol)       |                 |                 |                                                                  |
| Vitamin B1      | 0.9-1.1 mg      | 0.7-0.8 mg      | See note below if administering protein restricted diet         |
| (thiamine)      |                 |                 |                                                                  |
| Vitamin B2      | 1.3-1.7 mg      | 1.0-1.2 mg      | See note below if administering protein restricted diet         |
| (riboflavin)    |                 |                 |                                                                  |
| Niacin          | 16-19 mg niacin equivalents | 11-13 mg niacin equivalents | See note regards homocystine, and if on erythropoietin therapy |
| Folate          | 200 µg          | 200 µg          | See note regards if administering protein restricted diet       |
| Vitamin B6      | 1.0-1.9 mg      | 0.8-1.4 mg      | See note regards if administering protein restricted diet       |
| (pyridoxine)    |                 |                 |                                                                  |
| Vitamin B12     | 2.0 µg          | 2.0 µg          |                                                                  |
| (cobalamin)     |                 |                 |                                                                  |
| Vitamin C       | 40 mg           | 30 mg           | May use more to aid iron absorptionss                           |
| (ascorbic acid) |                 |                 |                                                                  |
| Vitamin E       | 10 mg (α-tocopherol equivalents) | 7 mg (α-tocopherol equivalents) |                                                                |
| (tocopherol)    |                 |                 |                                                                  |

* Additional supplementation is recommended for pregnant and lactating women

Ref: Caring for Australians with Renal Impairments- Nutrition and growth in the kidney disease. Vitamins in Pre-dialysis patients. Author: David Voss
## ANNEXURE 8

### DIFFERENCE BETWEEN ALLOPATHIC AND ALTERNATIVE MEDICINE

<table>
<thead>
<tr>
<th>S.NO.</th>
<th>ALLOPATHIC</th>
<th>ALTERNATIVE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Allopathic medicine is preferred in the treatment of trauma and emergencies</td>
<td>Alternative medicine excels in the treatment of chronic disease, although homeopathy can also be very effective as a first-aid.</td>
</tr>
<tr>
<td>2</td>
<td>Allopathic medicine focuses on the relief of symptoms and rarely places emphasis on prevention or the treatment of the cause of a disorder.</td>
<td>All alternative systems, on the other hand, strive to find and treat the cause of a disorder and frown on covering up the symptoms. Alternative therapies are also much more focused on prevention.</td>
</tr>
<tr>
<td>3</td>
<td>Allopathic medicine is organ specific, hence ophthalmologists, cardiologists, nephrologists, neurologists, etc.</td>
<td>Alternative medicine, without exception, considers each person as a unique individual and uses a holistic approach in treatment.</td>
</tr>
<tr>
<td>4</td>
<td>Allopathic medicine believes in aggressive intervention to treat disease. It revels in terms such as &quot;magic bullet&quot; and &quot;war&quot; (&quot;the war on cancer&quot;), and prefers quick fixes (as do many patients).</td>
<td>Alternative medicine believes in gentle, long-term support to enable the body's own innate powers to do the healing.</td>
</tr>
<tr>
<td>5</td>
<td>Allopathic medicine's main &quot;arsenal&quot; consists of surgery, chemotherapy, radiation, and powerful pharmaceutical drugs.</td>
<td>Alternative medicine uses time-tested, natural remedies and gentle, hands-on treatments.</td>
</tr>
<tr>
<td>6</td>
<td>Allopathic medicine practitioners are guided in their treatment by strict</td>
<td>Practitioners of alternative medicine, on the other hand, treat each patient as an</td>
</tr>
<tr>
<td>7</td>
<td>Allopathic medicine sees the body as a mechanical system (the heart is a pump and the kidneys are a filter) and believes most disorders can be traced to chemical imbalances and therefore are best treated with powerful chemicals (drugs).</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alternative medicine systems, almost without exception, accept that the body is suffused by a network of channels (meridians) that carry a subtle form of life energy. Imbalances or blockages of this energy are what lead to disease and clearing of the blockages and strengthening of the energy is the ultimate goal of alternative medicine.</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Allopathic medicine prefers patients to be passive and accept their treatment without too many questions.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alternative medicine, in contrast, prefers and indeed, in many cases, requires the patient to take a highly active part in both prevention and treatment.</td>
<td></td>
</tr>
</tbody>
</table>
ANNEXURE 9

DIFFERENCE BETWEEN ALLOPATHIC AND HOMEOPATHIC SYSTEM OF TREATMENT

<table>
<thead>
<tr>
<th>ALLOPATHIC TREATMENT</th>
<th>HOMEOPATHIC SYSTEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) In comparison to lifelong treatment in allopathy.</td>
<td>1) Homeopathic medicines can be stopped in 2 to 5 years depending on the severity and underlying cause of the disease.</td>
</tr>
<tr>
<td>2) Damaged Kidneys cannot be repaired in allopathy. Modern medicine try to facilitate the bodily waste products to pass out by the process of dialysis through artificial or by transplanted natural kidney and simultaneously by doing forced diuresis with the help of tab. Lasix, Dytor etc. that eventually lead to more damage to the remaining renal cells.</td>
<td>2) Though Homeopathy does not provide any substitute (dialysis or new Kidney) for any organ (Kidney) but it can revive the damaged organ as it has been proved at many times in TB cases where normal tissue are found in place of fibrocavitory lesions (Completely damaged tissue).</td>
</tr>
<tr>
<td>3) The process of dialysis could not be stopped once a patient starts on it. As the time passes this procedure has to done more frequently. Even doctors cannot imagine that it can be slow down (gap in dialysis can be increased) so they (doctors) compel their patient to come for dialysis more frequently.</td>
<td>3) Patient’s kidney starts improving as soon as he starts taking homeopathic treatment. It helps the patient withdrawing from dialysis. That’s why homeopath advises his patient to increase gap in the subsequent dialysis.</td>
</tr>
<tr>
<td>4) There is no curative procedure for CRF in modern medicine. Kidney transplantation and Dialysis does not cure renal failure, but instead keeps a person</td>
<td>4) But homeopathy stimulates someone’s immune system to perform normal functions, in this way damaged kidneys and other organ’s functions start</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>alive by performing the crucial functions of the kidneys.</td>
<td>improving.</td>
</tr>
<tr>
<td>5) There is very high percentage of rejection of new kidney.</td>
<td>5) If such patient starts taking homeopathic treatment that would minimize the rejection percentage.</td>
</tr>
<tr>
<td>6) In allopathic system patient has to spend Rs.5000 to 20000/- per month exclusively on medicines. Expenses of hospital’s fee and investigations are superfluous.</td>
<td>6) Homeopathic treatment cost depends on the severity of disease and knowledge and experience of the doctor, even a costliest doctor's treatment remains quite economical than allopathic expenses.</td>
</tr>
<tr>
<td>7) In case of kidney transplanted patients kidney’s linked diseases like Diabetes mellitus and Hypertension may become uncontrolled instead of taking full medical care.</td>
<td>7) Classical Homeopathic treatment not only repairs the damaged kidneys but simultaneously it helps in maintaining the blood sugar level and blood pressure to a normal level.</td>
</tr>
<tr>
<td>8) In allopathic system damage is a continuous process; it can be slow down up to a certain limit but cannot be stopped.</td>
<td>8) Very well selected Homeopathic medicine can stop the further damage of diseased organ without delay and further treatment may revive the damaged tissue.</td>
</tr>
<tr>
<td>9) In the similar way it can not treat (correct) the cysts or tumors of kidneys.</td>
<td>9) It can treat the tumors as well as polycystic kidneys also.</td>
</tr>
<tr>
<td>10) Doctors and patient always remain worried about the bio-chemistry (Sodium, potassium, calcium, phosphorus, etc.) otherwise some complications may likely to occur. Even with taking care of all the measures patient starts complaining of symptoms related to electrolyte</td>
<td>10) Once patient's immunity improves, all organs' functions will start improving simultaneously bio-chemistry also becomes normal naturally.</td>
</tr>
<tr>
<td>Annexures</td>
<td></td>
</tr>
<tr>
<td>-----------</td>
<td></td>
</tr>
<tr>
<td>imbalance almost every day.</td>
<td></td>
</tr>
<tr>
<td>11) Doctors (except classical homeopaths) remain busy in managing one or the other problem.</td>
<td></td>
</tr>
<tr>
<td>11) Classical Homeopaths have to take lot of time for the first prescription then things become quite easier in comparison to other doctors.</td>
<td></td>
</tr>
<tr>
<td>12) Allopathic doctors know that they have very limited role in such cases so they keep on experimenting on different food supplements etc.</td>
<td></td>
</tr>
<tr>
<td>12) While Homeopaths have enough evidences of more damage to the remaining nephrons by experimenting with any artificial food material or chemicals.</td>
<td></td>
</tr>
</tbody>
</table>
## ANNEXURE 10
### GLOSSARY OF TERMS

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdomen</td>
<td>The part of the body containing the internal organs. Lies between the pelvis and the chest cavity.</td>
</tr>
<tr>
<td>Access</td>
<td>A means to get into the body. Accesses to the bloodstream for hemodialysis are fistulas, grafts, catheter, etc. Access to the peritoneal cavity for peritoneal dialysis is a catheter.</td>
</tr>
<tr>
<td>Acute</td>
<td>Rapidly developing; severe; short duration.</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>Sudden and severe decrease in kidney function that is short term.</td>
</tr>
<tr>
<td>Ambulatory</td>
<td>Able to walk; movable.</td>
</tr>
<tr>
<td>Anemia</td>
<td>A condition in which there is a reduction of red blood cells.</td>
</tr>
<tr>
<td>Antibiotic</td>
<td>Medication used to kill bacteria and fight infection.</td>
</tr>
<tr>
<td>Anticoagulant</td>
<td>Medication that delays or stops the clotting of blood such as Coumadin (or heparin).</td>
</tr>
<tr>
<td>Antihypertensive</td>
<td>Medication that lowers blood pressure.</td>
</tr>
<tr>
<td>Antiseptic</td>
<td>Chemical that stops growth and reproduction of bacteria and viruses, but doesn’t necessarily destroy them as a disinfectant would.</td>
</tr>
<tr>
<td>Arterial line</td>
<td>A tube that carries blood away from the body into the dialyzer.</td>
</tr>
<tr>
<td>Arteriosclerosis</td>
<td>Hardening of the arteries.</td>
</tr>
<tr>
<td>Artery</td>
<td>A blood vessel carrying blood away from the heart to other parts of the body.</td>
</tr>
<tr>
<td>Artificial kidney</td>
<td>A filtering device used with an artificial kidney machine to remove excess fluid and waste products from the body. Also known as “dialyzer” and “hemodialyzer.”</td>
</tr>
<tr>
<td>Artificial kidney</td>
<td>A machine that supports and monitors the functioning of the artificial kidney (dialyzer). Also called “hemodialysis machine.”</td>
</tr>
<tr>
<td>Arterioseptic</td>
<td>Chemical that stops growth and reproduction of bacteria and viruses, but doesn’t necessarily destroy them as a disinfectant would.</td>
</tr>
<tr>
<td>Bacteria</td>
<td>Single cell organisms, capable of rapid reproduction. They are present everywhere; some are harmless, others are known to produce infectious diseases.</td>
</tr>
<tr>
<td>Bath</td>
<td>See “dialysate.”</td>
</tr>
<tr>
<td><strong>Annexures</strong></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td></td>
</tr>
<tr>
<td><strong>Blood chemistries</strong></td>
<td>Measurement of certain chemicals in the blood.</td>
</tr>
<tr>
<td><strong>Blood flow rate</strong></td>
<td>In dialysis, the rate at which the patient’s blood is pumped through the artificial kidney.</td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td>Pressure blood exerts on the walls of the blood vessels. This is expressed in two numbers, such as 120/80. The systolic (top) number is the pressure when the heart is contracting. The diastolic (bottom) number is the pressure when the heart is at rest.</td>
</tr>
<tr>
<td><strong>Blood pump</strong></td>
<td>A pump that moves blood from the patient’s access through the blood tubing to an artificial kidney and back to the patient without damage to the blood cells.</td>
</tr>
<tr>
<td><strong>Bruit</strong></td>
<td>Sound produced by the blood flowing through a graft, fistula, or shunt.</td>
</tr>
<tr>
<td><strong>BUN (blood urea nitrogen)</strong></td>
<td>Combination of waste products (nitrogen and urea) in the blood normally excreted by the kidneys.</td>
</tr>
<tr>
<td><strong>Cadaver donor</strong></td>
<td>Someone who has died and whose kidneys have been donated for transplantation.</td>
</tr>
<tr>
<td><strong>Calcium</strong></td>
<td>Element found in the body. Important to bone growth and formation, blood clotting, nerve and muscle functioning and the activation of certain enzymes.</td>
</tr>
<tr>
<td><strong>Carbohydrates</strong></td>
<td>Category of food that is easily used by the body for energy.</td>
</tr>
<tr>
<td><strong>Cardiac</strong></td>
<td>Anything to do with the heart.</td>
</tr>
<tr>
<td><strong>Catheter</strong></td>
<td>A flexible, hollow tube through which fluids enter or leave the body. A catheter is implanted in the abdomen for peritoneal dialysis.</td>
</tr>
<tr>
<td><strong>Chronic</strong></td>
<td>Of long duration or recurring often.</td>
</tr>
<tr>
<td><strong>Chronic renal (kidney) failure</strong></td>
<td>Damage to the kidneys that cannot be reversed, usually progressive in nature.</td>
</tr>
<tr>
<td><strong>Clearance</strong></td>
<td>Rate that waste products in the blood are removed through dialysis expressed in milliliters/minute.</td>
</tr>
<tr>
<td><strong>Coagulation</strong></td>
<td>The process of forming a blood clot.</td>
</tr>
<tr>
<td><strong>Concentration</strong></td>
<td>Strength of a solution.</td>
</tr>
<tr>
<td><strong>Congestive heart failure</strong></td>
<td>A condition in which the body is fluid overloaded, causing the heart to pump less effectively. Congestive heart failure may</td>
</tr>
<tr>
<td>Term</td>
<td>Definition</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>---------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Contaminate</td>
<td>Allowing microorganisms to contact a sterile area, making it unsterile and creating potential for infection.</td>
</tr>
<tr>
<td>Continuous Ambulatory Peritoneal Dialysis (CAPD)</td>
<td>A form of dialysis in which dialysate drains into and out of the peritoneal cavity by gravity several times a day.</td>
</tr>
<tr>
<td>Continuous Cycling Peritoneal Dialysis (CCPD)</td>
<td>A form of dialysis that uses a cycling machine to infuse and drain dialysate from the peritoneal cavity several times during the night while the patient sleeps.</td>
</tr>
<tr>
<td>Convulsion</td>
<td>Involuntary muscle contractions and relaxation.</td>
</tr>
<tr>
<td>Creatinine</td>
<td>One of the many waste products in the blood produced by normal tissue breakdown and removed by the kidneys, or by dialysis in patients with renal failure.</td>
</tr>
<tr>
<td>Cross-matching</td>
<td>Testing of blood and tissues to check compatibility of donor kidney and patient for kidney transplantation.</td>
</tr>
<tr>
<td>Culture</td>
<td>A sample of organisms taken from a site to identify the specific organism causing infection.</td>
</tr>
<tr>
<td>Dialysate</td>
<td>Solution containing water and chemicals (electrolytes) that passes through the artificial kidney to remove excess fluids and wastes from the blood, also call “bath.”</td>
</tr>
<tr>
<td>Dialysis</td>
<td>Cleansing the body of unwanted toxins, waste products and excess fluid by filtering them from the blood through a semi permeable membrane.</td>
</tr>
<tr>
<td>Dialysate flow rate</td>
<td>Rate at which dialysate flows through the dialyzer.</td>
</tr>
<tr>
<td>Dialysis treatment plan</td>
<td>Regimen based on the individual needs of the renal failure patient to reestablish physical balance.</td>
</tr>
<tr>
<td>Dialyzer</td>
<td>See “artificial kidney”.</td>
</tr>
<tr>
<td>Diffusion</td>
<td>Passage of particles from an area of high concentration to a solution of low concentration resulting in an even distribution of particles.</td>
</tr>
<tr>
<td>Disinfectant</td>
<td>An agent which will kill most microorganisms (bacteria, viruses) it contacts.</td>
</tr>
</tbody>
</table>
**Annexures**

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dry weight</strong></td>
<td>The weight of a dialysis patient when excess fluid has been removed. Also known as “ideal weight”.</td>
</tr>
<tr>
<td><strong>Dwell time</strong></td>
<td>Length of time dialysis solution stays in the peritoneal cavity during peritoneal dialysis.</td>
</tr>
<tr>
<td><strong>Edema</strong></td>
<td>Swelling or puffiness due to buildup of excess fluid in the tissues most noticeable in ankles, hands and face. Occurs in dialysis patients as a result of excess fluid intake or decreased ultrafiltration.</td>
</tr>
<tr>
<td><strong>End-stage-renal disease (ESRD)</strong></td>
<td>Stage of kidney damage that requires dialysis or kidney transplantation to maintain life. Another term for permanent kidney failure.</td>
</tr>
<tr>
<td><strong>Exchange</strong></td>
<td>The process of changing used dialysate for fresh solution in peritoneal dialysis.</td>
</tr>
<tr>
<td><strong>Exit site</strong></td>
<td>The area where the needles enter or exit through the access. Also, where a peritoneal dialysis catheter or sub clavian catheter exits the skin.</td>
</tr>
<tr>
<td><strong>Fistula</strong></td>
<td>The surgical joining of an artery and a vein so that the vein enlarges due to the flow of arterial blood. A fistula is a type of access, also known as an ‘arteriovenous fistula’.</td>
</tr>
<tr>
<td><strong>Fluid overload</strong></td>
<td>Point at which extra fluid in the body causes edema, difficulty in breathing or extra strain on the heart.</td>
</tr>
<tr>
<td><strong>Glomerulonephritis</strong></td>
<td>Inflammation of the kidney’s filters (glomeruli).</td>
</tr>
<tr>
<td><strong>Graft</strong></td>
<td>In dialysis, surgical placement of a material between an artery and vein to create a circulatory access for hemodialysis. Graft also refers to a transplanted kidney.</td>
</tr>
<tr>
<td><strong>Hematocrit</strong></td>
<td>Ration of red blood cells to whole blood.</td>
</tr>
<tr>
<td><strong>Hemodialysis</strong></td>
<td>Removal of excess fluids and waste products by passage of blood through an artificial kidney.</td>
</tr>
<tr>
<td><strong>Hemodialyzer</strong></td>
<td>See “artificial kidney”.</td>
</tr>
<tr>
<td><strong>Hemoglobin</strong></td>
<td>Red, protein portion of the red blood cell which carries oxygen from lungs to body tissues.</td>
</tr>
<tr>
<td><strong>Heparin</strong></td>
<td>An anticoagulant given in hemodialysis to slow clotting time so that blood will not clot in the lines or dialyzer.</td>
</tr>
<tr>
<td><strong>Hepatitis</strong></td>
<td>Inflammation of the liver caused by virus, bacteria, toxic agents or medication.</td>
</tr>
<tr>
<td><strong>Hepatitis B surface antigen</strong></td>
<td>Substance that indicates infection with hepatitis B. Testing for this antigen is performed frequently on clinic staff and patients.</td>
</tr>
<tr>
<td><strong>Hypertension</strong></td>
<td>High blood pressure.</td>
</tr>
<tr>
<td><strong>Hypotension</strong></td>
<td>Low blood pressure.</td>
</tr>
<tr>
<td><strong>Immunosuppressive drug</strong></td>
<td>Type of medication that suppresses the body’s immune response. Given to transplant recipients to help prevent rejection of the transplanted kidney.</td>
</tr>
<tr>
<td><strong>Infection</strong></td>
<td>Invasion of the body by disease - producing organisms and the reaction of the tissues to their presence.</td>
</tr>
<tr>
<td><strong>Inflammation</strong></td>
<td>Localized heat, redness, swelling and pain; usually as a result of infection or irritation.</td>
</tr>
<tr>
<td><strong>Intravenous</strong></td>
<td>Within a vein.</td>
</tr>
<tr>
<td><strong>Kt/V</strong></td>
<td>Clearance X time / volume. Measure that indicates how well waste products have been removed by dialysis treatment.</td>
</tr>
<tr>
<td><strong>Kidneys</strong></td>
<td>Two bean-shaped organs located at the back of the abdominal cavity, one on each side of the spinal column. Kidneys maintain the body’s chemical balance by excreting waste products and excess fluid in the form of urine.</td>
</tr>
<tr>
<td><strong>Kilogram</strong></td>
<td>1,000 grams, one kilogram equals 2.2 pounds.</td>
</tr>
<tr>
<td><strong>Liter</strong></td>
<td>The basic unit of volume measurement in metric system, approximately equal to one quart.</td>
</tr>
<tr>
<td><strong>Membrane</strong></td>
<td>Thin layer of tissue or material, usually an outer layer or lining of organs or group of organs.</td>
</tr>
<tr>
<td><strong>Metabolism</strong></td>
<td>Physical and chemical changes occurring within the body in order to produce energy.</td>
</tr>
<tr>
<td><strong>Minerals</strong></td>
<td>Inorganic substances necessary to normal body function, but toxic in high concentrations.</td>
</tr>
<tr>
<td><strong>Monitor</strong></td>
<td>(noun) Electronic device used to check, remind, or warn. (verb) Watching patients during their treatments, or checking the adequacy of treatments over time.</td>
</tr>
<tr>
<td><strong>Nausea</strong></td>
<td>Upset stomach.</td>
</tr>
</tbody>
</table>
### Annexures

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nephrectomy</td>
<td>Surgical removal of the kidney.</td>
</tr>
<tr>
<td>Nephrologist</td>
<td>Doctor specializing in kidney disorders.</td>
</tr>
<tr>
<td>Nephron</td>
<td>Unit of kidney which maintains the body’s chemical balance. There are approximately one million nephrons in each kidney.</td>
</tr>
<tr>
<td>Neuropathy</td>
<td>Any disease of the nerves.</td>
</tr>
<tr>
<td>Nocturnal Hemodialysis</td>
<td>Is hemodialysis done at night while you sleep.</td>
</tr>
<tr>
<td>Osmosis</td>
<td>Movement of fluid through a semipermeable membrane to achieve equal concentration on both sides of the membrane.</td>
</tr>
<tr>
<td>Palpitation</td>
<td>Irregular beating of the heart.</td>
</tr>
<tr>
<td>Parathyroid glands</td>
<td>Small glands located in the neck that produce a hormone which regulates calcium and phosphorus levels in the blood.</td>
</tr>
<tr>
<td>Parathyroidectomy</td>
<td>Surgical removal of the parathyroid glands. Can be partial or complete.</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>Inflammation of the pericardium, the sac-like structure surrounding the heart.</td>
</tr>
<tr>
<td>PD Plus</td>
<td>A combination of CCPD and CAPD mostly used by PD patients who need more dialysis.</td>
</tr>
<tr>
<td>Peritoneal Cavity</td>
<td>Space surrounding the abdominal organs located under the abdominal muscles.</td>
</tr>
<tr>
<td>Peritoneal Dialysis</td>
<td>A form of dialysis in which the lining of the abdomen, the peritoneal membrane, acts as a natural filter.</td>
</tr>
<tr>
<td>Peritoneum (or Peritoneal membrane)</td>
<td>Smooth, semipermeable membrane that covers the abdominal organs and lines the abdominal cavity.</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>Infection of the peritoneal cavity that must be treated by a doctor.</td>
</tr>
<tr>
<td>Phosphate Binders</td>
<td>Medications that bind with dietary phosphorus so that it may be eliminated from the body; helps keep calcium and phosphorus in balance for dialysis patients.</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>An element necessary for normal body functions, especially bone formation.</td>
</tr>
<tr>
<td>Polycystic Kidney Disease</td>
<td>Hereditary disease involving growth of cysts on kidney tissue.</td>
</tr>
<tr>
<td>Potassium</td>
<td>An element needed by the body for normal muscle and nerve function and cell maintenance.</td>
</tr>
<tr>
<td><strong>Protein</strong></td>
<td>Group of nitrogen containing compounds found in the body that are essential to life.</td>
</tr>
<tr>
<td>------------------</td>
<td>-----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Pruritus</strong></td>
<td>Itching.</td>
</tr>
<tr>
<td><strong>Pulse</strong></td>
<td>Throbbing sensation felt in the arteries in time with the heartbeat.</td>
</tr>
<tr>
<td><strong>Red Blood Cell</strong></td>
<td>Type of blood cell containing hemoglobin which carries oxygen to the tissues.</td>
</tr>
<tr>
<td><strong>Renal</strong></td>
<td>Referring to the kidneys.</td>
</tr>
<tr>
<td><strong>Semipermeable membrane</strong></td>
<td>Material that allows only fluids and small particles to flow through.</td>
</tr>
<tr>
<td><strong>Sodium</strong></td>
<td>Element found in the body that helps regulate the body’s fluid content.</td>
</tr>
<tr>
<td><strong>Sterile</strong></td>
<td>Totally free from any living microorganisms.</td>
</tr>
<tr>
<td><strong>Thrill</strong></td>
<td>Pulsation (vibration or buzzing) felt over the graft or fistula.</td>
</tr>
<tr>
<td><strong>Tissue typing</strong></td>
<td>Matching the blood cells of potential transplant recipients with donors.</td>
</tr>
<tr>
<td><strong>Toxin</strong></td>
<td>Waste product in the blood or any substance that is poisonous.</td>
</tr>
<tr>
<td><strong>URR</strong></td>
<td>Urea reduction ratio. Percentage based on how much blood urea nitrogen (BUN) was removed during a dialysis treatment. Indicates how effectively urea and other waste products have been removed.</td>
</tr>
<tr>
<td><strong>Ultrafiltration</strong></td>
<td>Process used to remove excess fluid from the blood during dialysis.</td>
</tr>
<tr>
<td><strong>Urea</strong></td>
<td>Nitrogenous waste product formed during the breakdown of protein in the body.</td>
</tr>
<tr>
<td><strong>Uremia</strong></td>
<td>Buildup of waste products in the blood due to the inability of the kidneys to excrete them.</td>
</tr>
<tr>
<td><strong>Vascular</strong></td>
<td>Pertaining to blood vessels.</td>
</tr>
<tr>
<td><strong>Vein</strong></td>
<td>Blood vessel that carries blood from the parts of the body back to the heart.</td>
</tr>
<tr>
<td><strong>Venous</strong></td>
<td>Referring to veins and the flow of blood to the heart.</td>
</tr>
<tr>
<td><strong>Venous line</strong></td>
<td>Tube that carries blood from the dialyzer back to the body.</td>
</tr>
<tr>
<td><strong>Waste products</strong></td>
<td>Substances formed from the breakdown of protein in foods and from normal muscle tissue.</td>
</tr>
<tr>
<td><strong>White Blood Cell</strong></td>
<td>Type of blood cell that fights infection in the body.</td>
</tr>
</tbody>
</table>
NKF-DOQI CLASSIFICATION FOR CHRONIC KIDNEY DISEASE

<table>
<thead>
<tr>
<th>STATE</th>
<th>GLOMERULAR FILTRATION RATE (ml/minute/1.73m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Greater than or equal to 90 *</td>
</tr>
<tr>
<td>2</td>
<td>60-89</td>
</tr>
<tr>
<td>3</td>
<td>30-59</td>
</tr>
<tr>
<td>4</td>
<td>15-29</td>
</tr>
<tr>
<td>5</td>
<td>Less than 15 ( include patients on dialysis)</td>
</tr>
</tbody>
</table>

* CKD can be present with a normal or near normal GFR if other markers of kidney disease are present, such as proteinuria, hematuria, biopsy results showing kidney damage, or anatomic abnormalities. NKF-DOQI, National Kidney Foundation-Dialysis Outcomes Qualitative Initiative.
### RISK FACTORS ASSOCIATED WITH CHRONIC KIDNEY DISEASE

<table>
<thead>
<tr>
<th>SUSCEPTIBILITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advanced age</td>
</tr>
<tr>
<td>Reduced kidney mass</td>
</tr>
<tr>
<td>Low birth weight</td>
</tr>
<tr>
<td>Racial/ethnic minority</td>
</tr>
<tr>
<td>Family history of kidney disease</td>
</tr>
<tr>
<td>Low income or education</td>
</tr>
<tr>
<td>Systemic inflammation</td>
</tr>
<tr>
<td>Dyslipidemia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>INITIATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Autoimmune disease</td>
</tr>
<tr>
<td>Polycystic Kidney Disease</td>
</tr>
<tr>
<td>Drug toxicity</td>
</tr>
<tr>
<td>Urinary tract abnormalities (infections, obstruction, stones)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PROGRESSION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperglycemia: poor blood glucose control (in patients with diabetes)</td>
</tr>
<tr>
<td>Hypertension: Elevated blood pressure</td>
</tr>
<tr>
<td>Proteinuria</td>
</tr>
<tr>
<td>Tobacco smoking</td>
</tr>
</tbody>
</table>
ADVANTAGES AND DISADVANTAGES OF HEMODIALYSIS

<table>
<thead>
<tr>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Higher solute clearance allows intermittent treatment.</td>
<td>1. Requires multiple visits each week to the hemodialysis center, which translates into loss of control by the patient.</td>
</tr>
<tr>
<td>2. Parameters of adequacy of dialysis are better defined and therefore under dialysis can be detected early.</td>
<td>2. Disequilibrium, dialysis, hypotension, and muscle cramps are common. May require months before patient adjusts to hemodialysis.</td>
</tr>
<tr>
<td>3. The technique's failure rate is low.</td>
<td>3. Infections in hemodialysis patients may be related to the choice of membranes, the complement-activating membranes being more deleterious.</td>
</tr>
<tr>
<td>4. Even though intermittent heparinization is required, hemostasis parameters are better corrected with hemodialysis than peritoneal dialysis.</td>
<td>4. Vascular access is frequently associated with infection and thrombosis.</td>
</tr>
<tr>
<td>5. In-center hemodialysis enables closer monitoring of the patient.</td>
<td>5. Decline in residual renal function is more rapid compared to peritoneal dialysis.</td>
</tr>
</tbody>
</table>

## ANNEXURE 14

### ADVANTAGES AND DISADVANTAGES OF PERITONEAL DIALYSIS

<table>
<thead>
<tr>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. More hemodynamic stability (blood pressure) due to slow ultrafiltration rate.</td>
<td>1. Protein and amino acid losses through the peritoneum and reduce appetite owing to continuous glucose load and sense of abdominal fullness predispose to malnutrition.</td>
</tr>
<tr>
<td>2. May explain good clinical status in spite of lower urea clearance.</td>
<td>2. Risk of peritonitis.</td>
</tr>
<tr>
<td>3. Better preservation of residual renal function.</td>
<td>3. Catheter malfunction, and exit site and tunnel infection.</td>
</tr>
<tr>
<td>4. Convenient intraperitoneal route of administration of drugs such as antibiotic and insulin.</td>
<td>4. Inadequate ultrafiltration and solute dialysis in patients with a large body size, unless large volumes and frequent exchanges are employed.</td>
</tr>
<tr>
<td>5. Suitable for elderly and very young patients who may not tolerate hemodialysis well.</td>
<td>5. Patient burnout and high rate of technique failure.</td>
</tr>
<tr>
<td>6. Freedom from the 'machine' gives the patient a sense of independence.</td>
<td>6. Risk of obesity with excessive glucose absorption.</td>
</tr>
<tr>
<td>7. Less blood loss and iron deficiency, resulting in easier management of anemia or reduced requirements for erythropoietin and parenteral iron.</td>
<td>7. Mechanical problems such as hernias, dialysate leaks, hemorrhoids, or back pain may occur.</td>
</tr>
<tr>
<td>8. No systemic heparinization requirement.</td>
<td>8. Extensive abdominal surgery may preclude peritoneal dialysis.</td>
</tr>
<tr>
<td>9. Subcutaneous versus intravenous erythropoietin or darbepoietin is usual, which may reduce overall doses and be more physiologic.</td>
<td>9. No convenient access for intravenous iron administration.</td>
</tr>
</tbody>
</table>
ANNEXURE 15

LABORATORY TESTS FOR PATIENTS ON REGULAR HEMODIALYSIS

The following tests should be performed, usually monthly (unless clinically indicated more frequently). Blood tests should be taken pre-dialysis.

<table>
<thead>
<tr>
<th>Test</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea</td>
<td>Absolute measure difficult to interpret (diet, catabolism, GI bleeding, residual function, liver disease, alcoholism) URR and formal UKM need measuring rather than blood urea in isolation</td>
</tr>
<tr>
<td>Creatinine</td>
<td>Absolute value difficult to interpret (muscle mass, nutrition) Higher values associated with lower mortality overall because of association with better nutrition.</td>
</tr>
<tr>
<td>Albumin</td>
<td>Strongly associated with outcome. Mortality increases at all levels below 40 g/l. May be marker of nutrition or else reflection of inflammatory response (opposite of CRP)</td>
</tr>
<tr>
<td>Calcium and phosphate</td>
<td>Renal bone disease not cured by dialysis. Calcium may be high, low or normal, and varies with treatment with vitamin D analogues, phosphate binders, etc. Calcium in dialysate may need reducing in patients with hyper calcaemia. Patients with high phosphates need dietary advice, modification of phosphate binder use. Difficult to increase clearance with dialysis unless using hemofiltration</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>Should be maintained in normal range. Can increase dialysate bicarbonate in persistently acidaemic patients</td>
</tr>
<tr>
<td>PTH</td>
<td>Increased secretion controlled with vitamin D analogues or calcimimetics. Measure 2-6 monthly depending on need</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>Cardiovascular disease remains major cause of death. triglyceridaemia more common than hypercholesterolemia. Patients should be managed as for secondary prevention of cardiovascular disease</td>
</tr>
<tr>
<td><strong>CRP</strong></td>
<td>Increased levels significantly associated with mortality, regardless of cause. High levels indicate that a raised ferritin not necessarily a marker of replete iron stores</td>
</tr>
<tr>
<td><strong>Glucose</strong></td>
<td>Especially in patients from high-risk groups for developing diabetes e.g. Asians</td>
</tr>
<tr>
<td><strong>Hb</strong></td>
<td>Higher values may not be helpful (increased mortality in high-risk patients) but anemia associated with cardiac disease, intra-dialysis hypotension, bleeding</td>
</tr>
<tr>
<td><strong>Ferritin, iron, transferrin saturation</strong></td>
<td>For iron stores. Maintain ferritin levels 400-800 ng/ml (assuming CRP normal)</td>
</tr>
<tr>
<td><strong>Platelets</strong></td>
<td>Thrombocytopenia may be induced by heparin</td>
</tr>
<tr>
<td><strong>Liver function tests (AST, ALT, bilirubin)</strong></td>
<td>Marker of hepatitis (especially viral). Especially if patients have dialyzed in high-risk units (e.g. while on holiday)</td>
</tr>
<tr>
<td><strong>Virology (HBV, HCV)</strong></td>
<td>Often only measured 3 monthly. Patients should be immunized against HBV to minimize risk</td>
</tr>
<tr>
<td><strong>Cytotoxic antibodies</strong></td>
<td>For all patients on a transplant waiting list, especially after blood transfusions (usually 3 monthly)</td>
</tr>
<tr>
<td><strong>UKM, URR, Kru, nPCR</strong></td>
<td>Certainly 3 monthly, preferably monthly</td>
</tr>
</tbody>
</table>
### DIFFERENCES BETWEEN ACUTE AND CHRONIC RENAL FAILURE

<table>
<thead>
<tr>
<th>Acute Renal Failure</th>
<th>Chronic Renal Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myocardial infarction - a heart attack may lead to temporary kidney failure.</td>
<td>Diabetic nephropathy - diabetes can cause permanent changes, leading to kidney damage.</td>
</tr>
<tr>
<td>Rhabdomyolysis - kidney damage that can occur from muscle breakdown. This condition can occur from severe dehydration, infection, or other causes.</td>
<td>Hypertension - chronic high blood pressure (hypertension) can lead to permanent kidney damage.</td>
</tr>
<tr>
<td>Decreased blood flow to the kidneys for a period of time. This may occur from blood loss or shock.</td>
<td>Lupus (SLE) - a chronic inflammatory/autoimmune disease that can injure the skin, joints, kidneys, and nervous system.</td>
</tr>
<tr>
<td>An obstruction or blockage along the urinary tract.</td>
<td>An obstruction or blockage along the urinary tract.</td>
</tr>
<tr>
<td>Hemolytic uremic syndrome - usually caused by an E. coli infection, kidney failure develops as a result of obstruction to the small functional structures and vessels inside the kidney.</td>
<td>Alport syndrome - an inherited disorder that causes deafness, progressive kidney damage, and eye defects.</td>
</tr>
<tr>
<td>Ingestion of certain medications that may cause toxicity to the kidneys.</td>
<td>Nephrotic syndrome - a condition that has several different causes. Nephrotic syndrome is characterized by protein in the urine, low protein in the blood, high cholesterol levels, and tissue swelling.</td>
</tr>
<tr>
<td>Glomerulonephritis - a type of kidney disease that involves glomeruli. During glomerulonephritis, the glomeruli become inflamed and impair the kidney's ability to filter urine. Glomerulonephritis</td>
<td>Polycystic kidney disease - a genetic disorder characterized by the growth of numerous cysts filled with fluid in the kidneys.</td>
</tr>
</tbody>
</table>
Renal Failure: Its Treatment in Current Systems of Medicines

<table>
<thead>
<tr>
<th>may lead to chronic renal failure in some individuals.</th>
<th>Cystinosis - an inherited disorder in which the amino acid cystine (a common protein-building compound) accumulates within specific cellular bodies of the kidney, known as lysosomes.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any condition that may impair the flow of oxygen and blood to the kidneys such as cardiac arrest.</td>
<td>Interstitial nephritis or pyelonephritis - an inflammation to the small internal structures in the kidney.</td>
</tr>
</tbody>
</table>
### ADVANTAGES AND DISADVANTAGES OF KIDNEY TRANSPLANTATION

<table>
<thead>
<tr>
<th>ADVANTAGES</th>
<th>DISADVANTAGES</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) A transplanted kidney works like a normal kidney</td>
<td>1) The patients require major surgery.</td>
</tr>
<tr>
<td>2) The patients may feel healthier and “more normal.”</td>
<td>2) The patients may need to wait for a donor.</td>
</tr>
<tr>
<td>3) The patients have fewer diet restrictions.</td>
<td>3) The patient’s body may reject the new kidney, so one transplant may not last a lifetime.</td>
</tr>
<tr>
<td>4) The patients won’t need dialysis.</td>
<td>4) The patients will need to take immunosuppressants, which may cause complications.</td>
</tr>
<tr>
<td>5) Patients who successfully go through the selection process have a higher chance of living a longer life.</td>
<td></td>
</tr>
</tbody>
</table>
## ANNEXURE 18

### FUNCTIONS OF THE KIDNEY

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Functions of the Kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td><strong>Elimination</strong></td>
</tr>
<tr>
<td></td>
<td>Of waste/surplus</td>
</tr>
<tr>
<td></td>
<td>Carbohydrate-derived</td>
</tr>
<tr>
<td></td>
<td>Nitrogenous</td>
</tr>
<tr>
<td></td>
<td>Other</td>
</tr>
<tr>
<td>2.</td>
<td><strong>Control</strong></td>
</tr>
<tr>
<td></td>
<td>Of fluid and electrolyte</td>
</tr>
<tr>
<td></td>
<td>balance</td>
</tr>
<tr>
<td></td>
<td>Total body water</td>
</tr>
<tr>
<td></td>
<td>Plasma osmotic pressure</td>
</tr>
<tr>
<td></td>
<td>pH</td>
</tr>
<tr>
<td></td>
<td>Na, K, Ca, Mg, etc</td>
</tr>
<tr>
<td></td>
<td>Chloride, Bicarbonate</td>
</tr>
<tr>
<td>3.</td>
<td><strong>Hormonal Homeostasis</strong></td>
</tr>
<tr>
<td></td>
<td>Blood pressure</td>
</tr>
<tr>
<td></td>
<td>Calcium and bone metabolism</td>
</tr>
<tr>
<td></td>
<td>Red blood cell production</td>
</tr>
</tbody>
</table>
Outline

1. Time And Frequency

Guideline 1.1
   Dialysis should be delivered at least 3 times per week and the total duration should be at least 12 hr per week, unless supported by significant renal function.

Guideline 1.2
   An increase in treatment time and/or frequency should be considered in patients with hemodynamic or cardiovascular instability.

Guideline 1.3
   Dialysis treatment time and/or frequency should be considered in patients with impaired phosphate control.

Guideline 1.4
   An increase of treatment time and/or frequency should be considered in patients with impaired phosphate control.

Guideline 1.5
   An increase of dialysis time and frequency should be considered in malnourished patients.

2. Flux And Convection

Guideline 2.1
   The use of synthetic high flux membranes should be considered to delay long-term complications of haemodialysis therapy. Specific indications include:
   i. To reduce dialysis – related amyloidosis (III)
   ii. To improve control of hyperphosphataemia (II)
   iii. To reduce the increased cardiovascular risk (II)
   iv. To improve control of anaemia (III)
Guideline 2.2
In order to exploit the high permeability of high-flux membranes, on-line haemodiafiltration or haemofiltration should be considered. The exchange volumes should be as high as possible, with consideration of safety (evidence Level II).

Dialysis Dose Methodology

Guideline 3.1
Delivered dialysis dose should be measured at least monthly.

Guideline 3.2
Dialysis dose should be measured using a validated method comparable with the reference method. The reference method is formal urea kinetic modeling using pre and post dialysis blood samples and taking ultrafiltration urea generation and the post dialysis rebound into account.

Guideline 3.3
Renal function may be taken into account in the dose measurement provided it is measured frequently enough to avoid over estimation as GFR falls, typically every two months.

Guideline 3.4
For three times weekly dialysis, dose should be quoted at ekt/v. For schedules other than three times weekly, dose should take frequency into account and be quoted as weekly standard kt/v (std kt/v), solute removed index (SRI) or equipment renal clearance (EKR).

3. Minimum Adequate Dialysis

Guideline 4.1
In anuric patients, treated by three times per week dialysis, the prescribed target ekt/v should be at least 1.2. Higher doses, up to 1.4 should be considered in females and those patients with high comorbidity.

Guideline 4.2
For patients with renal function or those with dialysis schedule other than 3 times per week, weekly dialysis dose should be at least equivalent to an SRI of 2.
ANNEXURE 20

CONCEPT OF INTEGRATED CARE

<table>
<thead>
<tr>
<th>MODALITY</th>
<th>STRENGTH</th>
<th>WEAKNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>PERITONEAL DIALYSIS</td>
<td>Increased patient freedom. Arm vessels not used for vascular access longer preservation of residual renal function</td>
<td>Risk of under dialysis when patients becomes anuric. Increase in membrane permeability and loss of UF with time on PD.</td>
</tr>
<tr>
<td>HEMODIALYSIS</td>
<td>Long term technique survival suitable for patients unable to perform own dialysis.</td>
<td>Limited by availability of vascular access. Availability dependent on local resources.</td>
</tr>
<tr>
<td>TRANSPLANTATION</td>
<td>Longer patient survival on dialysis</td>
<td>Limited by lack of kidneys, depends on patient's age and comorbidities.</td>
</tr>
</tbody>
</table>
DRUGS TO BE AVOIDED IN SEVERE RENAL FAILURE, HAEMODIALYSIS AND PERITONEAL DIALYSIS

<table>
<thead>
<tr>
<th>DRUGS TO BE AVOIDED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alendronic acid</td>
</tr>
<tr>
<td>Celecoxib</td>
</tr>
<tr>
<td>Combivir</td>
</tr>
<tr>
<td>Ertapenem</td>
</tr>
<tr>
<td>Foscarnet</td>
</tr>
<tr>
<td>Giblenclamide</td>
</tr>
<tr>
<td>Itraconazole IV</td>
</tr>
<tr>
<td>Mesalazine</td>
</tr>
<tr>
<td>Moxifloxacin</td>
</tr>
<tr>
<td>NSAIDs</td>
</tr>
<tr>
<td>Pethidine</td>
</tr>
<tr>
<td>Rosedronate Sodium</td>
</tr>
<tr>
<td>Rosuvastatin</td>
</tr>
<tr>
<td>Sucralfate</td>
</tr>
<tr>
<td>Tenofovir</td>
</tr>
<tr>
<td>Trizivir</td>
</tr>
<tr>
<td>Voriconazole</td>
</tr>
</tbody>
</table>
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<table>
<thead>
<tr>
<th>Country</th>
<th>Incidence (PMP Per Year)</th>
<th>Prevalence (PMP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>US</td>
<td>336</td>
<td>1403</td>
</tr>
<tr>
<td>Australia</td>
<td>97</td>
<td>663</td>
</tr>
<tr>
<td>Canada</td>
<td>152</td>
<td>841</td>
</tr>
<tr>
<td>Chile</td>
<td>123</td>
<td>672</td>
</tr>
<tr>
<td>Denmark</td>
<td>138</td>
<td>679</td>
</tr>
<tr>
<td>Finland</td>
<td>88</td>
<td>609</td>
</tr>
<tr>
<td>Germany</td>
<td>184</td>
<td>921</td>
</tr>
<tr>
<td>Italy</td>
<td>131</td>
<td>788</td>
</tr>
<tr>
<td>Japan</td>
<td>252</td>
<td>1642</td>
</tr>
<tr>
<td>New Zealand</td>
<td>119</td>
<td>655</td>
</tr>
<tr>
<td>Norway</td>
<td>95</td>
<td>612</td>
</tr>
<tr>
<td>Russia</td>
<td>15</td>
<td>73</td>
</tr>
<tr>
<td>Taiwan</td>
<td>331</td>
<td>1423</td>
</tr>
<tr>
<td>Turkey</td>
<td>144</td>
<td>380</td>
</tr>
<tr>
<td>UK</td>
<td>93</td>
<td>600</td>
</tr>
</tbody>
</table>

**CAUSES OF END STAGE RENAL FAILURE ACROSS THE WORLD**

<table>
<thead>
<tr>
<th>S.N O.</th>
<th>PRIMARY DISEASE</th>
<th>RENAL DISEASE</th>
<th>ANZTA</th>
<th>EDTA</th>
<th>UK</th>
<th>USRD S</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>GLOMWERULONEPHRITIS</td>
<td>27</td>
<td>25</td>
<td>15</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>DIABETES</td>
<td>25</td>
<td>15</td>
<td>11</td>
<td>35</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>HYPERTENSION</td>
<td>15</td>
<td>9</td>
<td>6</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>RENOVASCULAR DISEASE</td>
<td>-</td>
<td>3</td>
<td>9</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>INFECTIVE OR OBSTRUCTIVE NEPHROPATHIES</td>
<td>4</td>
<td>12</td>
<td>14</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>CONGENITAL DISEASE</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>FAMILIAL OR HEREDITARY DISEASE</td>
<td>6</td>
<td>9</td>
<td>3</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>MISCELLANEOUS</td>
<td>16</td>
<td>14</td>
<td>19</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>UNKNOWN</td>
<td>7</td>
<td>13</td>
<td>22</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

(Ref: Oxford Handbook of Dialysis)
## Annexures

### ANNEXURE 24

**PRACTICAL GUIDE TO DRUGS USED IN SEVERE RF, HD AND CAPD**

<table>
<thead>
<tr>
<th>S.NO.</th>
<th>DRUG</th>
<th>S.NO.</th>
<th>DRUG</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Allopurinol</td>
<td>17</td>
<td>Lisinopril</td>
</tr>
<tr>
<td>2</td>
<td>Atenolol</td>
<td>18</td>
<td>Losartan</td>
</tr>
<tr>
<td>3</td>
<td>Azathioprine</td>
<td>19</td>
<td>Met Cloprimide</td>
</tr>
<tr>
<td>4</td>
<td>Captopril</td>
<td>20</td>
<td>Metoprolol</td>
</tr>
<tr>
<td>5</td>
<td>Cimetidine</td>
<td>21</td>
<td>Morphine</td>
</tr>
<tr>
<td>6</td>
<td>Cyclosporin</td>
<td>22</td>
<td>Mycophenolate</td>
</tr>
<tr>
<td>7</td>
<td>Codeine phosphate</td>
<td>23</td>
<td>Nifedipine</td>
</tr>
<tr>
<td>8</td>
<td>Co-dydramol</td>
<td>24</td>
<td>Penicillamine</td>
</tr>
<tr>
<td>9</td>
<td>Colchicine</td>
<td>25</td>
<td>Perindopril</td>
</tr>
<tr>
<td>10</td>
<td>Diamorphine</td>
<td>26</td>
<td>Phenytoin</td>
</tr>
<tr>
<td>11</td>
<td>Diazepam</td>
<td>27</td>
<td>Propranolol</td>
</tr>
<tr>
<td>12</td>
<td>Diclofenac</td>
<td>28</td>
<td>Ramipril</td>
</tr>
<tr>
<td>13</td>
<td>Digoxin</td>
<td>29</td>
<td>Ranitidine</td>
</tr>
<tr>
<td>14</td>
<td>Enalapril</td>
<td>30</td>
<td>Simvastatin</td>
</tr>
<tr>
<td>15</td>
<td>Posinopril</td>
<td>31</td>
<td>Valsartan</td>
</tr>
<tr>
<td>16</td>
<td>Furosemide</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Exercise can be prescribed for either on or off dialysis. The following table summarizes the exercise prescription for End Stage Renal Failure.

<table>
<thead>
<tr>
<th>Rx Variables</th>
<th>On Dialysis</th>
<th>Off Dialysis</th>
</tr>
</thead>
</table>
| Mode         | Stationary Cycling | Most Cardiovascular Activities  
|              |              | Avoid contact to shunts and catheters |
| Frequency    | During Dialysis | 3/wk  
|              |              | depending on co-existing conditions |
| Duration     | 10-15 min to tolerance  
|              | 30 min in 3 months  
|              | 45 min in 6 months  |
| Intensity    | RPE only (13-14)  
|              | No target heart rates |
| Precautions  | • Medications  
|              | • Electrocardiogram  
|              | • Type of Dialysis  
|              | • Complications of ESRD  
|              | • Co-existing Diseases  
|              |   o Diabetes  
|              |   o Hypertension  
|              |   o Hyperlipidemia |
| Recommendations | • Diet for ESRD  
|                 | • Other: based on co-existing disease |

URL: http://www.indiana.edu/~k562  
Webmaster: Janet P. Wallace, PhD, FACSM  
Contact: wallacej@indiana.edu  
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Graphical Representation of Dialysis and Kidney Transplantation

Rates of Treated-Eskd By Sex and Age, 2009

Source: AIHW analysis of ANZDATA Registry data.

Proportion of Eskd Patients Receiving Dialysis or With a Functioning Kidney Transplant, 1991 To 2009

Source: AIHW analysis of ANZDATA Registry data.
Renal Failure: Its Treatment in Current Systems of Medicines

Number of Transplants from Deceased Donors and Living Donors, 1991 To 2009


Trends in the Number of Haemodialysis and Peritoneal Dialysis Patients, 1991 To 2009

Source: AIHW analysis of ANZDATA Registry data.
Annexures

Trends in the Number of People Receiving Peritoneal Dialysis for EsKD, by Peritoneal Dialysis Type, 1991 To 2009

Source: AIHW analysis of ANZDATA Registry data.

Proportion of New Cases Treated-EsKD By Cause of EsKD, 1991 To 2009

Source: AIHW analysis of ANZDATA Registry data.
ANNEXURE 27

ABBR EVIATIONS

ACE= Angiotensin Converting Enzyme
ACEI = Angiotensin Converting Enzyme Inhibitor
ADQI= Acute Dialysis Quality Initiative
AKI=Acute Kidney Infection
Alk.Phos = Alkaline Phosphatase
ARBs= Angiotensin II Receptor Antagonist
ARF= Acute Renal Failure
ATN = Acute Tubular Necrosis
BUN= Blood Urea Nitrogen
Ca= Calcium
CAPD = Continuous Ambulatory Peritoneal Dialysis
CCPD = Continuous Cycling Peritoneal Dialysis
CKD = Chronic Kidney Disease
Cl =Chloride

Cr = Creatinine
CRF= Chronic Renal Failure
CRP= C - reactive protein
CRRT= Continuous Renal Replacement Therapy
CT= Computed Axial Tomography
CVD = Cardiovascular disease
ESRD = End-Stage Renal Disease
GFR = Glomerular Filtration Rate
GI=Gastrointestinal
Hb = Hemoglobin
HBV = Hepatitis B Virus
Hct = Hematocrit
HCV = Hepatitis C Virus
HD= Hemodialysis
HDF= Hemodiafiltration
HLAs = Human Leukocyte Antigens
<table>
<thead>
<tr>
<th>Acronym</th>
<th>Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>HMGCoA</td>
<td>3 Hydroxy-3 Methyl-Glutaryl-CoA</td>
</tr>
<tr>
<td>K/DOQI</td>
<td>The Kidney Foundation Dialysis Outcomes Quality Initiative</td>
</tr>
<tr>
<td>K</td>
<td>Potassium</td>
</tr>
<tr>
<td>Mg</td>
<td>Magnesium</td>
</tr>
<tr>
<td>MRFIT</td>
<td>Multiple Risk Factor Intervention Trial</td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
</tr>
<tr>
<td>Na</td>
<td>Sodium</td>
</tr>
<tr>
<td>NKF-DOQI</td>
<td>National Kidney Foundation Kidney Disease Outcomes Quality Initiative</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>Non-Steroidal Anti-Inflammatory Drugs</td>
</tr>
<tr>
<td>P</td>
<td>Phosphorus</td>
</tr>
<tr>
<td>PD</td>
<td>Peritoneal Dialysis</td>
</tr>
<tr>
<td>PTH</td>
<td>Parathyroid hormone</td>
</tr>
<tr>
<td>RF</td>
<td>Renal Failure</td>
</tr>
<tr>
<td>RRT</td>
<td>Renal Replacement Therapy</td>
</tr>
<tr>
<td>SCR</td>
<td>Serum Creatinine</td>
</tr>
<tr>
<td>SLE</td>
<td>Systemic Lupus Erythematous</td>
</tr>
<tr>
<td>TMP</td>
<td>Trans membrane</td>
</tr>
<tr>
<td>UA/CR</td>
<td>Urine Albumin-to-Creatinine Ratio</td>
</tr>
<tr>
<td>UAER</td>
<td>Urinary Albumin Excretion Rate</td>
</tr>
<tr>
<td>UF</td>
<td>Ultra Filtration</td>
</tr>
<tr>
<td>UK</td>
<td>United Kingdom</td>
</tr>
<tr>
<td>URR</td>
<td>Urea Reduction Ratio</td>
</tr>
<tr>
<td>US</td>
<td>United States</td>
</tr>
<tr>
<td>USS</td>
<td>Ultrasound Scan</td>
</tr>
<tr>
<td>HAART</td>
<td>Highly active anti-retroviral therapy</td>
</tr>
<tr>
<td>MG</td>
<td>Membrane glomerulonephritis</td>
</tr>
<tr>
<td>MCGN</td>
<td>Mesangiocapillary glomerulonephritis</td>
</tr>
<tr>
<td>PAN</td>
<td>Polyarteritis nodosa</td>
</tr>
<tr>
<td>HIVAN</td>
<td>Human immune deficiency virus associated nephropathy</td>
</tr>
</tbody>
</table>
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SUBJECT INDEX

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