UNIT 16 NUTRITIONAL MANAGEMENT OF RENAL DISEASES

Structure

16.1 INTRODUCTION

In our previous units, we discussed about gastrointestinal tract disorders and their nutritional management. In this unit, **we** shall study about one of the most prevalent disorders, that is, renal disorders.

We shall first review the basic physiology of kidneys and its functions, which you have already studied in your Applied Physiology Course (MFN-001) in Unit 7. We suggest you look up this unit, i.e. Unit 7 once again and revise your knowledge of kidney physiology, as this will help you understand the discussion presented in this unit.

Next, in this unit, we will learn about various kidney function tests that involve both biochemical analysis and clinical examination. Later on, we shall get to know about renal diseases such as nephritis, nephrotic syndrome, acute renal failure (ARF), chronic renal failure (CRF), end stage renal disease (ESRD) etc. along with their dietary management. This would include the list of foods to be excluded during these diseases.

Objectives

After studying this unit, you will be able to:

- recapitulate and describe the physiology of the kidneys,
- discuss the renal function and diagnostic tests,
- identify different renal disorders, their etiology, clinical and metabolic manifestation, and
- rationalize the dietary modifications in renal disorders, especially proteins, minerals and fluids.

16.2 PHYSIOLOGY OF THE KIDNEY

You will remember from your course in Applied Physiology, Unit 7 that the kidneys are a major organ for *excretion* of metabolic *wastes* from the body. Thrmgh glomerular filtration, tubular secretion and selective reabsorption that takes place in the 'nephrons' (the functional units of the kidneys), the kidneys maintain the internal balance of body fluids. This includes normal osmotic pressure, composition and volume of blood, acid base balance and fluid electrolyte balance. Let us elaborate a bit on the kidney functions.

16.2.1 Kidney Functions

Figure 16.1 highlights the kidney functions. The primary functions of the kidney include:

- Excretion
- Endocrine
- Metabolic

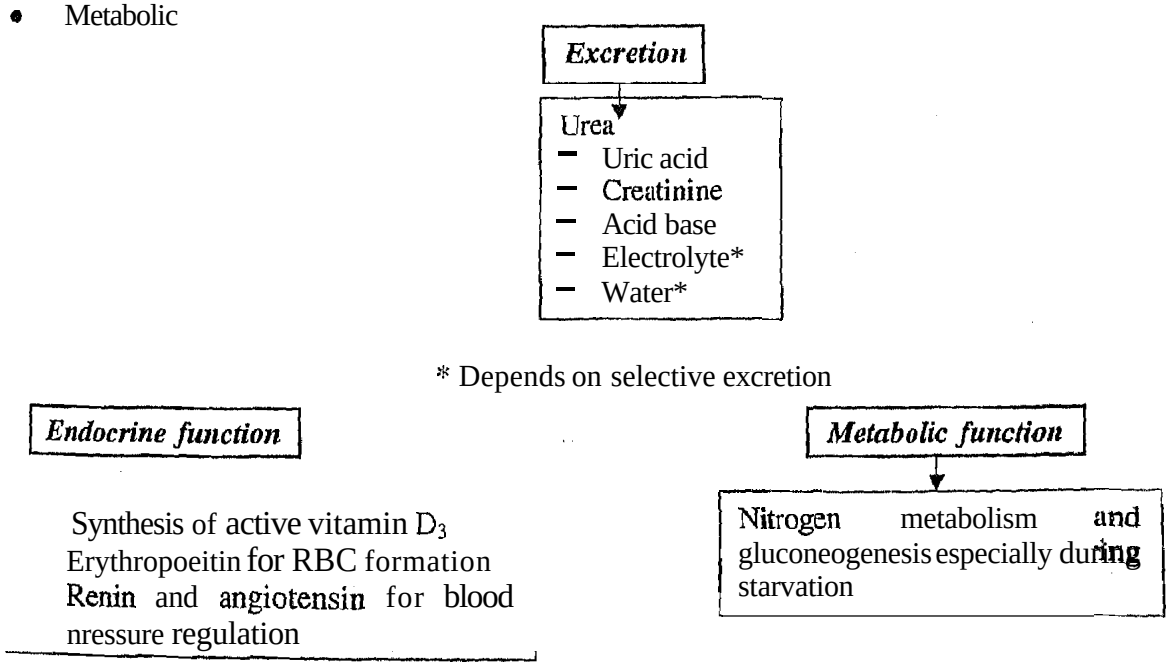

Figure 16.1 : Functions of the kidney

You may recall that we studied about the contribution of the kidneys in urine formation and its role in maintaining the pH and performing other regulatory functions in the body in the Applied Physiology Course in sub-section 7.3.3. Hence, we shall not go into the details of the excretory functions of kidneys here. We suggest you look up the

relevant portion in the Applied Physiology course now before you proceed further on this topic. The endocrine and the metabolic function are reviewed briefly next.

A. Endocrine Functions

The endocrine functions of kidneys include:

- *Synthesis of active vitamin* D_3: Kidneys maintain calcium-phosphorous homeostasis, which involves the complex interactions of PTH, calcitonin, vitamin D and three effector organs – gut, kidney and bone. Kidneys produce active vitamin D_3, as well as, eliminate both calcium and phosphorous. Vitamin D_3 you might be aware promotes efficient absorption of calcium (Ca) and is one of the substances necessary for bone remodelling and maintenance.
- *Eythropoietin for RBC formation:* Eythropoietin, a critical determinant in RBC formation is produced by the kidneys.
- *Renin and angiotensin for regulation of blood pressure:* A lowered blood volume causes the cells of the glomerulus to react by secreting rennin – a proteolytic enzyme. Renin acts in the plasma to form angiotensin I, which is converted to angiotensin II (a powerful vasoconstrictor) and a potent stimulus of aldosterone secretion by the adrenal gland. As a'consequence, sodium is reabsorbed and blood pressure returns to normal. The renin-angiotensin mechanism is highlighted in Figure 16.2.

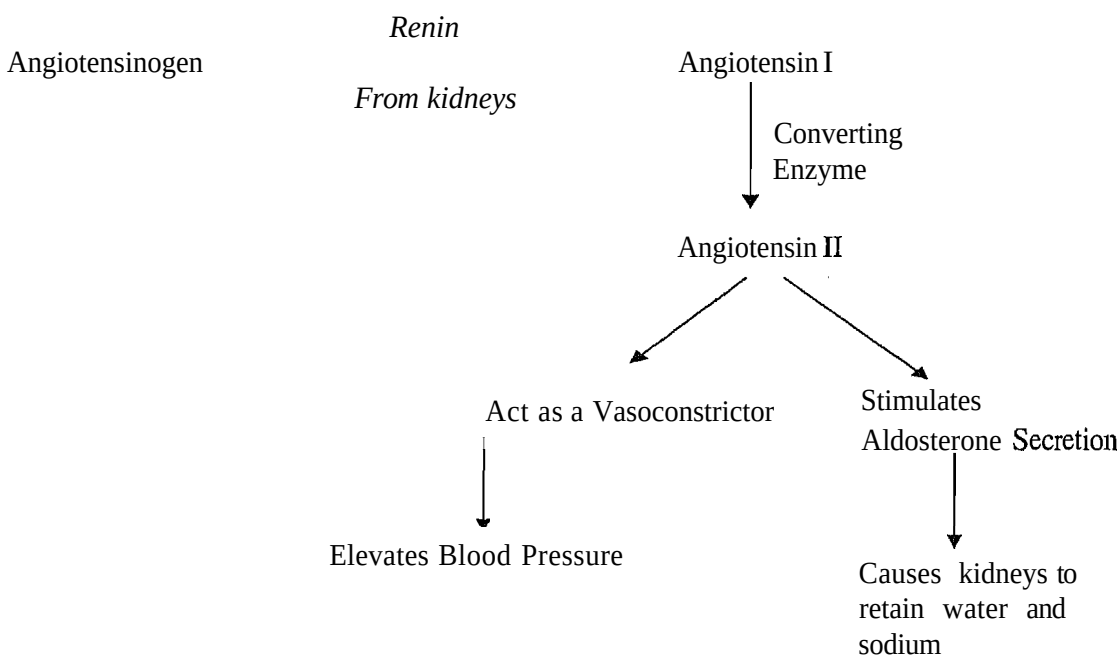

Figure 16.2: Renin-angiotensin aldosterone mechanism

B. Metabolic Function

The main metabolic role of kidney is linked to nitrogen metabolism. However, research indicates that kidneys may also play an important role in glucose counter regulation. The main purpose of gluconeogenesis, as you may already be aware, is to synthesize glucose from non-carbohydrate precursors. This process occurs mainly in the liver and kidney. The role of kidney in eliminating the end products of protein metabolism is highlighted next.

Nitrogen Metabolism: The majority of solute load consists of nitrogenous wastes, which are the end products of protein metabolism. These include urea, uric acid, creatinine and ammonia. In case of inappropriate elimination of these waste products, they get collected in abnormal quantities in blood. The inability of the kidneys to excrete these nitrogen wastes leads to renal failure.

Having briefly reviewed the functions of the kidney, can you now suggest what the important outcomes of functions are? These are enumerated next for your reference.

Important Outcontes of Functions

It will be useful to recall, from Unit 7 in the Applied Physiology Course some of the important outcomes of kidney functions. These include:

- The kidneys have a large blood flow.
- About 180 L ultra filtrate is formed at the glomeruli / day but daily urine output is only 1.2-1.5 L/day.

URINE HAS A SPECIFIC GRAVITY RANGING BETWEEN 1.003-1.030. THE MEAN PH IS BETWEEN THE RANGE IS 4.7-8.0. IT CONTAINS 90% WATER, 0.5% SOLID WASTE OF WHICH 60% IS NITROGENOUS WASTE AND 40% INORGANIC SALTS.

- For greater solute load, a greater volume of fluid and urine output is required.
- Healthy kidneys have a capacity to produce urine with wide variability of volume, osmolality and pH.
- Healthy kidneys can regulate one substance at the expense of another, based on the need.
- In this context, control of blood volume has priority over other parameters.
- Healthy kidneys have considerable reserve capacity and health can be maintained even on half a functional kidney.
- With progressive impairment (when more than 60% nephrons are damaged), the urine output falls and products of metabolism start accumulating in the body. Gradually metabolic and endocrine functions also get impaired and renal failure can develop.

With this knowledge, we will be able to understand the diagnostic tests for kidney function and various kidney disorders, in relation to treatment. Let us then move on to learn about the methods/techniques used to assess kidney functions,

16.3 ASSESSMENT OF KIDNEY FUNCTION: DIAGNOSTIC TESTS

The kidney functions can be assessed through various biochemical tests and clinical examination. These are described in this section.

Biochemical Tests

The kidney function can be assessed through various biochemical tests. These mostly measure glomerular and / or tubular function of the kidneys. As the functional capacity of the nephrons reduces in kidney disorders, changes occur in the blood and urine chemistry. The normal values for various parameters of blood and urine are given in the Table 16.1 and 16.2. You would recall reading about the renal function tests in your Applied Physiology Course, Unit 7 undersection 7.8, We suggest you go back and refer these for a better understanding of the diagnostic tests. Here, however we present you with a brief review of these tests. So let us get started.

Common diagnostic tests to assess renal function include measurement of:

1. *Plasma /serum concentration of urea, creatinine and uric acid:* Levels of these tend to increase in blood due to inadequate excretion in impaired kidney function.

2. *Serum electrolytes* like sodium, potassium, chloride, inorganic phosphorus, and bicarbonate: Their levels may increase or decrease depending on the kidney disorder.

3. *Glomerular Filtration Rate (GFR)* is measured by the creatinine clearance: Clearance tests are *a measure of the efficiency with which kidneys remove a substance from the blood.* Normal creatinine clearance in adults is about 1255 ml/ minute, which can be reduced to 30 ml/minute or less when kidneys fail.

4. *Alkaline phosphatase* in blood: This can be altered as vitamin D synthesis is affected in impaired kidney function.

5. *Blood Haemoglobin:* Its levels are dependent on erythropoietin synthesis.

6. *Urine Volume and Extra Cellular Fluid (ECF)* : Increase or decrease in urine volume and volume of body, based on excretory capacity.

7. *Urine examination*: Urine analysis for clarity, colour, microscopic examination, pH, specific gravity, osmolality and presence of abnormal constituents like blood, protein, casts etc.

8. *Urine, electrolytes:* Urine electrolytes like sodium, potassium, magnesium especially in tubular disorders, where excess loss may occur.

Table 16.1 gives the normal blood parameters for diagnosis in kidney diseases.

Table 16.1: Normal blood parameters for diagnosis in kidney diseases.

Blood Parameters	Normal Range
BUN (Blood Urea Nitrogen)	7-22 **mg** %
Creatinine	0.8-1.8 mg%
Uric acid	2.4-7.0 mg %
Sodium	132-148 mEq/L
Potassium	3.5-5.5 mEq/L
Phosphorus	3.0-4.5 mg %
Calcium	8.1-10.4 mg %
Chloride	98-108 mEq/L
Total protein	6.6-8.7 gm %
Albumin	3.8-4,4 gm %
Globulin	1.8-3.6 gm %
A/G ratio	2:1
Alkaline phosphatase	30-150 unit per litre
Total cholesterol	150-250 mg %
Triglyceride	0-200 mg %

Next let us review the normal urine parametes which are listed in Table 16.2.

Tabb 16.2: Normal urine parameters

Volume	800-2000 ml/day
Glucose	0-0.8 mmol/L(0-15 mg/dL)
Protein	Less than 150 mg/day
Ketones	negative is normal
Presence of acetone	Small: < 20 mg/dl Moderate 30-40 mg/dl Large: > 80 mg/dl
Urine Electrolytes:	
Chloride	110-250 mEq
Sodium	15-250 mEq /L
Potassium	25-120 mEq/L
Magnesium	Less than 150 mg/day
Calcium	100-300 mg
Creatinine	1.1-3.4 g
Creatine	Little or none
Specific gravity	1.015-1.020
pH	6.0

Abnormalities in the parameters listed above are an indication of kidney disorders. What are these disorders? Next, let us move on to the understanding of these common renal disorders.

16.4 COMMON RENAL DISEASES

Kidney disorders may be *infective, inflammatory* in origin, or *degenerative* in nature. With an increasing severity or long duration, these can result in renal failure. Diseases of the kidney may involve the nephrons, tubules or glomerulus. Inflammation of the nephrons is termed *nephritis*. *Glomerulonephritis* (GN) refers to involvement of specifically the *glomeruli*. With glomerular damage, usually tubules also get affected. Stones formation can also take place in the kidneys, this is known as *renal calculi* or *nephrolithiasis*. Degenerative or vascular disorders can lead to *nephrosclerosis*, where blood vessels of the kidneys become narrow. This leads to reduced blood and oxygen supply, and hence kidney damage.

Kidney diseases may be *acute* or *chronic* and have several underlying causes. The treatment is dependent on the disease origin, extent and type of damage and clinical and metabolic outcomes. As kidneys have a direct effect on nutritional status via homeostasis of fluid, electrolytes and nutrients in the body, diet plays an important role in treatment. Some of the common renal disorders are:,

- Acute and chronic nephritis
- Nephrotic syndrome
- Acute renal failure
- Dialysis
- Renal transplantation
- Renal calculi

The etiological factors for each of these disorders are discussed later in this unit under each renal disorder covered separately. You will realize that the management of these disorders also varies, however, there are a few common aspects linked with the dietary management in renal diseases. These general principles are described here next, before we take up each disease separately.

16.5 GENERAL PRINCIPLES OF DIETARY MANAGEMENT IN RENAL DISEASES

Several common principles apply to the dietary management of various renal diseases. This is because various symptoms, clinical and biochemical manifestations may be common in different renal disorders as you will find out soon. The main objectives of dietary management in renal diseases are to:

- reduce the excretory work of the kidneys, while maintaining as near normal fluid, acid base and electrolyte balance.
- maintain satisfactory nutritional status.
- prevent progression of renal damage and development of uremia (accumulation of nitrogenous waste products in blood).

To meet these objectives, modifications in the diet are required mainly for

a) Proteins

b) Electrolyles, especially sodium and potassium

c) **Fluids**

Let us now understand the general dietary principles.

Energy: It should be normal or increased in case of weight loss. A simple diet low in proteins, moderate to high in carbohydrates and unsaturated fats is preferred.

Protein: The amount of protein to be given is dependent on GFR. Proteins must be enough to prevent muscle wasting and malnutrition, but it needs adjustment to prevent accumulation of nitrogenous wastes in the blood and uremia. Good quality proteins from milk and eggs are recommended to meet the body's need of essential amino acids and yet not burden the body with excess non essential amino acids.

Electrolytes:

1. Sodium intake mostly needs restriction depending on whether it is retained in the body or excessive loss in urine occurs. Retained sodium and water can result in oedema, hypertension and congestive cardiac failure (CCF).
2. Potassium (K), if retained, needs restriction to prevent hyperkalemia (excess K in blood), which can result in cardiac arrest. Both electrolytes, if lost in excess amounts in urine may need replacement.

*Vitamins and **Minerals***:

1. Calcium may require supplements in chronic or severe cases along with vitamin D.
2. Phosphate mostly needs restriction as it tends to be retained in the body.
3. Iron supplements are needed with impaired erythropoietin synthesis,
4. Vitamin B and C supplements are required due to increased needs in renal patients.

Fluid: This mostly has to be restricted due to a fall in the GFR in most patients. If however, polyuria occurs, fluid needs may increase.

If you have understood these basic principles, you will be able to modify the diet in various renal disorders on a more rationale basis as you will find out while learning about the various renal disorders described next.

16.6 ACUTE AND CHRONIC NEPHRITIS

We shall learn how to plan diets for most of the renal diseases being discussed in this chapter in the Practical Manual 005.

As mentioned earlier, nephritis refers to *the inflammatory disease of the nephrons due to infection, degenerative processes or vascular disease.* In most cases, the inflammatory process affects the capillaries of the *glomeruli,* this disorder is termed as *glonzerulonephritis (GN).* There may be damage to the tubules also. The most common and well understood of the different types of glomerulonephritis, is *post streptococcal proliferative glomerulonephritis,* about which we will discuss here in this section. GN may be self-limiting or progress to serious renal damage. Let us learn about the etiology, clinical and metabolic manifestations and dietary management of glomerulonephritis next.

16.6.1 Etiology

Acute form of glomerulonephritis is commonly seen in 3-10 year oldchildren, although in 5% or more cases the initial attack occurs in adults past the age of 50 years. Previous streptococcal infection, 7-20 days prior to onset, is a common cause of this disease and antigen-antibody reaction is mostly the basis of damage of nephrons. In acute glomerulonephritis, there is a usually sudden onset and the condition is usually completely cleared in a year or two.

In chronic cases, progressive renal damage occurs involving an increased amount of renal tissue, eventually requiring dialysis and other support treatments.

16.6.2 Clinical and Metabolic Manifestations

Classic symptoms include gross *haematuria* (presence of blood in urine) and proteinuria (protein in the urine). Due to sodium and water retention and circulatory congestion, varying degree of *oedema* with shortness of breath may be observed. The patient generally is anorexic, which results in feeding problems. If the disease leads lo renal insufficiency, *oliguria* (reduced urine output) or *anuria* (no urine output) occurs, which indicates chances of development of acute renal failure. The summary of the clinical symptoms is presented in Box 16.1.

Box 16.1 Clinical Symptoms of Glomerulonephritis

The clinical manifestations include:

- Fever i.e. elevation of body temperature above 98.4°F or 37°C.
- Uremia: accumulation of nitrogenous waste products and other urinary constituents in blood, particularly haematuria and proteinmea
- Oedema: fluid and electrolyte retention in tissues
- Hypertension: Systolic and Diastolic blood pressure above 120/80 mm Hg
- Oliguria and anuria because of reduced GFR.

Having learnt about the disease and its symptoms, let us next get to know about the dietary management of this problem.

16.6.3 Dietary Management

Keeping in mind the general objectives of dietary management and principles of dietary modificationsalready discussed earlier in section 16.4, the following dietary guidelines is recommended.

Calories: A high-energy diet is recommended to spare the proteins for tissue repair rather than being used for energy. Adults may need 30-40 ***Kcal*** / kg dry ***weight*** and children about 100 Kcal /*kg* dry *weight* or more, based on age. Increased energy requirements are also recommended since elevation of body temperature i.e. fevers may be present in patients with glomerulonephritis. Elevation of body temperature results in increase in basal metabolic rate (BMR) and hence the energy intake may be increased to about 10% (from the level suggested by RDI).

Proteins: Blood urea nitrogen (BUN) and oliguria determine the restriction of protein in the diet. Initially, 0.5 to 0.6-g protein/kg Ideal Body Weight (IBW) is provided using principally high quality protein. Normal levels of protein (1 g/kg IBW) may be provided if BUN levels remain within the normal range.

Emphasis should be to include good quality protein or proteins of high biological value (milk and milk products, egg white, meat etc.) in the diet of the patients.

Carbohydrates: Liberal carbohydrate intake is important for protein sparing action, for reducing catabolism of protein, as well as, for preventing starvation ketosis. Both simple carbohydrates such as sugar, as well as, complex form such as starches can be included in the diet.

Fats: Based on tolerance levels, fat is included to provide non-protein calories for energy needs. Being energy dense, fat reduces the bulk of the diet and makes the diet more palatable.

Sodium: The restriction of sodium is dependent on the degree of oliguria and hence sodium retention. If renal function is impaired, sodium may be restricted to 500 to 1000 mg/day. With recovery, the intake may be increased. A list of sodium-rich foods is included in Table 16.2 for your reference. You may need to avoid these foods in the diet of the patient suffering from nephritis.

Table 16.2: Foodstuffs high in sodium

Baking soda
Salt
Ajinomoto
Salted wafers, popcorns, salted biscuits, salted namkeens
Papads – all varieties.
Salted pickles, chutneys, curry powder – commercial
Commercial salad dressings and sauces
Soup cubes
Soft drinks containing sodium benzoate
Bakery products, bread, biscuits, patties, cakes etc
Nuts such as salted cashewnuts, pistachio, walnuts, peanuts
Commercial processed cheese
Foods containing preservatives such as noodle mixes, pastas
Canned and tinned foods
Salted or canned meat
Sea food, chicken, dry fish, bacon, ham.
Meat and yeast extracts like marmite
Proprietary drinks – Bournvita, chocolate drinks, Horlicks
Milk and curds
Pulses and legumes – all varieties
Vegetables such as – cauliflower , snakegourd, beetroot , carrot , coriander leaves, fenugreek(methi) leaves, lettuce, spinach (palak), amaranth, radish

Potassium: In case of oliguria, renal clearance of potassium is impaired resulting in hyperkalemia (increased level of potassium in blood). In the initial stages, therefore potassium may have to be restricted to 1200-1500 mg/day. In addition, fruit, fruit juices, nuts and coconut water may be restricted or avoided, as they are rich in potassium.

Fluid: Intake of fluids needs to be restricted in case of reduced GRF and oliguria to 500-700 ml/day plus the volume of urine output in previous 24 hours. Without oliguria fluid intake may be normal.

Dietary guidelines for acute chronic nephritis are summarized in Table 16.3.

Table 16.3: Dietary guidelines **in nephritis**

Nutrients	Comments
Protein	BUN and oliguria determine the protein restriction. Give 0.5 to 0.6 g protein/kg of ideal body weight and then increase gradually depending on BUN values.
Kilocalories	Adults given 30-40 Kcal/kg dry weight and children about 100 Kcal/kg dry weight based on age. Sufficient simple and complex carbohydrates have a protein spairing action. Fat should be given based on tolerance.
Sodium	Sodium depends on sodium retention and is restricted to 500-100 mg/day.
Potassium	Potassium restriction to 1200-1500 mg/day especially if excess in blood (hyperkalemia).
Fluid	If reduced GRF and oliguria, restrict to an amount equal to urine volume plus 500 ml.

With this, we end our study on nephritis. Next, we shall take up thenephrotic syndrome and understand the dietary management of this disorder.

16.7 NEPHROTIC SYNDROME

Nephrotic syndrome is a disorder where the kidneys have been damaged, causing them to leak protein from the blood into the urine. It is a fairly benign disease when it occurs in childhood, but may lead on to chronic renal failure, especially in adults.

Nephrotic syndrome is also termed as nephrosis. This disorder is characterized by massive oedema and proteinuria resulting from degenerative lesions of the tubules, mesangium (central part of the renal glomerulus) or basement membrane of the glomeruli.

What is the cause for these degenerative lesions to occur? Let us find out.

16.7.1 Etiology

There are many etiological factors that cause a nephrotic syndrome. Progressive glomerulonephritis, disease such as diabetes, collagen disease or drug reactions, from exposure to heavy metals, or even from a reaction to toxin venom following a bee sting can cause this syndrome.

The clinical manifestations of this disorder are highlighted next,

16.7.2 Clinical and Metabolic Manifestations

Nephrotic syndrome is characterized by massive oedema and proteinuria, hypoalbuminemia, hypercholesterolemia and abnormal bone metabolism. Let us get to see how this happens.,

Denegerative lesions of the capillary basement membrane of the glomeruli lead to loss of the glomerular barrier to protein. Large amount of proteins (upto 4-10 g/day) are thus lost in the urine as albumin. This proteinuria results in plasma proteins being substantially lowered and causing massive hypoproteinemic oedema to occur due to reduction in plasma albumin, which is mainly responsible for maintaining balance between tissue and circulating fluids. Pedal (foot) and periorbital (around the orbit of the eye) oedema and ascites (fluid in abdominal cavity) are common. Other proteins lost include globulins, thyroid and iron binding protein. The continued loss of proteins results in tissue breakdown and malnutrition, often masked by the oedema. Development of fatty liver and sodium retention worsens the oedema. Another feature of this disorder, linked to hypoproteinemia is elevation of serum lipids, especially cholesterol to above 300 mg/dl. Nephrotic syndrome may be characterized by spontaneous period of remission and exacerbation.

The progression of nephrotic syndrome is illustrated in Figure 16.3.

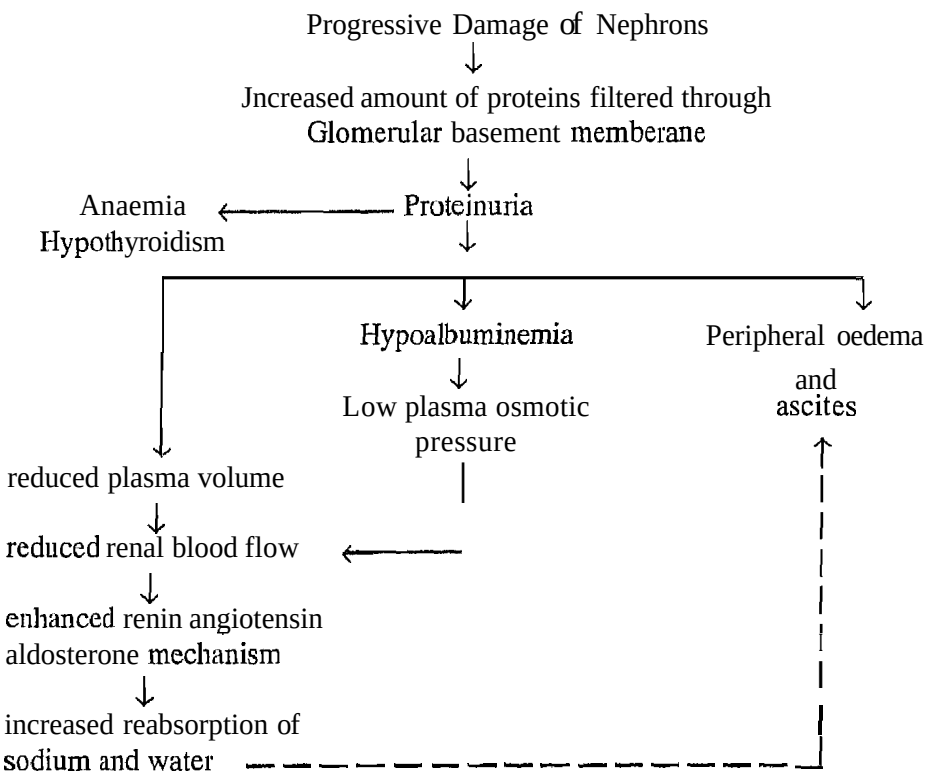

Figure 16.3 : Flow diagram for nephrotic syndrome

As the disease progresses, calcium or phosphate levels in blood maybe altered due to altered vitamin D levels, resulting in renal osteodystropy and osteomalacia.

From our discussion above it is evident that nephrotic syndrome occurs due to loss of proteins in the urine in large quantities which reduces the amount of protein in blood, So, then what is the dietary management for this disorder? The next seclion presents a detail review on this aspect.

16.7.3 Dietary Management

The major objectives of dietary management arc 1) to control and correct protein deficiency, and *2)* correct and prevent oedema and 3) maintain adequate nutrition to afford better resistance to infection.

To help meet this objective the dietary guidelines include:

Calories: High daily intake of 35 to 60 Kcal/kg of ideal body weight for adult and about 100 Kcal / kg or more for children is prescribed so as to conserve proteins. This ensures adequate amount of energy and optimal utilization of protein for tissue synthesis,

Protein: The major cause of nephrotic ascites and oedema is reduction of 20% or less than normal value in plasma albumin level. Therefore,' replacement of prolonged protein

loss is most immediate and fundamental. A daily protein intake upto 1.25 g/kg/day in adults is advocated. As for children, since Indian children usually have a low intake of protein (even less than the RDA), adequate protein intake of upto 2 g/kg/day and not more than 3 g/kg/day in infants is advocated to replenish the depleted stores and to enhance synthesis of albumin and thereby reduce the oedema. However, please note, a very high protein diet may cause tubular damage to the kidneys as the kidneys will have to filler more of the proteins.

Foodstuffs rich in protein are cow's milk, skimmed milk, eggs, fish, dry fish, chicken, lean meat, paneer made from cow's milk, cheese, sprouts, pulses and legumes. At least 60-70 % of this protein should be of good / high biological value (milk and milk products, egg whites and meats).

Carbohydrates: A high carbohydrate intake is recommended for the protein sparing action.

Fat: High amount of fats should be avoided as the cholesterol and triglyceride levels tend to be high in patients with nephrotic syndrome. The diet must be high in calories so as to conserve proteins, yet low in fats. Excess of oily food and saturated fats (ghee, margarine, etc) should be avoided. If patient has hyperlipoproteinemia and hypercholesterolemia, the total fat, as well as, cholesterol intake needs to be restricted to less than 30% energy from fat and < 300 mg cholesterol per day.

Sodium: Reduction in sodium intake is required to reduce the oedema. Approximately 2-3 g of sodium/day may be recommended. Usually added salt is prohibited in these patients. Refer to Table 16.2 given earlier which presents the foodstuffs high in sodium. Avoid these foods in the diet of the nephrotic syndrome patient. Diuretics are usually used to prevent further ocdema.

Potassium: If oliguria and anuria is not present, potassium restriction is not necessary. In fact adequate potassium is important as losses may occur due to tissue protein breakdown and diuretic use.

Calcium: If deficiency of calcium results leading to bone rarefaction, increased calcium intake or calcium supplementation is recommended along with moderate increase in protein.

Fluid: May be normal unless GFR is reduced.

The dietary guidelines described above are summarized in Table 16.4 for easy reference.

Table **16.4:** Dietary guidelines **for** nephrotic syndrome

Nutrients	Comments
Calories	For adults' – 35-60 Kcal/kg/IBW and children 100 Kcal/kg. Sufficient calories provide for optimum utilization of proteins for tissue synthesis.
Protein	For adults 1.25g/kg IBW/day and children 2-3 g/kg IBW. Of this, 60-70% proteins must be of high BV to prevent ascites and oedema.
Carbohydrates	High intake due to its protein sparing action.
Fats	Limit to 30% energy from fat and < 300 mg cholesterol, especially if the patient has hyperlipoproteinemia and hypercholesterolemia.
Sodium	2-3 g/day and diuretics in order to reduce oedema.
Potassium	Required in adequate amounts as its losses may occur due to tissue protein breakdown and diuretic use.
Calcium	Additional amounts needed in case of bone rarefaction.
Fluid	Normal – unless GFR is reduced.

Before we move any further, let us recapitulate what we have learnt so **far.**

Check Your Progress Exercise 1

1. Briefly review the important physiological functions of kidneys.

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2. Mention any five significant kidney function tests.

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3. Enlist the diseases caused due to kidney dysfunction.

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4. What are the causes of nephrotic syndrome? What dietary measures help to correct oedema in nephrotic syndrome?

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5. What are the two major symptoms of glomerulonephritis'? What are the recommended protein and fluid intake of glomerulonephritis?

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Therefore, your understanding so far has been quite good. Let us now continue with our study of kidney disorders by reviewing acute renal failure, next,

16.8 ACUTE RENAL FAILURE

Acute renal failure (ARF) is a rapid loss of renal function due to damage to the kidneys, resulting in retention of nitrogenous (urea and creatinine) and non-nitrogenous waste products that are normally excreted by the kidney. Depending on the severity and duration of the renal dysfunction, this accumulation is accompanied by metabolic disturbances, such as metabolic acidosis (acidification of the blood) and hyperkalaemia (elevated potassium levels), changes in body fluid balance, and effects on many other organ systems. It can be characterized by oliguria or anuria (decrease or cessation of urine production), although *nonoliguric ARF* may occur. It is a serious disease and treated as a medical emergency.

Acute renal failure, in fact is a sudden loss of the ability of the kidneys to excrete waste, concentrate urine and conserve electrolytes. It is a serious condition characterized by a sudden shutdown of kidney function as mentioned above due to decreased renal flow, acute glomerular or a tubular damage. It results in a decline in glomerular filteration rate (GFR), usually associated with azotemia (accumulation of nitrogenous waste products in the blood) and a fall in urine output.

The causes for this serious condition are discussed next.

16.8.1 Etiology

Several conditions can lead to ARE These include:

- Circulatory shock, large blood loss and reduced renal blood flow as in traumatic injury, shock, severe burns, surgery, septicemia, dehydration and fluid loss,
- Mismatched blood transfusions,
- Nephrotoxins like carbon tetrachloride, certain poisonous mushrooms,

- Infections, snake bite, bee stings etc.
- Immunological reactions to drugs like certain antibiotics, and
- Renal disease like acute glomerulonephritis.

The most common cause is altered renal haemodynamics or ingestion of nephrotoxins.

What then are the clinical manifestations of this disorder? Let us find out.

16.8.2 Clinical and Metabolic Manifestations

The onset of ARF is sudden, with the course of the disorder having two phases, namely:

a) oliguria or initial acute phase, and

b) . diuretic phase.

The latter indicates restoration of renal function, although it may still remain poor for several days.

The major clinical features of ARF are oliguria or anuria (urine output 20-200 ml), due to drastic reduction of GFR to 1-2% of normal. Along with this, haematuria and proteinuria are usually present. There is an elevation of serum urea nitrogen and creatinine due to reduced GFR and tissue protein breakdown. Uremia may develop along with associated symptoms like disorientation, lethargy, nausea, vomiting and anorexia. Blood pressure elevation and increased levels of potassium, phosphate and sulphate occurs with lowered levels of sodium and bicarbonate. Water balance is a crucial factor and unless controlled, the condition can prove fatal mostly due to potassium intoxication or excess fluid retention leading to cardiac failure. Return to renal function is characterized by an increase in urine output or diuresis.

When diuresis is established, the urine volume gradually increases to between 3 to 5 litres/day and the excretion of sodium, potassium, urea and other solutes also increase. The blood urea falls to normal in 7 to 10 days, indicating that glomerular filtration has effectively improved. Although the excretory function of kidney is restored, the recovery of regulatory function of the tubules is slower. The internal environment of the patient is still at risk because of excessive losses of water, sodium, potassium, bicarbonate and magnesium.

In majority of patients, the kidneys will recover with little or no residual damage if the patient can survive the oliguric phase. However, in a few cases, residual damage of tubular function may sometimes be detected even long after the blood urea has returned to normal levels.

Having gone through the discussion above, you would realize acute renal failure is a medical emergency. Management of the life-threatening complications becomes very important. Diet too plays an important role. We shall learn about the dietary management next.

16.8.3 Dietary Management

In ARF the common nutritional problems include: (1) Poor appetite, (2) Inability to take food/or fluids orally due to intubation , and (3) Hypercatabolism (increased metabolism) due to underlying illnesses such as infection, postoperative healing.

Diet therapy in acute renal failure (ARF), therefore, focuses on:

- reestablishment of fluid electrolyte balance,
- maintenance of acceptable levels of blood urea and creatinine while supporting tissue healing and making up catabolic losses, and
- preventinginfection.

With conservative medical and diet treatment, recovery may occur within a few days or weeks. However, if oliguria continues with a rise in nitrogenous wastes and

potassium, aggressive therapy including haemodialysis may be required with nutritional support. Thus dietary management is a challenge and plays an important role. Oral feeding is best, but if nutritional support is needed, caution is necessary to avoid fluid overload and uremia.

The dietary guidelines for acute renal failure include:

Calories: In most adults, energy requirement amounts to 30-40 Kcal/kg body weight with upto 40-45 Kcal /kg for hypercatabolic cases. The major source of energy is carbohydrates followed by fat. An intake of 100-200 g or more of sugar /glucose per 24 hours is administered when the oral intake is poor because of vomiting and diarrhoea. Children need enough calories to support growth. In some cases enteral or parental non-protein concentrated calories sources may have to be provided because of fluid restriction.

Protein: This needs restriction and intake is dependent on GFR and extent of hypercatabolism. Initially the intake may range from 0.5 to 0.6 g/kg IBW, subsequently, increased to 0.8-1.2 g/kg IBW/day. Therefore, depending on the degree of protein catabolism 0.5-1g/kg/day of protein may be given. With improvement, at least 60-70% of good quality proteins are recommended to reduce unnecessary nitrogen load.

In case total parenteral nutrition (TPN) is required, a balanced amino acid solution containing both essential and nonessential amino acids should be administered. In addition to essential amino acids, arginine, histidine, serine, taurine and tyrosine may be recommended.

Sodium: During the oliguric phase, sodium may need to be restricted to 500-1000 mg (20-40 mEq) daily. It can be liberalized with onset of diuresis.

Potassium: Since, hyperkalaemia is a life-threatening complication of acute renal failure, it needs to be treated urgently. Potassium intake is restricted to 1000-2000 mg (25 to 50 mEq) and should be monitored strictly and regularly. As the renal function improves, the intake may be increased.

Fluid: Intake is based on fluid balance but is usually reslricted to a basic allowance of 500 mllday for an average adult with addition made for losses via other routes. The fluid allowance is usually regulated in accordance with urinary output and any additional losses from vomiling or diarrhoea. Strict monitoring of fluid balance is important, the patient's weight and blood sodium levels are good indicators of fluid balance, and the amount of fluid required. If fluid intake is not adequate for excretion of metabolic wastes, dialysis is usually recommended.

The dietary guidelines enumerated above are summarized in Table 16.5 for your easy reference.

Table 16.5: Dietary guidelines in acute renal failure

Nutrients	Comments
Protein	If not on dialysis, 0.6 g/kg/day. If on dialysis, 1-1.2 g/kg from dietary or parenteral sources.
Calories	Provide sufficient amount for weight maintenance or 30-40 Kcal. Increase to 40-45 Kcal/kg in hypercatabolic subjects. Encourage non-protein calories from fats and simple carbohydrates.
Sodium	In oliguric phase, restrict to 500-1000 mg (20-40 mEq). On diuresis, the amount nay be increased.
Potassium	If hyperkalemia is present restrict to 1000-2000 mg (25-50 mEq). On improvement, increase to 60-70 mEq.
Fluids	Limit fluids lo an amount equal to urine volume plus 500 ml.

After a detail review on acute renal failure, we move on to chronic renal failure.

16.9 CHRONIC RENAL FAILURE (CRF)

Chronic renal failure is a slow progressive loss of renal function over a period of months or years and defined as an abnormally low glomerular filtration rate, which is usually determined indirectly by the creatinine level in blood serum.

CRF, therefore, is a condition that arises due to advanced and progressive damage of kidneys with impairment of renal function. Few functional nephrons remain and CRF results in what is usually termed *uremia.* Uremia, you may already know by now is a toxic condition resulting from renal failure, when kidney function is cotnpromised and urea, a waste product normally excreted in the urine, is retained in the blood.

Unlike acute renal failure, with its sudden reversible failure of kidney function, chronic renal failure is a gradual and progressive loss of the ability of the kidneys to excrete wastes, concentrate urine, and conserve electrolytes. CRF can range from mild dysfunction to severe kidney failure. CRF that leads to severe illness and requires some form of renal replacement therapy (such as dialysis) is called end-stage renal disease (ESRD) about which we shall learn in the next section.

What then are the causes for this disorder? Let us find out.

16.9.1 Etiology

A number of diseases that involves the nephrons may result in CRF like primary glomerular diseases such as:

- immune complex glomerulonephritis,
- metabolic disease with renal involvement such as diabetes mellitus, especially IDDM,
- hypertension,
- exposure to toxic substances,
- kidney stones and infections,
- renal vascular diseases,
- renal tubular diseases,
- chronic pyelonephritis, and
- congenital abnormalities of both kidneys.

These conditions can cause extensive changes in kidney structure and function. CRF results in accumulation of fluids and waste products which can lead to many clinical signs and complications which are enumerated next.

16.9.2 Clinical and Metabolic Manifestations

Progressive loss of nephrons with a decreased renal blood flow and glomerular filtration results in a marked impairment of not only excretory but also metabolic and endocrine functions of the kidney. There is a decreased ability of the kidneys lo maintain body water balance, concentrate solutes in body fluid (osmolality) and electrolyte and acid-base balance, Other clinical manifestations that develop may relate to almost every system of the body due to an overall pervasive metabolic derangement of the body.

Increased solute load of metabolic wastes results in osmotic diuresis initially, due to an impaired ability of the kidney to concentrate urine. This leads to loss of sodium and potassium. However, with continued renal damage and reduced GFR, sodium, potassium and nitrogenous wastes tend to be retained in the body. This contributes to oedema, hypertension, hyperkalemia and azotemia, respectively, Azotemia is the buildup of nitrogen waste products in the blood. Retention of phosphate, sulphate and organic

acids causes metabolic acidosis due to loss of bicarbonate. Impaired calcium and phosphorus balance due to decreased vitamin D_3 and consequent secondary hyperparathyroidism leads to renal osteodystrophy or renal bone disease, with bone and joint pains. Calcification of soft tissues is another complication that can develop.

Other clinical features include anaemia due to impaired RBC synthesis. Hypertension arises due to stimulation of the renin angiotensin system by the reduced renal blood flow, resulting in vasoconstriction. The resultant cardiovascular damage worsens renal function. Other related symptoms are shortness of breath and fatigue. Azotemia and other metabolic changes cause anorexia, weight:loss, gastrointestinal irritability, nausea, vomiting and diarrhoea. Subcutaneous nasal or GI bleeding can occur due to increased capillary fragility. Mouth ulceration, taste changes, neurological symptoms, increased susceptibility to infection due to malnutrition also commonly observed.

Controlling the symptoms, minimizing complications, and slowing the progression of the disease, therefore is the main focus for the management of CRF, particularly the dietary management which is discussed next.

16.9.3 Dietary Management

Feeding is a challenge in CRF as anorexia and taste changes reduce food intake. The main focus of dietary management is on protein, sodium, potassium, phosphate, water and adequate non-protein calories. Individual modifications are required based on clinical profile, treatment and response of the patient. Keeping in mind the general objectives of diet treatment in renal disease, the following modifications are required.

Energy: About 2000-2500 Kcal/day are recommended or 30-40 Kcal/kg/day for adults and about 100-150 Kcal/kg/day for children. If the calorie intake is inadequate, endogenous protein catabolism and gluconeogenesis occur to supply energy and further aggravate uremia. Therefore, 300-400 g of carbohydrates are recommended.

Protein: Protein needs to be restricted. However, enough has to be provided to minimize tissue catabolism. About 0.5 g/kg/day is recommended but has to be regulated depending on declining renal function. A protein intake of 35-40 g /day (60-70% of high biological value protein) with liberal calorie intake can maintain the nitrogen equilibrium for long periods while reducing azotemia. If BUN rises, the protein intake may need to be restricted to 20 g/day. High biological value proteins from milk and eggs are recommended to provide all the essential amino acids. To reduce the nitrogen load, in advanced cases, mixture of essential amino acids or nitrogen free precursors of the essential amino acids like a keto or α hydroxy analogs may be recommended.

The potential benefits of protein restriction include:

- Decreases glornerular hyperfiltration, which may slow progression of glomerulosclerosis.
- Protein restricted diets are phosphorus restricted, which delays onset of renal secondary hyperparathyroidism and may slow progression of glomerulosclerosis,
- Reduces proteinuria in glomerulopathies.
- Reduce net acid load.
- May reduce serum lipids.
- Improves the symptoms of uremia.

Sodium: Sodium intake will vary between 500 mg to 2.0 g/day. Weight loss and decreasing urine volume usually indicate a need for additional sodium, whereas if hypertension and oedema are present, the sodium intake needs to be restricted.

Potassium: The failing kidney cannot excrete potassium adequately and therefore intake is kept at about 1500 mg/day (35 to 40 mEq /day). The potassium intake has to be adjusted to maintain normal levels in blood. In severe vomiting and diarrhoea, significant losses of potassium can occur and in these conditions, careful potassium supplementation may be needed. Potassium content of various vegetables and the

methods of leaching potassium are given in subsection 16.11.4. Read this information carefully.

Calcium ***andphosphorus:*** To maintain calcium and phosphorus balance and prevent or delay renal bone diseases, calcium supplements (1-2 g/day) are recommended and phosphate is restricted to 800-1200 mg/day. Phosphate binding agents may be used if required to reduce absorption. Calcium carbonate supplements can help buffer metabolic acidosis, if present. It is iinportant to remember not to start calcium supplements, unless phosphate is restricted, to avoid soft tissue calcification.

Vitamin: Multivitamin supplements are recommended for a diet with < 40 g/day of protein. Supplements of vitamin D_3 may be recommended, based on need.

Fluid: Intake is dependent on urine output and water balance. Fluid intake should be adequate to stimulate urine output for excretion of wastes but should avoid excess fluid retention at the same time.

Some nutritional supplements are available in the inarket that are very specific for this condition and can be recommended if **the** dietary intake is unsatisfactory. Details related to commonly available commercial enteral nutrition formulas for renal patients is given later in section 16.12. You may like to review these formulas now. So go ahead and review these formulas.

The summary of the dietary guidelines for CRF is given in Table 16.6 for your ready reference.

Table 16.6: Dietary guidelines for CRF

Nutrients	Comments
Energy	30-40 Kcal/kg/day for adults and 100-150 Kcal/kg/ day for children.
Carbohydrates	300-400 g/day to avoid endogenous protein catabolism, gluconeogenesis and subsequently uremia
Proteins	0.5 g/kg/day, with 60-70% as high BV protein. To reduce N_2 load, a mixture of essential amino acids is recommended.
Sodium	500 mg-2.0g/day. Additional Na in case of weight loss and decreasing urine volume and restriction of Na in case of oedema and hypertension.
Potassium	Intake must be kept at 1500 mg/day (3-0 mEq/day) and in case of significant losses, potassium supplements should be given,
Calcium and Phosphorous	Calcium supplementation–1 to 2 g/day and phosphate to be restricted to 800-1200 mg/day
Vitamin	Multivitamin supplements, specially vitamin D_3
Fluid	Intake is dependent on urine output and water balance.

Now let us recall what we have learnt *so* far by answering the questions given in check your progress exercise 2.

Check Your Progress Exercise 2

1. Enlist the etiological factors leading *to* acute renal failure?

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2. What is the recommended protein intake for ARF and CRF patients?

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3. Why proteins of high biological value are recommended in renal failure?

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4. What sodium intake is recommended for CRF patient?

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In the last section you may recall studying that CRF leads to severe illness and requires some form of renal replacement therapy (such as dialysis) which is called end-stage renal disease (ESRD). Let us get to learn about this condition next.

16.10 END STAGE RENAL DISEASE (ESRD)

Do you know that in India with a population of more than 1 billion an approximate 100,000 patients develop ESRD? Of these, 90% never see a nephrologist and 10% who do consult a specialist find it difficult to afford the treatment.

The condition when kidneys have lost all or most of their ability to function with GFR < 5 ml/minute is called end stage renal disease (ESRD). Today, there are new and better treatments for ESRD that replace the work of healthy kidneys. The treatment of choice includes dialysis – haemodialysis (HD) and peritoneal dialysis (PD) – and kidney transplantation.

We shall learn about these treatments next. To get you started we suggest you refer to the Applied Physiology Course (MFN-001), Unit 7, section 7.10 and 7.11 which focuses on dialysis and kidney transplant. Go through the discussion presented in these sections to understand what is dialysis and renal transplant. This information will help you understand the dietary management elaborated here in this section.

16.10.1 Dialysis

So what does the process of dialysis involve? Yes, the process of dialysis involves cleansing the blood of metabolic wastes, based on the principle of osmosis and diffusion. A semi permeable porous membrane is used in dialysis to separate the patient blood carrying excess fluid and metabolic wastes and the hypotonic "dialysis fluid" called dialysate. Through osmosis and diffusion, the metabolic waste and excess water move into the dialysate. The pores of the semi permeable membrane do not permit large particles like protein and RBC to pass through, but smaller water-soluble molecules can pass.

You may also recall studying that there are two types of dialysis. These include:

b) peritoneal dialysis

A brief review of these two types follows.

Haemodialysis (HD) – In this, patient's blood circulates outside the body through what is commonly referred to as an "artificial kidney machine". An opening is created to connect an artery and a vein. Blood leaves the body via the artery, into the dialyses and after cleansing, flow back to the body via the vein as shown in Figure 16.4.

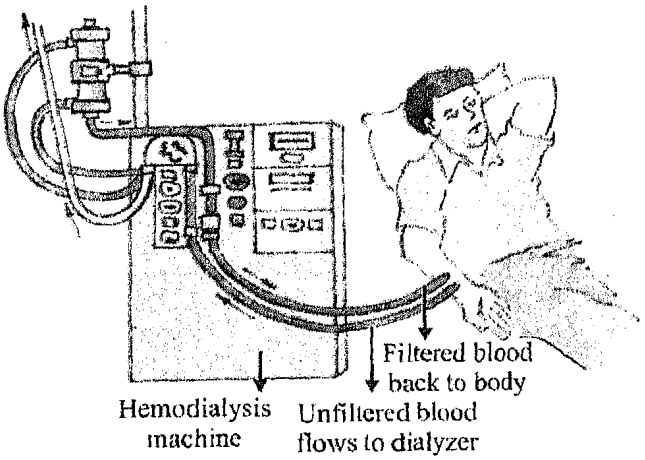

Figure 16.4: Haemodialysis

Peritoneal dialysis (PD) – In this, the patient's peritoneum is used as the semi permeable membrane and excess water and metabolic wastes are removed by injecting the dialysis fluid into the peritoneal cavity as illustrated in Figure 16.5. After same time, the fluid with the metabolic waste is drained out from the peritoneum. Peritoneal dialysis is less effective than haemodialysis and can result in loss of intact large molecular proteins also.

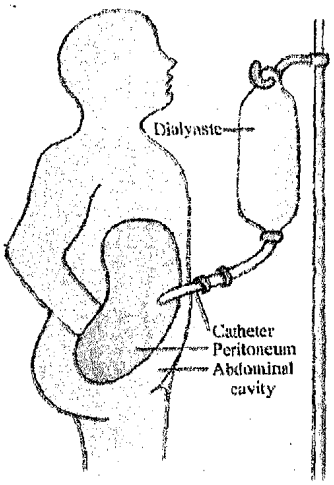

Figure 16.5: Peritoneal Dialysis

For long-term use, continuous ambulatory peritoneal dialysis (CAPD) may be used based on facilities available. In this, the dialysis fluid is exchanged 4-5 times daily. It is also important to prevent/control infection. In some cases, continous cyclic peritoneal dialysis CCPD or Intermittent Peritoneal Dialysis (IPD) may be used.

What is the role of dietary management during dialysis is discussed next.

16.10.2 Dietary Management during Dialysis

Once dialysis is started, the diet in ESRD can be liberalized, taking care that accumulation of metabolic wastes and water is prevented between treatments and biochemical balance is maintained.

The objectives of dietary management thus are to maintain balance of protein, energy, fluid and electrolytes, calcium and phosphorus, while making up losses of water-soluble nutrients lost in the dialysate. The dietary guidelines include:

Energy: Up to 35-40 Kcal/kg/day for adults and 100 Kcal or more /kg/day for children is recommended to meet the body needs and minimize tissue protein breakdown. Fats and carbohydrates are the main energy sources used. Some restriction of total and saturated fats may be needed, as dialysis patients are prone to cardiovascular disease.

Protein: The requirement is increased due to losses in the dialysate. In haemodialysis, 1.2-1.5 g/kg/day is required. At least 70% of the protein given should be of high biologicalvalue from eggs, fish, chicken and milk, though milk may need to be limited being a rich source of potassium.

This protein intake helps to maintain positive nitrogen balance, replace losses and prevent undue accumulation of nitrogen wastes, between treatments. Amino acid replacement may also be required in case of large losses.

Sodium: A daily intake of 1500 to 2500 mg may be permitted to control fluid retention and hypertension. This restriction helps to prevent pulmonary oedema or congestive heart failure because of fluid overload. Regular assessment of the kidneys ability to handle sodium and water is important to determine the intake.

Potassium: A daily intake of 1500-2500 mg is prescribed to prevent hyperkalcmia. Potassium accumulations easily cause cardiac arrhythmias or cardiac arrest.

Phosphorus: This may need some restriction.

Vitamins and Minerals: **A** daily supplement of water-soluble vitamins and minerals are usually given, as these are lost in the dialysate. Fat-soluble vitamins may be retained. Thus, their supplements are avoided except vitamin D. Supplements of minerals like calcium, iron and zinc are recommended.

Fluid Usually 400-500 ml (basal losses) plus the urine output is recommended. The fluid intake must take into account all sources of fluid input and output to maintain balance. Mild fluid retention between treatments usually occurs.

Patient counseling and support is an important part of dietary management to help renal patients understand their dietary modifications, the foods permitted and those to be avoided. Counseling about the ways to increase the palatability of the restricted diets can encourage the patient to increase their dietary intake.

Next, we move on to kidney transplant.

16.10.3 Kidney Transplantation

Kidney transplantation, you may recall studying, is a *procedure* that surgically places a healthy kidney *from* a donor into the recipient's body. This new kidney does the work of the failed kidneys. Donated kidneys may come preferably from blood relatives, after tissue and blood matching. Success of kidney transplant has improved with the use of immunosuppressive drugs and steroids to prevent organ rejection and infection. Post transplant nutritional support is required for this major surgical procedure. Optimal energy and protein intake are important for recovery, as you may recall studying earlier in Unit 5 dealing with surgery. Initially, while on medication, some restriction of sodium, simple sugars, total fat, cholesterol and saturated fat may be required. This is because of the side effects of immunosuppressant and steroids. With recovery and reduction or withdrawal of medication, the diet can be normalized. Table 16.7 gives the nutrient requirements for adult with end stage renal disease based on the type of therapy.

Table **16.7:** Nutrient requirement **for** adult with end stage renal **renal** disease based on type **of therapy**

Theraphy	**Energy (Kcal/kg BW)**	**Protein (g/kg IBW)**	Fluid **(ml/day)**	**Sodium (g/day)**	Potassium **(g/day)I**	Phosphorus **(g/day)**
Haemodialysis	35	1 output	500+urine 2-3	Variable	2-3	1-1.2
Imtermittent Peritoneal Dialysis (IPD)	30(40-50 for repletion)	1.2	500+urine output	2-3	2-3	1-1.2
Continuous Ambulatory Peritoneal Dialysis (CAPD)	**25 (40-50** for repletion)	**1.2**	Adlibitum (minimum or **2000** ml/day + urine output)	6-8	3-4	1.5-2
Transplant (4 to 6 weeks after transplant)	30-35	1.5-2	Adliditum	Variable	Variahle	1.2, calcium 1-2 g/day
Transplant (six weeks or longer	To achieve **IBW**	1	Adlibitum	Variable	Variable	Calcuium 1-2 g/day

Modified from: Krause's food, Nutrition and Diet Therapy by Mahan Cesott Stump and C 2000)

The discussion presented ahovc, we hope will help you in dietary management of ESRD. To recapitulate what you have Icarnt so far, answer the questions given in check your progress excrcisc 3.

Check **Your Progress** Exercise **3**

1. Enlist the different choices of therapy for treatment of end stage renal disease patients.

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2. Why is extra protein needed for renal patients undergoing dialysis?

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3. What is the recommended potassium intake for haemodialysis patients and why?

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4. Why sodium restriction is required for dialysis patients?

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The discussion so far focused on disorders linked to the loss of renal functions due to damage to the kidneys. Condition related to the presence of stones in the urinary tract are discussed in the next section.

16.11 RENAL CALCULI

Renal calculi or stones may be formed in the kidney, pelvis or ureter, when the concentration of components in the urine reaches a level in which crystallization is possible. The process of stone formation is also called *nephrolithiasis* or *urolithiasis*. Figure 16.6 illustrates the kidney stone. A kidney stone is a solid mass that consists of a collection of tiny crystals. There can be one or more stones present at the same time in the kidney or in the ureter. They generally are composed of calcium salt, uric acid, cystic or struvite (triple salt of ammonium, magnesium and phosphorus). Crystals of these substances interspersed in an organic matrix or base can form stones of varying size.

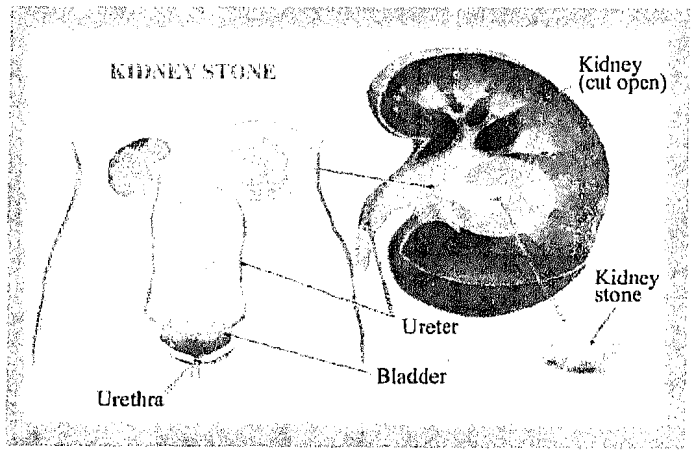

Figure 16.6: Kidney stone

What causes the formation of these stones? Let us find out.

16.11.1 Etiology

Kidney stones may form when the urine becomes too concentrated with certain substances. These substances may create small crystals that become stones. Different types of stones form under different circumstances. Although, the exact cause of renal stones is not known, but multiple factors may play a role directly or indirectly, mostly related to urine composition and urinary tract environment. Some possible etiological factors in different types of calculi are enumerated herewith:

- *Calcium stones (oxalate, phosphate and carbonate):* Excess intake of calcium, oxalate, hypervitaminosis D, hyperparathyrodism, prolonged bed rest, renal tubular acidosis, idiopathic hypercalciuria.
- *Struvite stones:* Mostly due to urinary tract infection (UTI).
- *Uric acid stones:* Impaired purine metabolism with increased urinary excretion of uric acid.

Cystine stones: Hereditary metabolic defect in renal tubular reabsoi-ption.

Hot climates leading to over concentrated urine, changes in pH of urine also predispose to stone formation. Although there is a high intake of animal protein, deficiency of vitamins B_6 and magnesium are reported to play a role in the causation of these stones.

Some types of stones tend lo run in families, Some types may be associated with bowel disease, ileal bypass for obesity, or renal tubule defects. So what ever may be the cause of the renal stones, it causes discomfort and typical symptoms. These symptoms are highlighted next.

16.11.2 Clinical Symptoms

The kidney stones may not produce symptoms until they begin to move down the ureter, causing pain. The pain is usually severe and often starts in the flank region, then moves down to the groin. The patient experiences blood in the urine, severe pain, weakness and in some cases fever. Laboratory examination and chemical analysis can help determine location, size and main constituent of stones to determine the treatment.

So then, what is the treatment or management strategy for renal calculi? We shall learn about this in the next section, with particular focus on dietary management.

16.11.3 Dietary Management

The goal of treatment of renal calculi is to relieve symptoms and prevent further complications. Treatment, therefore, varies depending on the type of stone and the extent of symptoms or complications.

Kidney stones usually pass on their own. In acute stage with stones less than 5 mm in diameter, it may pass in the urine by drinking large quantities of fluid especially water and needs no specific treatment. Stones more than 7 mm in diameter may require surgical treatment or lithotripsy by which large stones are broken down and excreted in the urine.

Although, role of diet in the formation of urinary stones is not well established, it is advisable to have liberal fluid intake, a balanced diet and restrict foods based on the main constituent of the stones. Table 16.8 gives information related to different stones and their corresponding diet restriction.

Table **16.8: Different stones and their corresponding** diet **restrictions**

Main constituents	Diet restriction	Urine pH
• Calcium stones	Calcium – 400-600 mg	
-phosphate	Phosphorus - 1000-1200 mg	Acid
-oxalate		
• Struvite stones	Low phosphorus diet	Acid
• Uric acid	Low purine diet	Alkaline
e Cystine	Low methionine diet	Alkaline

Besides liberal fluid intake and some dietary restriction, urine pH control helps based on the chemical composition of the stone, mainly via acidifying or alkalinizing agents or diet. Binding agents to bind the stone constituent may also be used.

Let us now have a look at the dietary sources of various constituents of the renal stones.

16.11.4 Dietary Sources of Various Constituents of the Renal Stones

Dietary sources of potassium, sodium, calcium, oxalate and uric acid are given in this section. We begin our study with the dietary sources of potassium.

A. Sources of potassium

Table 16.8 presents the sources of potassium. The methods or direction for leaching potassium is also highlighted subsequently in this section.

Table **16.9:** Sources of **potassium in** the diet **(Potassium / 100 g** Vegetable)

(0-100 mg)	(101-200 mg)	(201 mg and above)
Beetroot	Bitter gourd	Amaranth
Bottle gourd	Brinjal	Coriander leaves
Broad beans	Cauliflower	Drumstick leaves
Cucumber	Cabbage	Spinach
Field beans	Carrot	Colocasia
Green Mango	Onion small	Potato
Peas	Radish white	Sweet potato
Lettuce	Ladies finger	Tapioca
Fenugreek leaves	Pumpkin	Green papaya
	Green tomato	Yam

On review of Table 16.9, you would realize that it is not possible to always consume only **low** potassium content foods. Therefore, next we present you a method by which we can leach potassium and reduce its content to a major extent in foods.

Directions / Methods for leaching Potassium

Method I – Wash, peel and cut vegetables into small pieces. Soak in warm water for **2-3** hours. Discard water. Add large volume of fresh water and cook the vegetables. Discard water.

Method II – Peel vegetables and cut into small pieces. Bring to boil in a large quantity of water. Discard excess water and cook in a large volume of fresh water. Discard excess water.

Let us next have a look at a few dietary sources of Na, Ca. oxalate and uric acid.

B. Sources of Sodium

Food items with a high sodium content (these food items should be avoided):

- Salt
- Baking powder
- Bicarbonate of soda
- Canned, preserved and processed food items as processed cheese, sauce, margarine, etc.
- Bacon, ham and sausages
- Meat and yeast extracts like marmite
- Salted chips, nuts, popcorn and biscuits
- Commercial salad dressings and sauces
- Soup cubes
- Malted beverages like Boost, Bournvita and Proteinex.
- Flavour enhancers such as Monosodium glutamate (MSG)

C. Sources of Calcium, Oxalate and Uric Acid

Calcium

Beans, cauliflower, egg yolk, figs, milk and milk products like cheese, parieer, curds, molasses and potatoes.

Oxalate

cashewnuts, chickoo, chocolate, cocoa, custard apple, groundnuts, spinach, strawberries, tomatoes and tea.

Uric Acid

Fish herring, fish roe, salmon, sardines, kidney, liver, meat extracts and soups, and sweet bread.

We just learnt about the dietary sources of various minerals which may or may not contribute to the formation of renal calculi. Lastly, we move on to a few commonly available commercial enteral nutrition formulas which could be of help to renal patients.

16.12 COMMONLY AVAILABLE COMMERCIAL ENTERAL NUTRITION FORMULAS FOR RENAL PATIENTS

Earlier in this course in Unit 4, you may recall studying about the enteral and parenteral feeding and the formulas commonly used for these feeding methods. Some common enteral nutrition formulas for renal patients are highlighted here in this section.

Reno – care I (For pre-dialysis state) Sachet –51.5 g provide:

Low protein (3.75 g/sachet)
Low sodium (4.10 mEq/sachet)
Low potassium (2.40 mEq /Sachet)
Low phosphorus (80 mg/sachet)

Reno - care II (For dialysis state) Sachet –52.5 g provide:

Moderate protein (8.80 g/sachet)
Low sodium (4.10 mEq/sachet)
Low potassium (240 mEq /sachet)
Low phosphorus(86.0 mg /sachet)

Nutrenal CRF

Composition per 100 g :

Protein 7.74 g
Sodium 202.72 mg
Potassium 288.83 mg
Phosphorus 188.86 mg

Nutrenal Dialysis

Composition per 100 g

Protein 18.09 g
Sodium 214.71 mg
Potassium 27356 mg
Phosphorus 177.65 mg

With this, we end our study of nutritional management of renal diseases.

Check Your Progress Exercise 4

1. What is a renal calculi? Give its etiological factors.

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2. What dietary advice would you give to a patient suffering from struvite stones?

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3. Give any five potassium rich food sources in the diet. Suggest a method for leaching potassium.

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16.13 LET US SUM UP

In this unit, we learnt that kidney performs a myriad of functions including excretory, endocrinal and metabolic. The kidney maintains the fluid, electrolyte balance and normal volume, pH and osmolality of blood. Metabolic waste products and toxins are excrctccl from the body while essential nutrients are reabsorbed by the kidneys.

Further, the unit focused on the diseases of the kidney. These diseases alter the capacity of the kidneys to perform the functions. The common diseases in which diet plays an important role are glomerulonephritis, nephrotic syndrome, acute renal failure, chronic renal failure and renal calculi. We learnt that these diseases have multiple etiological factors and the clinical manifestations and biochemical parameters govern the dietary management of these diseases.

The main objectives of the dietary management arc to reduce the excretory workload of the kidney and prevent progression of renal damage while maintaining satisfactory nutritional status and as near normal fluid, acid base and electrolyte balance. To meet these objectives, modifications in the diet are required mainly for protein, sodium, potassium, fluid, calcium and phosphate. The main objective is to try and maintain the internal milieu of the body. In case of poor oral intake, commercially available supplements in the market may have to be used. When the kidney function fails dialysis or transplantation is recommended.

Finally, the unit focused on renal calculi. Patients with kidney stones have to be treated according to the type of stones and diet therapy is recommended thereafter. Thus, nutritional care will depend largely on the type of renal disease and biochemical and clinical manifestations.

16.14 GLOSSARY

Anuria : complete lack of urine excretion.

Anorexia : loss of appetite.

Ascites : an accumulation of fluid in the abdominal cavity.

Azotemia	:	an accumulationof abnormal quantities of urea, uric acid, creatinine and other nitrogenous wastes.
Catabolism	:	the destructive phase of inetabolism in which complex substances are converted into simpler ones.
Congenital	:	of genetic origin.
Dialysate	:	the solution used in dialysis to remove waste products and excess fluid from the blood.
Diuretic	:	an agent that increases urine secretion.
Oedema	:	swelling of tissues.
End Stage Renal Disease	:	a disease characterized by the kidney's inability to excrete waste products, maintain fluid and elecrolyte balance and produce hormones.
Haematuria	:	presence of blood in the urine.
Haemodialysis	:	a method of clearing waste products from the blood in which blood passes through a semi permeable membrane of the artificial kidney.
Hyperkalemia	:	high levels of potassium in blood.
Hypoproteinemia	:	low levels of protein in blood.
Mesangium	:	it is the central part of the renal glomerulus between capillaries.
Metabolic **calcification**	:	the deposition of calcium in tissues as a result of abnormalities in calcium and phosphate levels in the blood and fluids.
Nephritic syndrome	:	the syndrome of hematuria, hypertension and loss of renal function that results from acute inflammation of the capillary loops of the glomerulus.
Nephrotic syndrome	:	a condition resulting from loss of the glomerular barrier to protein and is characterized by massive oedema and proteinuria, hypoalbuminemia, hypercholesterolemia and abnormal bone metabolism.
Oliguria	:	the condition characterized by a urinary output of less than 500 ml/day.
Osmosis	:	the passage of solvent through a semi permeable membrane that separates solutions of different cuncentrations, The solvent is usually water.
Osteodystrophy	:	A bone disease characterized by softening and fibrous degeneration of bone and the formation of cysts in bone tissue, caused by chronic renal failure,
Periorbital Oedema	:	refers to accumulation of fluid around the orbit of the eye.
Peritoneal dialysis	:	the method of removing wastes from the blood in which diffusion carries them from the blood through the semi-permeable peritoneal membrane into the dialysatc.

Proteinuria	:	protein in urine.
Pyelonephritis	:	is an ascending urinary tract infection that has reached the pyelurn (pelvis) of the kidney.
Renal failure	:	the inability of a kidney to excrete the daily load of wastes.
Uremia	:	a clinical syndrome of weakness, nausea, vomiting, muscle cramps, metallic taste and often neurological impairment.

16.15 ANSWERS TO CHECK YOUR PROGRESS EXERCISES

Check Your Progress **Exercise 1**

1. The three major functions of the kidneys-include: excretion, endocrine and metabolic. Excretion of urea and its products and maintenance of electrolyte and fluid balance are the main excretory function of kidneys. Endocrine functions pertain to synthesis of vitamin D_3, release of erythropoeitin factor and stimulation of renin-angiotensin mechanism, where as metabolic functions involve nitrogen metabolism and gluconeogenesis during starvation.

2. Five significant kidney function lest include: Creatinine clearance test, alkaline phosphatase, haemoglobin levels, urine analysis, serum electrolytes.

3. The diseases caused due to kidney dysfunction include acute and chronic nephritis, nephrotic syndrome, acute renal failure, chronic renal failure, dialysis, renal transplantation and renal calculi.

4. Nephrotic syndrome primarily occurs due progressive glomerulonephritis, diabetes, collagen disease or drug reactions from exposure to heavy metals or reaction to toxin venom. Oedema can be corrected by making up for the protein losses and by bringing the plasma albumin levels to normal. Reduction in sodium intake is required to correct the oedema. Approximately 2-3 g of sodium is recommended. Diuretics are usually used to prevent further oedema.

5. Hematuria and proteinuria are the two major symptoms of glomerulonephritis. Initially, 0.5-0.6 g protein/kg IBW is provided. Higher levels of protein (1g/kg/ IBW) are provided if BUN levels remain within the normal range. Fluid intake is 500 to 700 ml/day plus the volume of urine output in previous 24 hours.

Check Your Progress Exercise 2

1. ARF can be due to circulatory shock, large blood loss and reduced renal blood flow as in traumatic injury, shock, severe burns, surgery, septicemia or due to mismatched blood transfusions, nephrotoxins like carbon tetrachloride, certain poisonous mushrooms, immunological reactions to drugs like certain antibiotics, renal disease like acute glomerulonephritis.

2. The recommended levels of protein for a patient with ARF are protein intake of 0.5-lg /kg IBW/day, and for CRF - protein intake of 0.5 g/kg IBW/day.

3. High biological value proteins are recommended to meet the body's need of essential amino acids and yet not burden the body with excess non-essential amino acids.

4. Sodium intake of 500-2000 mg /day is recommended for CRF patient.

Check Your Progress Exercise 3

1. The different choices available for patients with ERSD are haemodialysis, CAPD, IPD, CCPD and kidney transplantation.

2. Extra amount of proteins, with the requirements ranging from 1.2-1.5 g/kg/day is needed due to losses of protein in the dialysate.

3. A daily intake of 1500-2500 mg is prescribed to prevent hyperkalemia. Potassium accumulation can cause cardiac arrhythmias or cardiac arrest.

4. Sodium is restricted in patients undergoing dialysis to prevent pulmonary oedema or congestive heart failure because of fluid overload.

Check Your Progress Exercise 4

1. Renal calculi or stones are composed of calcium salt, uric acid, cystic or struvite (triple salt of ammonium, magnesium and phosphorus). The etiological factors range from concentrated urine in hot weathers, high animal protein intake, deficiency of vitamin B and alterations in pH of the urine.

2. A patient suffering from struvite stones is advised to have liberal fluid intake, a balanced diet and to restrict foods based on the main constituent of the stones; which in the case of struvite stones is low phosphorous diet.

3. Rich sources of potassium include amaranth, coriander leaves, drumstick leaves, spinach, colocasia, potato, sweet potato, tapioca, green papaya and yam.

The method for potassium leaching is given in subsection 16.11.4. Read carefully and answer on your own.