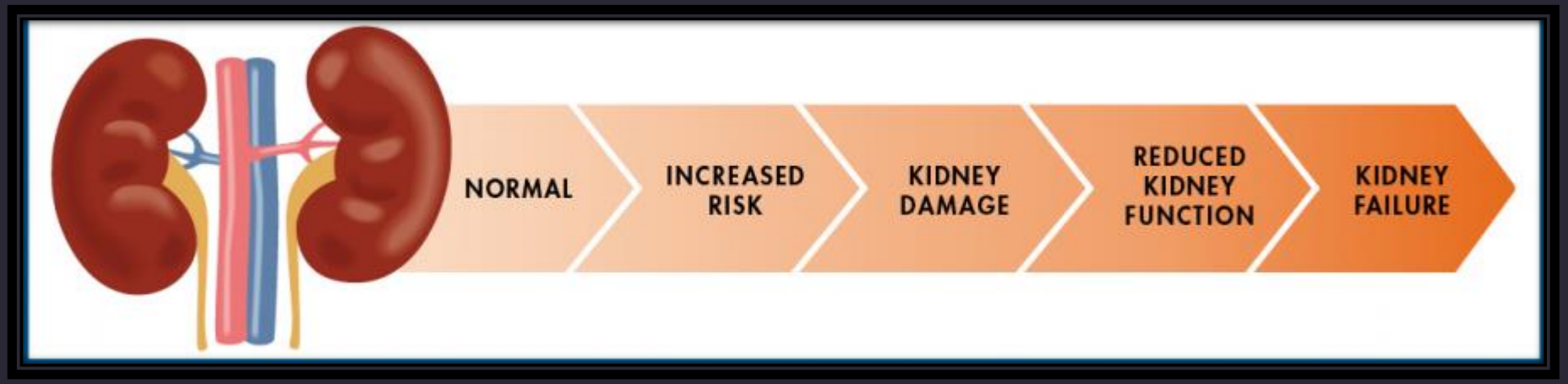
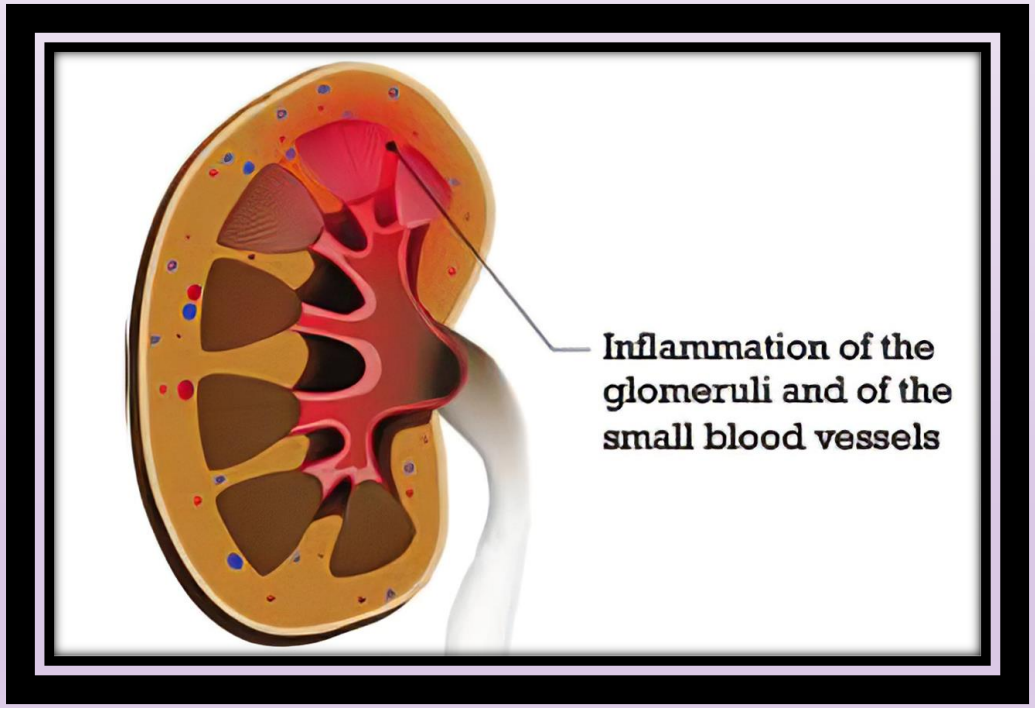
UNIT 3 , CHAPTER 7

RENAL DISEASES



GLOMERULONEPHRITIS

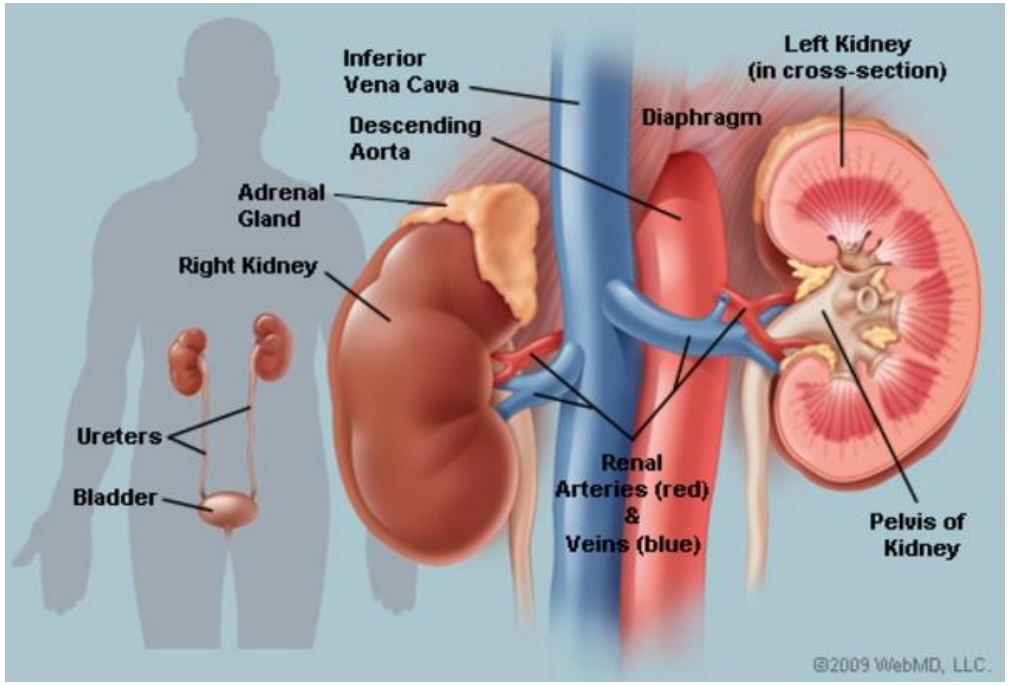


SUBMITTED TO- Mrs. NAMRITA MAM
SUBMITTED BY- SURBHI(611) TOPIC- GLOMERULONEPHRITIS

INTRODUCTION

The kidneys are the main organs of the body through which nitrogenous wastes are excreted in the forms of urea. They are carried through the blood stream where the blood capillaries are coiled up in lakhs of tufts called GLOMERULUS. The water soluble small molecular compounds such as urea, waste salts, and other materials, nutrients such as amino acids, sugars, uric acid, along with water, pass through walls of these capillaries and into small cavity that surrounds each glomerulus. Blood cells and large molecular substances are retained. The fluid that remains in the tubule is the urine. The excreted fluid empties from the cavity through a long-coiled tubule.

An adult human being has a blood volume of about 7-8% of his body weight, i.e. an approximate range of 4-5 litres of blood which flow through the kidneys. A normal healthy human being has a pair of kidneys that filter about 135-150 litres of fluid every 24 hours while only about 1.5 litres of urine is eliminated from the body during the same period. In brief, the kidney is a very efficient filter of the body.

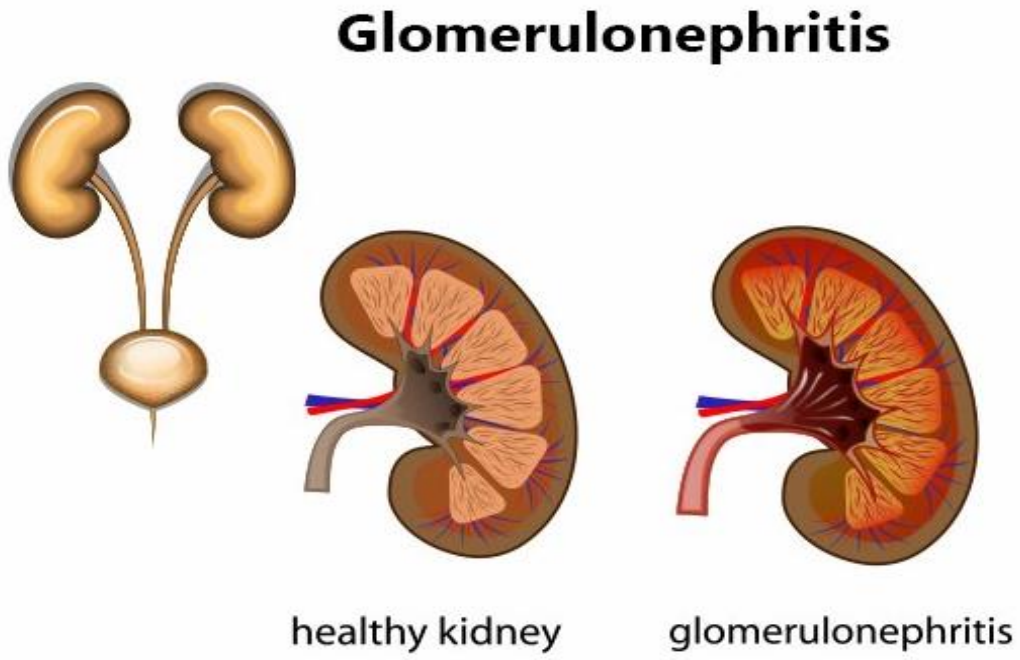


GLOMERULONEPHRITIS

GLOMERULONEPHRITIS, also known as nephritis, literally means an inflammation of the nephrons. The inflammatory process affects the glomeruli i.e. the tuft of the capillaries in the head of the nephron. It is most common in its acute form in children between 3-10 years of the age and young adults, although a few cases of initial attacks do occur in adults over the age of 50 years. Although, the glomeruli are particularly affected, the functioning of tubules is also affected.

CLASSIFICATION
Generally, two types are seen –

- ACUTE GLOMERULONEPHRITIS
- CHRONIC GLOMERULONEPHRITIS



ACUTE GLOMERULONEPHRITIS

In acute cases, primarily the glomeruli are affected. This disease normally occurs commonly after a streptococcal fever, tonsillitis, pneumonia or respiratory infections. It occurs most frequently in children than in adults.

CLINICAL SYMPTOMS- Blood and protein (albumin) may be found in the urine along with some nitrogen retention. There may be varying degrees of edema, hypertension and renal insufficiency. Urine output decreases or in some cases there may be no urine output. Swelling around the ankles, puffiness around the eyes, headache nausea and vomiting are very common symptoms.

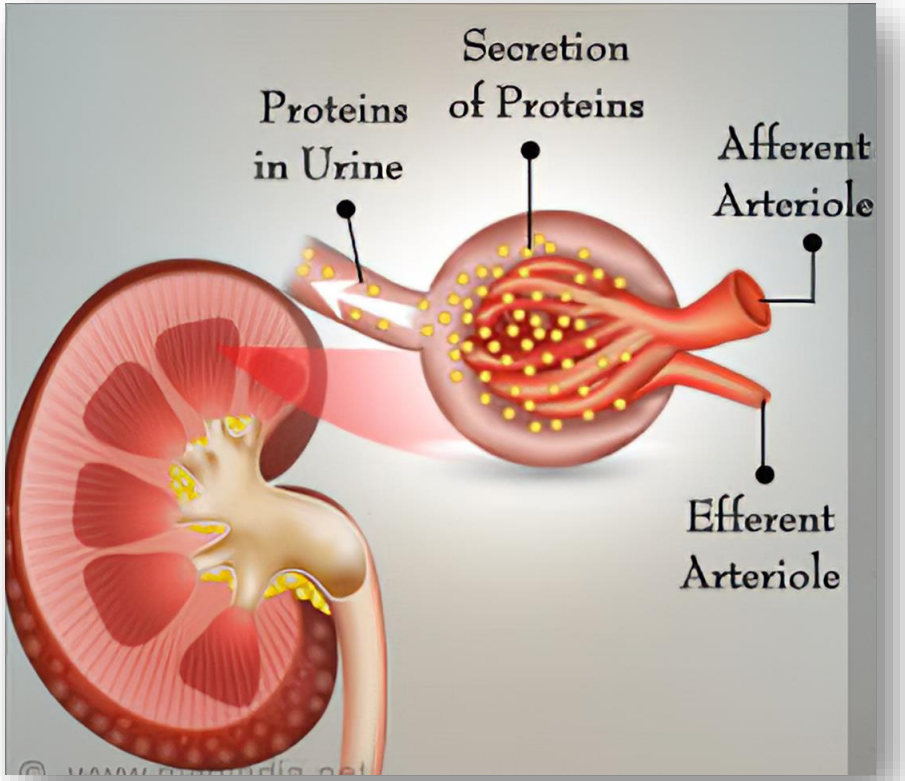
CHRONIC GLOMERULONEPHRITIS

CLINICAL CHARACTERISTICS- It is not yet confirmed as to what causes this but it may be asymptomatic for several years and may indicate gradually increasing involvement- proteinuria, hematuria, hypertension, and vascular changes in the retina. Frequent urination and nocturia are seen. In some patients nephrotic syndrome characterized by massive edema and severe protein losses through urine may develop. Eventually renal failure may occur.

ETIOLOGY

The most common cause is a previous streptococcal infection. In the past, acute glomerulonephritis was common, frequently following infection with ß- haemolytic streptococci in children and young adults. At present, with the use of penicillin, the streptococci are usually rapidly obliterated, and so acute glomerulonephritis has become less common.

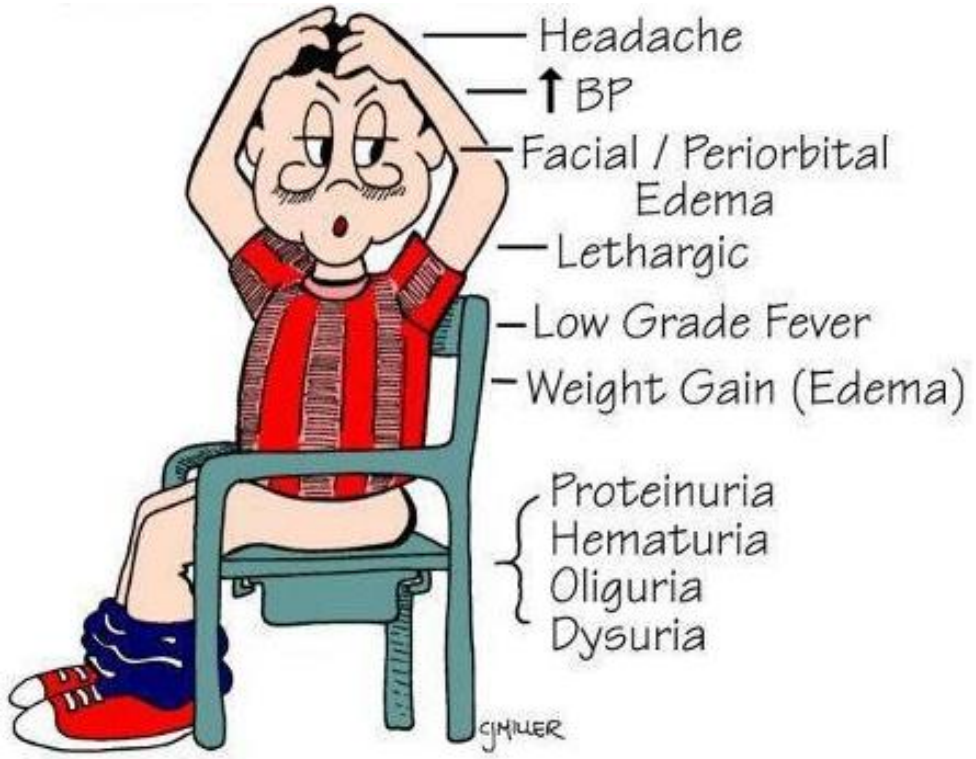
In post streptococcal glomerulonephritis, glomerular damage is not caused directly by the infection of the kidneys. The glomerular damage is caused by deposition in the glomeruli of the soluble immune complexes of streptococcal antigen with antibody formed in response to foreign organisms. This antigen-antibody complex binds components of complement- a complex series of enzymatic proteins in the blood that serves as part of the body's immune system. This activated complement in turn provides active chemical factors that attract leukocytes. The lysosomal enzymes of these leucocytes cause injury or damage to the glomeruli.



Antigen- antibody complexes are deposited between the epithelial cells of the nephron capsule and the basement membrane of the glomeruli. As the disease progresses, lesions develop, scar tissue is formed and thus, obstruction to the glomerular circulation results. This is followed by fatty degeneration and necrosis of the tubules and ultimately results in nephron destruction.

CLINICAL SYMPTOMS-

Hematuria, and proteinuria are the classical symptoms. Oedema and the shortness of breath can occur as a result of sodium and water retention and circulatory congestion. Tachycardia and elevated blood pressure may be present. The patient is generally anorexic which may contribute to feeding problems. There may be oliguria or anuria and uremia which signals development of acute renal failure.



DIAGNOSTIC TESTS

Any disturbance in the functioning of the kidneys can be judged from a series of tests conducted on the blood and urine of the patients. The extent of damage to the kidneys can be easily detected on the basis of the following biochemical tests.

IN BLOOD

- BLOOD UREA
- SERUM CRETININE
- SERUM URIC ACID

a) SERUM TOTAL PROTEINS
b) SERUM ALBUMINS
c) SERUM GLOBULINS
d) ALBUMIN: GLOBULIN RATIO

- SERUM ELECTROLYTES; SODIUM, POTASSIUM, CHLORIDE
- SERUM BICARBONATES
- SERUM INORGANIC PHOSPHORUS

Normal and Abnormal observation in the blood as related to kidney function tests can be

DETERMINATION	NORMAL VALUES	INCREASED IN	DECREASED IN
1. SGPT	5-35 IU at 37°C	-	Renal insufficiency
2. albumin	3.3-4.8 gm/dl	-	Renal disease
3. Calcium	9.0-11.0 mg/dl 4.5-5.5 mEq/dl	-	Chronic renal disease
4. Chloride	95- 106mEq/dl	Renal tubular disease	Salt-losing renal disease
5. Cholesterol	150-250 mg/dl	Nephrotic syndrome	-
6. Creatinine	0.7-1.5 mg/dl	Impaired renal function	-
7. Glucose	70-110 mg/dl	Chronic renal failure	-
8. Magnesium	1.2-2.4 mEq/dl	Renal disease	-
9. Inorganic phosphates	2.5-4.5 mg/dl (adults) 4-6 mg/dl (children)	Renal insufficiency	-
10. Potassium	3.8-5.6 mEq/dl	Renal glomerular disease	Renal tubular disease
11. Sodium	113-146 mEq/dl	-	Salt- losing nephritis
12. Total protein	6-8 gm/dl	-	Renal disease (protein losing)
13. Triglycerides	10-190 mg/dl	Nephrotic syndrome	-
14. uric acid	2-7 mg/dl	Renal failure	Renal tubular defect
15. urea nitrogen	7.0-23 mg/dl	impaired renal function post renal conditions	-

PREVENTION

- Lose weight if you are overweight.
- Get active. Physical activity helps control blood sugar levels.
- Quit smoking.
- Occasionally kidney checkups.
- Take medications as directed.
- Keep your blood pressure below 140/90.
- If you have diabetes, stay in your target blood sugar range as much as possible.
- Stay in your target cholesterol range.
- Eat foods lower in salt.
- Eat more fruits and vegetables

DIETARY TREATMENT

FLUID- During the first stage of treatment the fluid should be decreased to allow for disposal of Oedema fluid. Daily weighing is needed to monitor overall fluid balance. In later stages, the fluid intake is based on the volume of fluid excreted and an allowance(500ml/day) is given for insensible water loss. The fluid is calculated taken into account the water consumed with the drugs, water present in milk, curds, tea, coffee, fruit juices etc. Daily fluid replacement should be 500ml + daily amount excreted in the urine.

Insensible water loss is –

- 30ml/kg body weight for infants

-20ml/kg body weight for older children

-10 ml/kg body weight for adults

If liquids are limited, controlling thirst may be a problem. This can be overcome by sucking on a lemon wedge or eating a sour candy or chew gum to moisten mouth. Diabetics should choose sugar-free types. Blood sugar should be controlled in diabetics. Salt should be restricted to feel less thirsty. Mouth can be rinsed with water without swallowing it. To quench the thirst a few chips of ice can be sucked.

ENERGY-

For children 80kcal/kg body weight and 10% for infection is suggested. Sufficient calories is given without increasing the protein intake by means of sugar, honey, glucose, sago, fats and oil and starchy foods. By giving carbohydrates liberally, protein catabolism and starvation, ketosis are reduced. Requirements are calculated for the particular age and weight and 10% more for infections.

PROTEIN-

If the blood urea nitrogen is elevated and oliguria is present, dietary protein must be restricted. Usually the diet contains 0.5 g/kg of ideal body weight for older children and 1-1.5g/kg per day for younger children. The intake of protein is reduced to a minimum by excluding protein rich foods. A low protein diet is recommended so as to give rest to the kidneys. Complete proteins are better, to ensure maximum utilization. If anuria develops proteins should be stopped.

An intake of 20-40g/day is considered sufficient. Foodstuffs containing high amounts of poor-quality protein should be restricted. For example, pulses and groundnuts increase urea levels in the blood. Rice is preferable as it has low amount of protein but better quality of protein than wheat. Sago can be included as it does not contribute to protein. Fruits and vegetables which are usually low in protein can be prescribed provided they are also poor source of sodium and potassium.

SODIUM-

The restriction of sodium varies with the degrees of oliguria and hypertension. If renal function is impaired, the sodium will be restricted to 500-1000mg/day. As recovery occurs, sodium can be increased. If Oedema is present, sodium is restricted.

In sodium restricted diets, the following foods are avoided-

- Salt during cooking or on the table.
- Baking powder and soda bicarbonate which are added to cakes and pastries.
- Sodium benzoate, potassium metabisulphite which are added as a preservative in pickles, squashes .
- Papads, cheese, salted chips, nuts , popcorn etc.
- Soft drinks

Foods containing moderate amounts of sodium like milk, curds, pulses, vegetables, coriander seeds, cumin seeds and jaggery should be taken in restricted amounts.

POTASSIUM-

When the kidneys do not work properly, potassium builds up in the body and causes the heart to beat unevenly and stops suddenly. Too little potassium in diet is also very dangerous.

Potassium is found naturally in almost all foods particularly in fruits, vegetables, and meats. Potassium content can be reduced in vegetables by cooking in excess water and then discarding the water. Spices and condiments can be used in small quantities as they are rich in potassium.

Table 19.2 Potassium content of fruits mg/100 g

Fruits low in Potassium		Fruits high in Potassium	
Orange	9	Mango	205
Pineapple	37	Amla	225
Papaya	69	Plums	247
Apple	75	Sapota	269
Banana	88	Lemon	270
Guava	91	Peaches	453
Watermelon	160	Sweet lime	490

Table 19.1 Potassium content of vegetables mg/100 g

Low 0–100 mg	Medium 101–200 mg	High 201 mg and above
Fenugreek leaves	Carrots	Amaranth
Lettuce	Onion-big	Coriander leaves
Beetroot	Radish-white	Drumstick leaves
Radish pink	Bitter gourd	Spinach
Bottle gourd	Brinjal	Colocasia
Broad beans	Cauliflower	Potato
Cucumber	French beans	Sweet potato
Knol Khol	Ladies finger	Tapioca
Green mango	Onion stalks	Yam
Ridge gourd	Plantain flower	Drumstick
Snake gourd	Green plantain	Green papaya
	Pumpkin	
	Green tomato	

PHOSPHORUS-

Phosphorus is a mineral found in almost all foods. Large amount are found in milk, cheese, nuts, dried beans, and peas. Eating foods high in phosphorus raise the phosphorus in blood and this can cause calcium to be pulled from the bones. This will make bones weak and cause them to break easily.

FOODS TO BE INCLUDED- rice, sugar, leached vegetables, sago, honey.

FOODS TO BE RESTRICTED- milk, curd, jaggery, pulses, fruits like pineapple, papaya, banana, guava, watermelon.

FOODS TO BE AVOIDED- salts, greens rich in sodium like amaranth, bakery products, pulses, fruits like mango, amla, plums, sapota, lemon, peaches.

CLINICAL AND THERAPEUTIC NUTRITION :I

RENAL DISEASES:

ACUTE AND CHRONIC RENAL FAILURE- DIALYSIS

BY : NISHTHA DANWER

FN SEM 2

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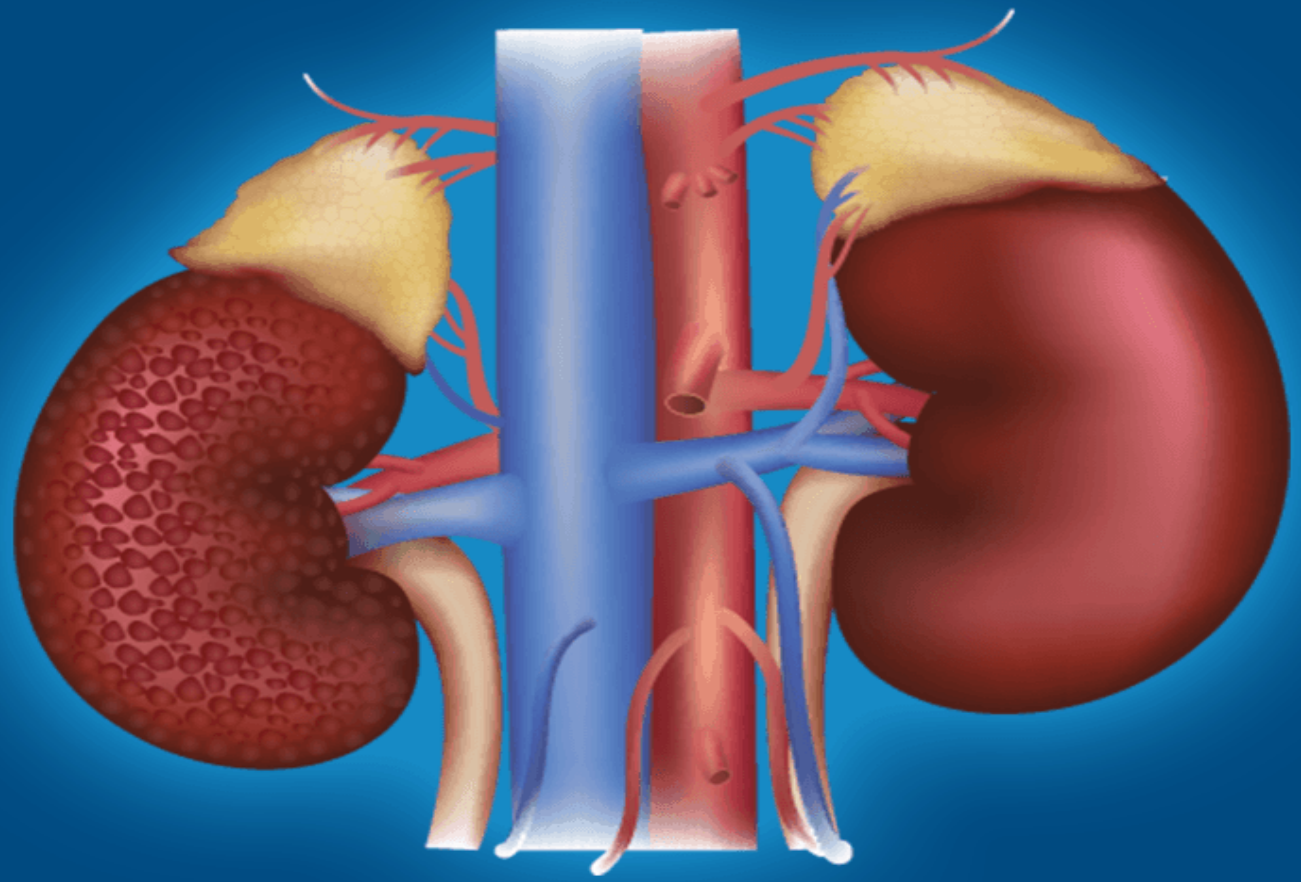
NURSE STUDY GUIDES | MEDICAL-SURGICAL NURSING

Acute Renal Failure

NURSING CARE MANAGEMENT



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ACUTE RENAL FAILURE(ARF):

- Acute renal failure causes a sudden stoppage of renal function as a result of metabolic insult or traumatic injury to the normal kidneys causing life threatening situations.
- Acute kidney failure occurs when your kidneys suddenly become unable to filter waste products from your blood. When your kidneys lose their filtering ability, dangerous levels of wastes may accumulate, and your blood's chemical makeup may get out of balance.
- Acute kidney failure — also called acute renal failure or acute kidney injury — develops rapidly, usually in less than a few days. Acute kidney failure is most common in people who are already hospitalized, particularly in critically ill people who need intensive care.

- General duration of ARF varies from a few days to several weeks. Therefore , depending on its severity and duration it is accompanied by:

a) Oliguria or anuria(the secretion of small amount or absence of urine)
b) Tissue destruction
c) Acidosis (acidification of blood)
d) Hyperkalemia(elevated serum potassium level)
e) Uremia(retention of urea and other urinary constituents of the blood)
f) Fluid and electrolyte balance.

ETIOLOGY:

- the condition was first recognized in 1940 , when it was common complication of crush injuries and shock. The causes of ARF are categorized as:

- **Impaired blood flow to the kidneys (prerenal):**
- Diseases and conditions that may slow blood flow to the kidneys and lead to kidney injury include:
- Blood or fluid loss
- Blood pressure medications
- Heart attack
- Heart disease
- Infection
- Liver failure
- Use of aspirin, ibuprofen (Advil, Motrin IB, others), naproxen sodium (Aleve, others) or related drugs
- Severe allergic reaction (anaphylaxis)
- Severe burns
- Severe dehydration

- **Damage to the kidneys:**
- These diseases, conditions and agents may damage the kidneys and lead to acute kidney failure:
- Blood clots in the veins and arteries in and around the kidneys
- Cholesterol deposits that block blood flow in the kidneys
- Glomerulonephritis , inflammation of the tiny filters in the kidneys (glomeruli)
- Hemolytic uremic syndrome, a condition that results from premature destruction of red blood cells
- Infection
- Lupus, an immune system disorder causing glomerulonephritis
- Medications, such as certain chemotherapy drugs, antibiotics and dyes used during imaging tests
- Scleroderma, a group of rare diseases affecting the skin and connective tissues
- Toxins, such as alcohol, heavy metals and cocaine
- Muscle tissue breakdown (rhabdomyolysis) that leads to kidney damage caused by toxins from muscle tissue destruction
- Breakdown of tumor cells (tumor lysis syndrome), which leads to the release of toxins that can cause kidney injury

- **Urine blockage in the kidneys:**
- Diseases and conditions that block the passage of urine out of the body (urinary obstructions) and can lead to acute kidney injury include:
- Bladder cancer
- Blood clots in the urinary tract
- Cervical cancer
- Colon cancer
- Enlarged prostate
- Kidney stones
- Nerve damage involving the nerves that control the bladder
- Prostate cancer
-

- **Risk factors:**
- Acute kidney failure almost always occurs in connection with another medical condition or event. Conditions that can increase your risk of acute kidney failure include:
- Being hospitalized, especially for a serious condition that requires intensive care
- Advanced age
- Blockages in the blood vessels in your arms or legs (peripheral artery disease)
- Diabetes
- High blood pressure
- Heart failure
- Kidney diseases
- Liver diseases
- Certain cancers and their treatments

Clinical symptom:

- Signs and symptoms of acute kidney failure may include:
- Decreased urine output, although occasionally urine output remains normal
- Fluid retention, causing swelling in your legs, ankles or feet
- Shortness of breath
- Fatigue
- Confusion
- Nausea
- Weakness
- Irregular heartbeat
- Chest pain or pressure
- Seizures or coma in severe cases

MANAGEMENT:

- The objectives of initial nutritional therapy are to support overall medical support. These include:
- Re-establishing fluid and electrolyte balance.
- Maintenance of adequate nutritional status in order to minimize the endogenous protein catabolism.
- Providing an optimum environment for wound healing.
- Preventing infection.

NUTRITIONAL MANAGEMENT:

- Nutritional care in acute kidney injury is particularly important because the patient not only has uremia, metabolic acidosis, and fluid and electrolyte imbalance, but also usually suffers from physiologic stress (example; infection or tissue destruction) that increases protein needs. The problem of balancing protein and energy needs with treatment of acidosis and excessive nitrogenous waste is complicated and delicate. In the early stages of AKI the patient is often unable to eat. Mortality in AKI is high, especially among those who are malnourished. Early attention to nutritional support and early dialysis improves patient survival
- **Protein:**

The amount of protein recommended is influenced by the underlying cause of AKI and the presence of other conditions. A range of recommended levels can be found in the literature, from 0.5 to 0.8 g/kg for non-dialysis patient to 1-2 g/kg for dialyzed patients.

- **Energy:**

Energy requirements are determined by the underlying cause of AKI and comorbidity. Energy needs can be measured at the bedside by indirect calorimetry in most ICUs. If this equipment is not available, calorie needs should be estimated at 30 to 40 kilocalorie per kg of dry body weight per day. Excessive calorie intake can lead to excess carbon dioxide production, depressing respiration.

A high calorie, low protein diet will be used in cases in which dialysis or hemofiltration is unavailable. In addition to the usual dietary sources of refined sweet and fats, special high calorie, low protein and low electrolyte formulas has been developed to augment the diet. However, care must be taken with this product because hyperglycemia is not uncommon as a result of glucose intolerance, and additional insulin is often needed.

- **Fluid and Sodium**

During the early (often oliguric) phase of AKI, meticulous attention to fluid status is essential. Ideally fluid and electrolyte intake should balance the net output.

With negligible urine output, significant contribution to total body water output include emesis and diarrhea, body cavity drains, and skin and respiratory losses. If fever is present, skin losses can be expressive; where as if the patient is on humidified air, almost no losses occur.

Sodium is restricted, based on decrease urinary production. In the oliguric face when the sodium output is very low, intake should be low as well, perhaps as low as 20 to 40 mg/day.

- **Potassium:**

Most of the excretion of potassium and the control of potassium balance are normal functions of the kidney. When renal function is impaired, potassium balance should be scrutinized carefully. In addition to dietary sources, all body tissues contain large amounts of potassium; thus tissue destruction can lead to tremendous potassium overload. Potassium intake should need to be individualized according to serum levels i.e; 30 to 50 mg/day in oliguric phase (depending on urinary output, dialysis, and serum K^+ level) replace losses in diuretic phase. The primary mechanism of potassium removal during AKI is dialysis.

- **Phosphorous:**

Limit as necessary

DIAGNOSIS:

- If your signs and symptoms suggest that you have acute kidney failure, your doctor may recommend certain tests and procedures to verify your diagnosis. These may include:
- **Urine output measurements**. Measuring how much you urinate in 24 hours may help your doctor determine the cause of your kidney failure.
- **Urine tests**. Analyzing a sample of your urine (urinalysis) may reveal abnormalities that suggest kidney failure.
- **Blood tests**. A sample of your blood may reveal rapidly rising levels of urea and creatinine — two substances used to measure kidney function.
- **Imaging tests**. Imaging tests such as ultrasound and computerized tomography may be used to help your doctor see your kidneys.
- **Removing a sample of kidney tissue for testing**. In some situations, your doctor may recommend a kidney biopsy to remove a small sample of kidney tissue for lab testing. Your doctor inserts a needle through your skin and into your kidney to remove the sample.

Chronic Renal Failure(CRF):

- Once approximately one half to two thirds of kidney function has been lost, regardless of the underlying disease, progressive further loss of kidney function ensues. In response to decreasing glomerular filtration rate (GFR), the kidney undergoes a series of adaptation to prevent this decline. Although in the short-term this leads to improvement in filtration rate, in the long term dates when accelerated loss of nephrons and progressive renal insufficiency. The nature of these adaptations involve a change in the human dynamic characteristics of the remaining glomeruli, specifically leading to increased glomerular pressure.
- Diabetes is the leading risk factor for the CKD followed by the hypertension. The National Kidney Foundation (NKF) divide CKD into 5 stages related to the estimated GFR. Stage 1 and 2 are early stages with markers like proteinurea, hematuria, or anatomic issues. Stage 3 and 4 are considered advanced stages. Stage 5 result in death unless dialysis or transplantation is initiated.


Chronic Kidney Disease
Symptoms, Causes, Diagnosis & Treatment

STAGES OF CHRONIC KIDNEY DISEASE		GFR*	% OF KIDNEY FUNCTION
Stage 1	Kidney damage with **normal** kidney function	90 or higher	
Stage 2	Kidney damage with **mild loss** of kidney function	89 to 60	
Stage 3a	**Mild to moderate** loss of kidney function	59 to 45	
Stage 3b	**Moderate to severe** loss of kidney function	44 to 30	
Stage 4	**Severe** loss of kidney function	29 to 15	
Stage 5	Kidney failure	Less than 15	

* Your GFR number tells you how much kidney function you have. As kidney disease gets worse, the GFR number goes down.

DIAGNOSIS:

- Dehydration or water intoxication, sodium depletion, high serum potassium, acidosis and increased susceptibility to infection are the most general manifestation.
- The symptoms of gastrointestinal system may be loss appetite, vomiting.
- Neurological symptoms like peripheral neuropathy convulsion and coma can also occur.
- Skin changes like pigmentation, itching and purpura.
- Renal osteodystrophy metastatic calcification and dwarfism and rickets growth failure can also occur.

NUTRITIONAL MANAGEMENT:

- With each level of CKF, a different nutritional therapy may be proposed. The primary objective of MNT are to manage the symptoms associated with the syndrome (edema, hypoalbuminemia and hyperlipidemia), decrease the risk of progression to renal failure, and maintain nutritional stores. Patients are primarily treated with statins to correct hyperlipidemia, low sodium diets, and diuretics.

some of the more common nutrition diagnosis in the CKF population include:

- Inadequate mineral intake
- Excessive mineral intake
- Imbalance of nutrients
- Excessive fluid intake
- Impaired nutrient utilization
- Altered nutrition-related laboratories values
- Food-medication interaction
- Food and nutrition related knowledge deficit

Depending on the nutrition diagnosis, interventions are adjusted for various intake of minerals, protein, and fluids.

❖PROTEIN

The recommended dietary protein level for CKD patient has changed over time. Historically, these patients received diets high in protein up to 1.5 g/kg/day in an attempt to increase serum albumin and prevent protein malnutrition. However, studies have shown that a reduction of protein intake to as low as 0.8 mg/kg/day may decrease proteinuria without adversely affecting serum albumin.

❖ENERGY

Energy intake should be approximately 35 kcal/kg/day for adults to spare protein for tissue repair and maintenance.

❖SODIUM

Edema, the most clinically apparent manifestation, indicates total body sodium overload. Additionally, because of low oncotic pressure from hypoalbuminemia, the volume of circulating that may be reduced. Therefore, control of edema in this group of diseases should be with dietary intake of 2 to 3g of sodium daily.

❖POTASSIUM

Many patients in early stage CKF take potassium-wasting diuretic and required supplementation. When urine output drops below 1 L/day, then patients may require potassium restriction as a kidney is no longer able to excrete all potassium ingested. This typically occurs rather late in stage 4 CKF.

❖ PHOSPHORUS

The importance of controlling phosphate in patients with early stage disease is often overlooked. Serum Phosphorus levels elevate at the same rate as eGFR decreases. Those with an eGFR of less than 60 should be evaluated for renal bone disease, and benefit from phosphorus restriction. The diet is typically modified to allow no more than 1000 milligram of phosphates daily, a limit that allows at approximately 1-2 dairy foods per day.

❖ LIPIDS

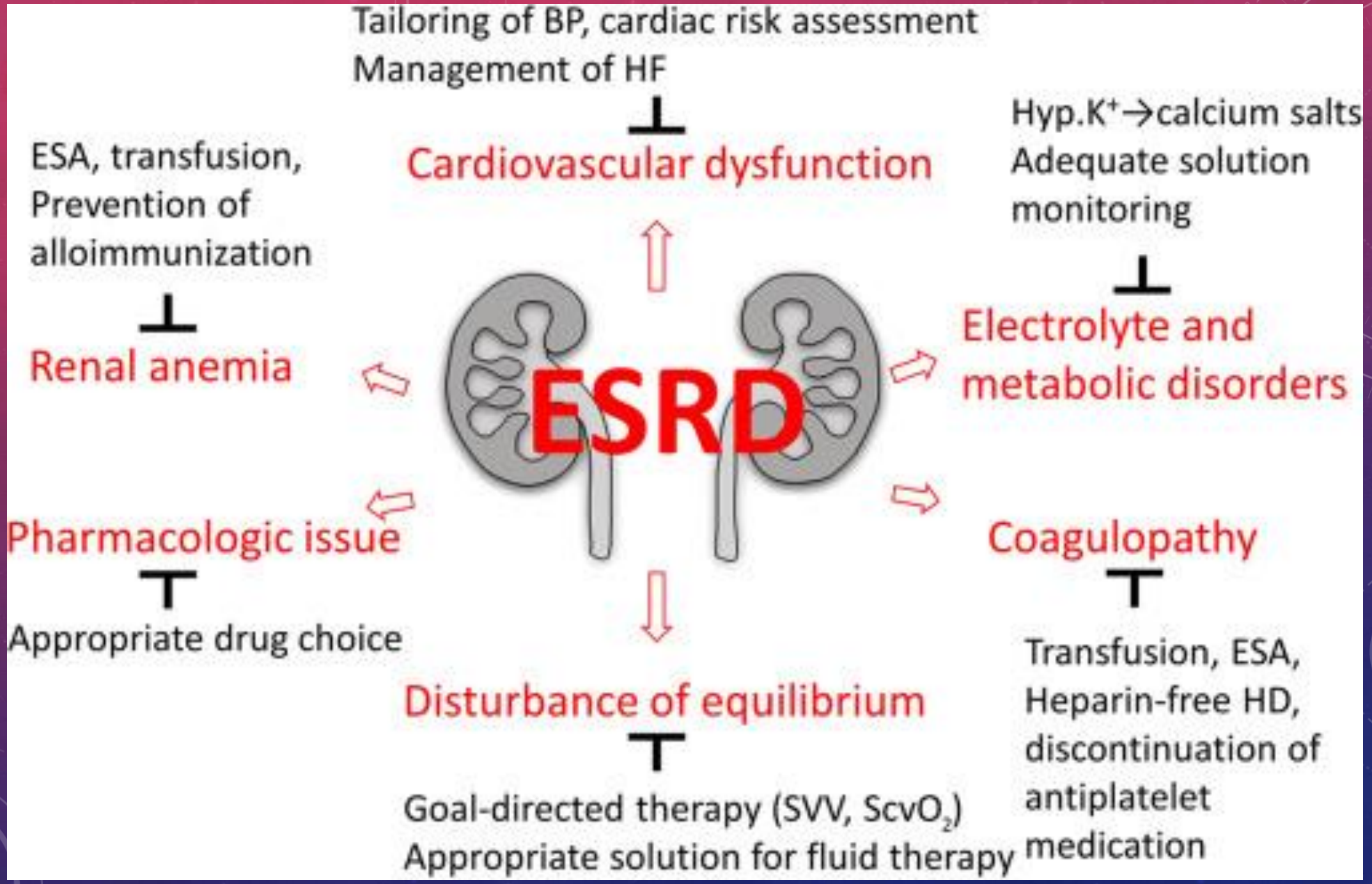
The important consequence of dyslipidemia is cardiovascular disease. Certain lipid lowering agents in combination with a cholesterol-lowering diet can reduce total cholesterol, low-density lipoprotein cholesterol and triglycerides in these patients. Lowering protein intake in adult patient may also lower fat and cholesterol intake from animal sources.

❖ VITAMINS

CKF patients are routinely recommended a water soluble renal customized vitamin supplement, because restrictions may cause the diet to be inadequate.

END STAGE RENAL DISEASE (ESRD)

- End stage renal disease reflects the Kidney's inability to excrete waste products, maintain fluid and electrolyte balance, and produce hormones. As renal failure slowly progresses, the level of circulating waste products eventually lead to symptoms of uremia. Uremia is a clinical syndrome of malaise, weakness, nausea and vomiting, muscle cramps, itching, metallic taste in the mouth and neurologic impairment that is brought about by an unacceptable level of nitrogenous wastes in the body.


Tailoring of BP, cardiac risk assessment
Management of HF
Cardiovascular dysfunction
ESA, transfusion,
Prevention of
alloimmunization
Renal anemia
Hyp.K+→calcium salts
Adequate solution
monitoring
Electrolyte and
metabolic disorders
ESRD
Pharmacologic issue
Appropriate drug choice
Coagulopathy
Transfusion, ESA,
Heparin-free HD,
discontinuation of
antiplatelet
medication
Disturbance of equilibrium
Goal-directed therapy (SVV, ScvO2)
Appropriate solution for fluid therapy

Medical Treatment:

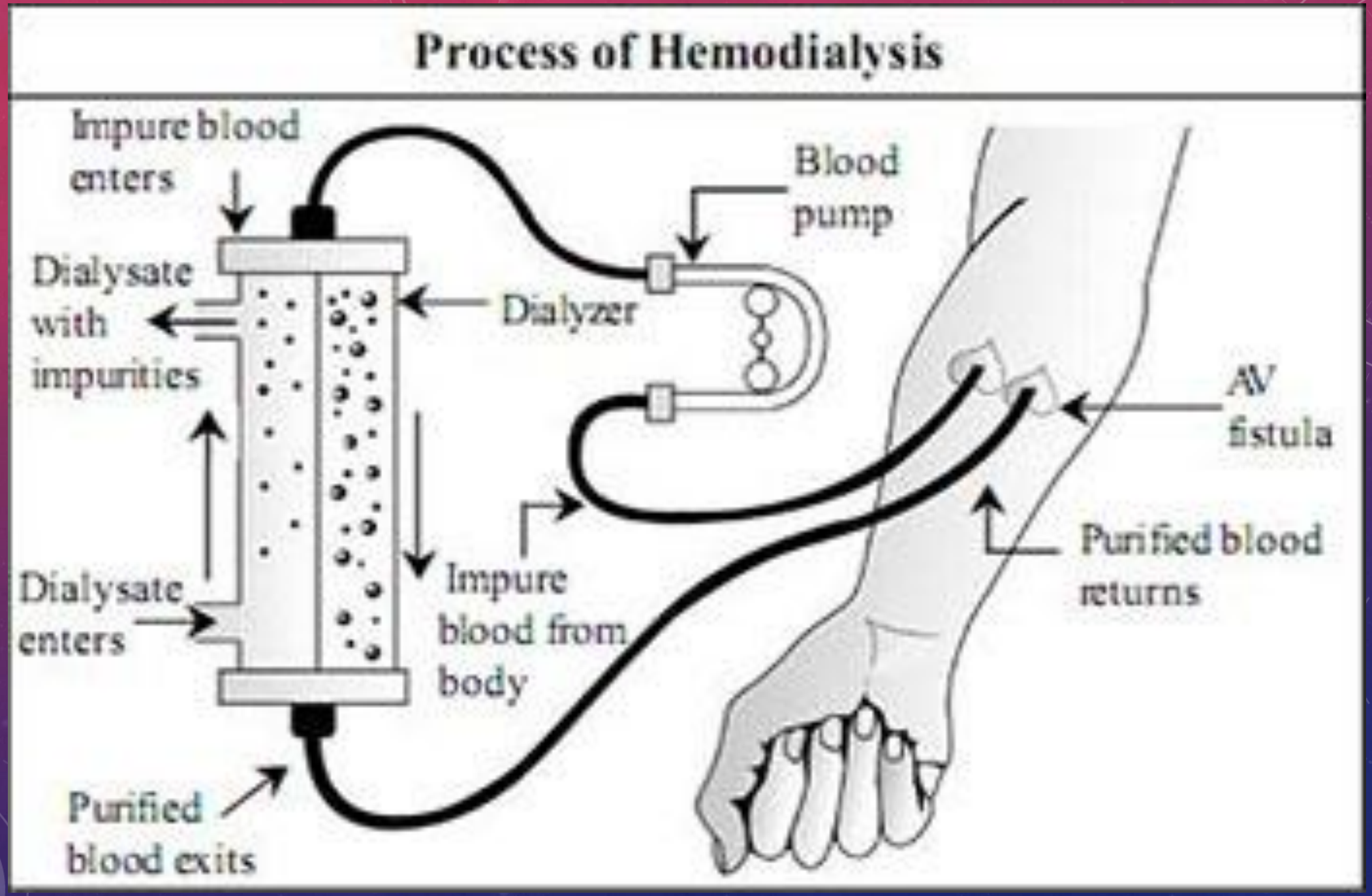
Once the patient progressive from stage 4 to stage 5 CKD, options for treatment for ESRD include dialysis, transplantation, or medical management progressing to death.

Dialysis:

Patients may choose to dialyze in an outpatient dialysis facility or they will prefer hemodialysis (HD) at home using either conventional daily or nocturnal dialysis. They may choose peritoneal dialysis (PD) and have a choice of continuous ambulatory peritoneal dialysis (CAPD) or continuous cyclic peritoneal dialysis (CCPD), or combination of the two.

Factors that come into account in this decision are:-

- Availability of family or friends to assist with therapy
- Type of water supply to the home
- Capability of the patient or
- Involved family (including eyesight and ability to perform sterile technique)
- Previous abdominal surgeries
- Membrane characteristics of the individual's peritoneal membrane
- Body size
- Cardiac status
- Presence of poor vascular access
- Desire to travel and a host of other considerations.


Process of Hemodialysis
Impure blood enters
Blood pump
Dialysate with impurities
Dialyzer
AV fistula
Purified blood returns
Dialysate enters
Impure blood from body
Purified blood exits

• HEMODIALYSIS (HD):

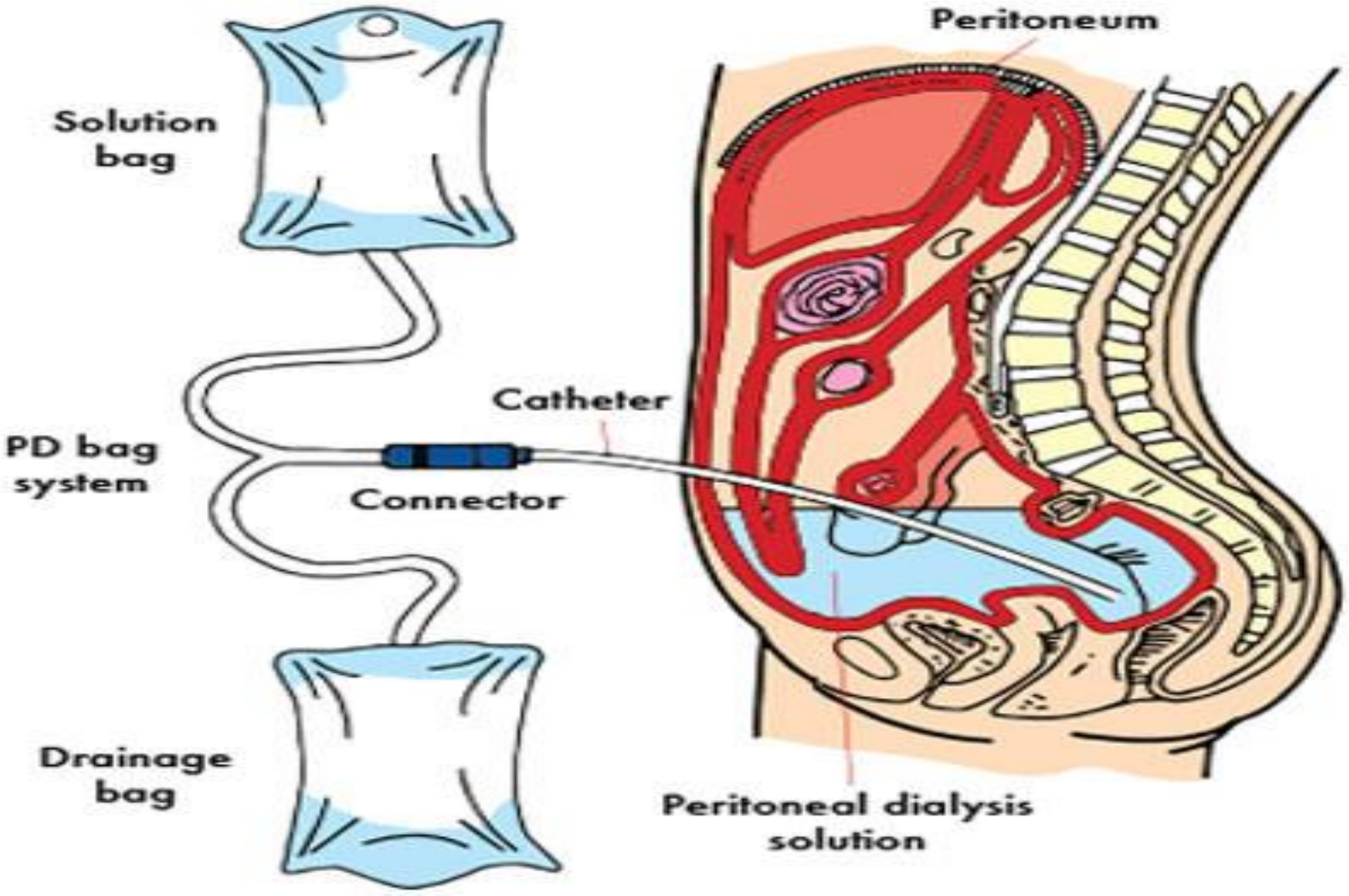
HD requires permanent access to the bloodstream through a fistula created by the surgery to connect an artery and a vein. If the patient's blood vessels are fragile, an artificial vessels called a graft may be surgically implanted. Large needles are inserted into the fistula or graft before is dialysis and removed when dialysis is complete.

The hemodialysis fluid and electrolyte content is similar to that of normal plasma. Waste products and electrolytes move by diffusion, ultrafiltration and osmosis from the blood into the dialysate and are removed.

Outpatient HD usually requires treatment of 3 to 5 hours 3 times per week in a dialysis unit. Newer therapies can shorten the duration of treatment by increasing its frequency. Patients on these more frequent dialysis therapies have lower mortality rates, approaching that of transplantation.

Patient on daily dialysis at home typically have treatment lasting from 2 to 3.5 hours 5 to 6 days a week, whereas some home dialysis patient received nocturnal dialysis 3 to 6 times a week for 8 hours, while they sleep.

Principle of Peritoneal Dialysis



• PERITONEAL DIALYSIS:

- Peritoneal dialysis (PD) make use of the body's on semipermeable membrane, the peritoneum. A catheter is surgically implanted in the abdomen and into the peritoneal cavity. Dialysate containing a height dextrose concentration is instilled into the peritoneum, where diffusion carries waste products from the blood through the peritoneal membrane and into the dialysate, water move by osmosis. This fluid is then withdrawn and discarded, and new solution is added.

Several **types of PD** exist.

1) In continuous ambulatory peritoneal dialysis (CAPD), the dialysate is left in the peritoneum and exchange to manually, by gravity. Exchanges of dialysis fluid are done 4to5 times daily, making it a 24-hour treatment.
2) In continuous cyclic peritoneal dialysis (CCPD), patient treatments are done at night by machine that does the exchanges. During the day these patients may keep a single dialysate exchange in the peritoneal cavity for extended period (called a long dwell), perhaps the entire day. Several combinations of CAPD and CCPD are possible and are referred to here as PD.

ADVANTAGES OF PERITONEAL DIALYSIS:

- Avoidance of large fluctuations in blood chemistry
- Longer residual renal function and
- The ability of the patient to achieve more normal lifestyle

COMPLICATIONS:

- Peritonitis
- Hypotension that requires fluid and sodium replacement and
- Weight gain:- tissue weight gain is experience by most patients as a result of absorbing 400 to 800 calories per day from the glucose in the dialysate.

MEDICAL NUTRITION THERAPY FOR ESRD:

1. Prevent deficiency and maintain good nutrition status through adequate protein, energy, vitamin and mineral intake.
2. Control edema and electrolyte imbalance by controlling sodium, potassium and fluid intake.
3. Prevent or retards the development of renal osteodystrophy by controlling calcium, phosphorus, vitamin D and PTH.
4. Enables a patient to eat a palatable, attractive diet that fit his or her lifestyle as much as possible.
5. Co-ordinate patient care with families, dietitians, nurses and physician in acute care, outpatient or skilled nursing facilities.
6. Provide initial nutrition education, periodic counseling and long-term monitoring of patients.

NUTRITIONAL MANAGEMENT:

➢Protein

Dialysis is a drain on body protein, so protein intake must be increased accordingly. Protein losses of 20 to 30 g can occur during a 24 hour PD, with an average of 1g/hr. Those receiving PD need 1.2 to 1.5 g/kg of body weight and those who receive HD 3 times per week require daily protein intake of 1.2 g/kg of body weight.

➢Energy

Energy intake should be adequate to spare protein for tissue protein synthesis and to prevent its metabolism for energy. Depending on the patient's nutrition status and degree of stress, between 25 and 40 kcal/kg of body weight should be provided.

➢ Fluid and Sodium Balance

The Kidney's ability to handle Sodium and water in ESRD must be accessed frequently through measurement of blood pressure, edema, fluid weight gain, serum sodium level and dietary intake.

The sodium intake should be 2 to 3 gram daily and limit on fluid intake (usually about 750 ml/day plus the amount equal to the urine output).

➢ Potassium

Potassium usually requires restriction, depending on the serum potassium level, urine output medications and the frequency of HD. The daily intake of potassium for most American is 3to4 gram. This is usually reduced in ESRD to 2.3 to 3.1 g/day and is reduced for the anuric patient on dialysis to 2 g/day.

Phosphorus

More than 99% of excess phosphate is excreted in the urine. However, as GFR decreases, phosphorus is retained in the plasma because of the last molecular weight of the phosphate molecules, it is not easily removed by dialysis and patient experience net "gain" of about one half of the phosphate we consume daily. Phosphate intake is lowered by restricting dietary sources to 1200 mg/day or less. The difficulty in implementing the phosphorus restriction comes from necessity for high-protein diet.

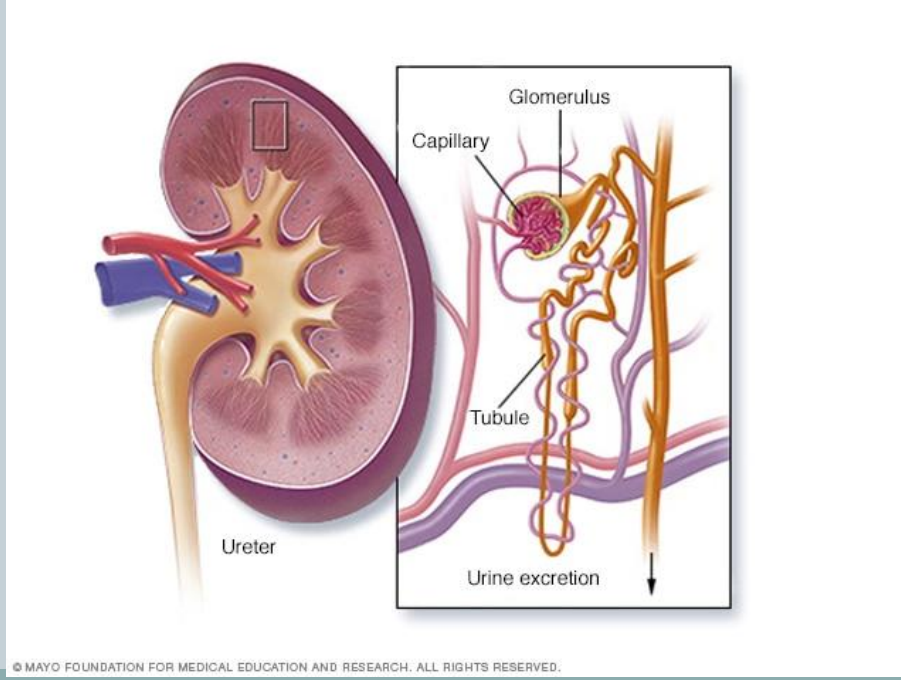

Thank
you

NEPHROTIC SYNDROME

SUBMITTED BY:
SHEENU GARG
SUBMITTED TO :
NAMRATA MAM

Nephrotic syndrome

- The nephrotic syndrome or nephrosis is characterized by a group of symptoms resulting from kidney tissue damage and impaired nephron function.



CLASSIFICATION

Primary or Idiopathic	Secondary
• no known aetiology • **Minimal change disease (MCD)** • Focal Segmental Glomerulosclerosis (FSGS) • Membranous nephropathy • congenital nephrotic syndrome	• **Systemic disease** - SLE - HSP - DM • **Infections** - Post-infectious GN - Hepatitis B - Syphilis - Malaria • **Drugs** • **Toxins and allergen** - bee sting - food allergy

Etiology

primary causes :-

- Progressive glomerulonephritis
- Minimum change disease.
- **Focal segmental glomerulosclerosis, or FSGS,** is a disease that scars the glomeruli.
- **Membranous nephropathy,** in which the membranes of the glomeruli thicken.

other causes :-

- Can also stem from diseases like diabetes or connective tissue disorders.
- May result from drug reactions specially exposure to heavy metals or reaction to toxic venom like bee sting
- Systemic lupus erythematosus
- Amyloidosis : buildup of substances called amyloid proteins in your blood. which can damage kidneys.

pathophysiology

Alteration in glomerular basement membrane

Altered glomerular protein permeability

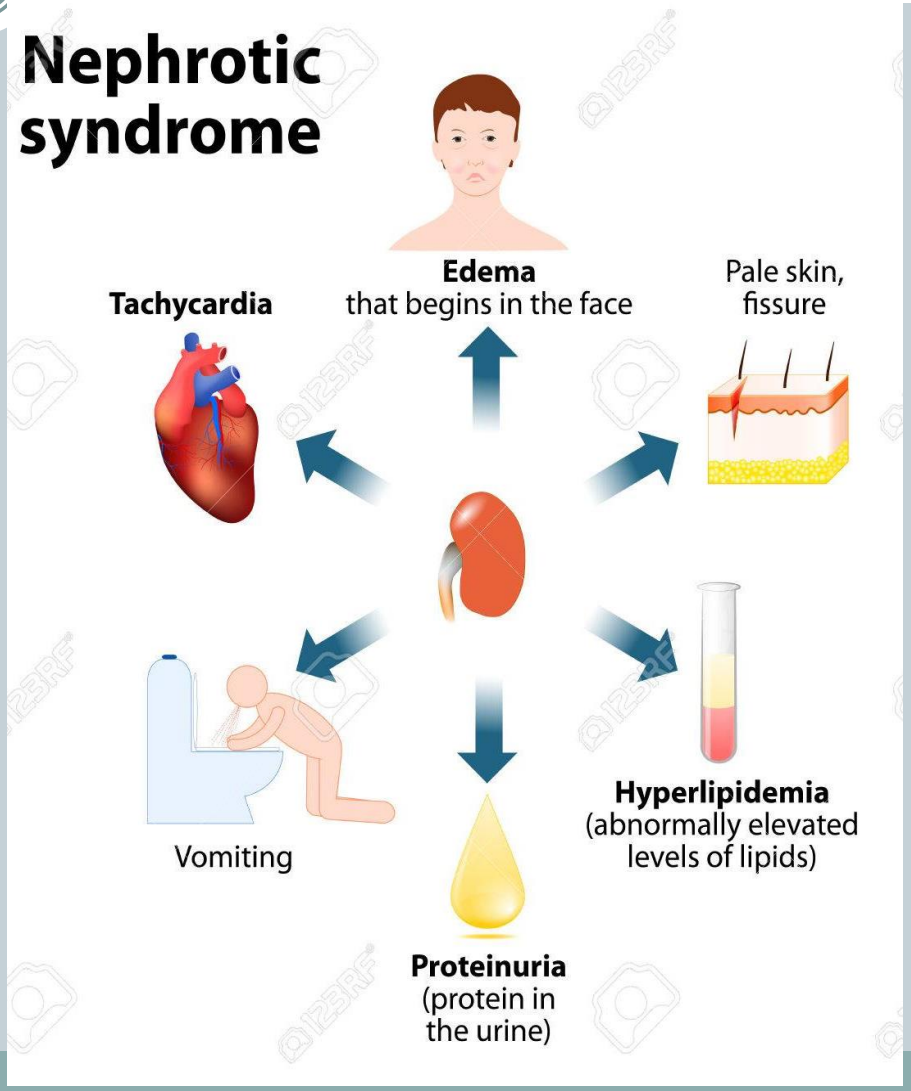
Increased loss of protein in urine

Decreased colloidal osmotic pressure and vascular volume

Increased secretion of aldosterone leading to more Tubular Na and H2O reabsorption

Clinical symptoms

- The cardinal symptom is massive edema (caused by gross loss of protein in urine some 4 to 10 gm/day).
- Ascites is common
- The abdomen becomes increasingly distended as fluid collects in serous cavities
- Often striae (stretch marks) appear on stretched skin of the extremities



Diagnosis

- Urine test,

  which will measure how much protein your kidneys are filtering out.
- **B**lood tests that check on other kidney functions.
- check for diseases that could be a secondary cause of nephrotic syndrome, such as diabetes.
- kidney biopsy, which requires taking a tissue sample for study under a microscope.

Treatment

Medications

- Blood-pressure medications to curb the pressure in glomeruli and lower the amount of protein in your urine
- Diuretics, or water pills, to reduce swelling
- Cholesterol-lowering drugs
- Blood thinners, or anticoagulants, to make blood clots less likely
- Medications that turn down your immune system, such as corticosteroids

Dialysis

a treatment in which a machine filters your blood because your kidneys can't do the job.

Dietary management

- **Energy :** sufficient kilocalories must be provided to ensure protein use for tissue synthesis; About 50 to 60 kcal/kg.
- **Carbohydrates :** provide sufficient carbohydrates for effective protein utilization and for prevention of starvation ketosis.
- **Protein** : replacement of the prolonged nitrogen deficit is a fundamental and immediate need. Daily protein requirement vary acc. to need ,from 1 gm/kg/day to larger amount in extreme cases.
- **Fats :** consume diet low in saturated fat and cholesterol
- **Vitamins and minerals :** reduce sodium to combat edema , usually 500 mg Na/day.
- **Fluid :** reduce the amount of liquids consumed each day.

Prevention

- Manage high blood pressure and diabetes, if you have them
- Be sure to get vaccines for common infections, especially if you work around people who have hepatitis or other diseases
- salt should be avoided in cooking as well as on table.
- Practice exercise and yoga and take proper rest.


THANK
YOU