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LECTURER

ICON

****Acute and Chronic renal failure****

1. Introduction:

Renal failure results when the kidneys cannot remove the body's metabolic wastes or perform their regulatory functions. The substances normally eliminated in the urine accumulate in the body fluids as a result of impaired renal excretion, leading to a disruption in endocrine and metabolic functions as well as fluid, electrolyte, and acid–base disturbances. Renal failure is a systemic disease and is a final common pathway of many different kidney and urinary tract diseases. Each year, the number of deaths from irreversible renal failure increases.

2. ACUTE RENAL FAILURE

2.1 Definition:

Acute renal failure is a syndrome of varying causation that results in a sudden decline in renal function. It is frequently associated with an increase in BUN and creatinine, oliguria (less than 500 mL urine/24 hours), hyperkalemia, and sodium retention.

2.2 Causes:

- Prerenal causes—result from conditions that decrease renal blood flow (hypovolemia, shock, hemorrhage, burns, impaired cardiac output, diuretic therapy)
- Postrenal causes—arise from obstruction or disruption to urine flow anywhere along the urinary tract
- Intrarenal causes—result from injury to renal tissue and are usually associated with intrarenal ischemia, toxins, immunologic processes, systemic and vascular disorders

2.3 Categories of acute renal failure

Three major categories of conditions cause ARF: prerenal (hypoperfusion of kidney), intrarenal (actual damage to kidney tissue), and postrenal (obstruction to urine flow).

Prerenal Failure

• Volume depletion resulting from: Hemorrhage, Renal losses (diuretics, osmotic diuresis), gastrointestinal losses (vomiting, diarrhea, nasogastric suction)
• Impaired cardiac efficiency resulting from: Myocardial infarction, Heart failure, Dysrhythmias, Cardiogenic shock
• Vasodilation resulting from: Sepsis, Anaphylaxis, Antihypertensive medications or other medications that cause vasodilation

Intrarenal Failure

• Prolonged renal ischemia resulting from: Pigment nephropathy (associated with the breakdown of blood cells containing pigments that in turn occlude kidney structures), Myoglobinuria (trauma, crush injuries, burns), Hemoglobinuria (transfusion reaction, hemolytic anemia)
• Nephrotoxic agents such as: Aminoglycoside antibiotics (gentamicin, tobramycin), Radiopaque contrast agents Heavy metals (lead, mercury), Solvents and chemicals (ethylene glycol, carbon tetrachloride, arsenic), Nonsteroidal anti-inflammatory drugs (NSAIDs), Angiotensin-converting enzyme inhibitors (ACE inhibitors)
• Infectious processes such as: Acute pyelonephritis, Acute glomerulonephritis

Postrenal Failure

• Urinary tract obstruction, including: Calculi (stones), Tumors, Benign prostatic hyperplasia, Strictures, Blood clots

2.4 Phases of acute renal failure

There are four clinical phases of ARF: initiation, oliguria, diuresis, and recovery.

- **Onset:** begins when the kidney is injured and lasts from hours to days. The initiation period begins with the initial insult and ends when oliguria develops.

- **The oliguria period** is accompanied by a rise in the serum concentration of substances usually excreted by the kidneys (urea, creatinine, uric acid, organic acids, and the intracellular cations [potassium and magnesium]). Oliguric-anuric phase: urine volume less than 400 to 500 mL/24 hours.
    - Accompanied by rise in serum concentration of elements usually excreted by kidney (urea, creatinine, organic acids, and the intracellular cations—potassium and magnesium).
    - There can be a decrease in renal function with increasing nitrogen retention even when patient is excreting more than 2 to 3 L of urine daily—called nonoliguric or high-output renal failure.
    - The minimum amount of urine needed to rid the body of normal metabolic waste products is 400 mL. In this phase uremic symptoms first appear and life-threatening conditions such as hyperkalemia develop. Some patients have decreased renal function with increasing nitrogen retention, yet actually excrete normal amounts of urine (2 L/day or more). This is the nonoliguric form of renal failure and occurs

predominantly after nephrotoxic antibiotic agents are administered to the patient; it may occur with burns, traumatic injury, and the use of halogenated anesthetic agents.

- **Diuretic phase**: begins when the 24-hour urine volume exceeds 500 mL and ends when the BUN and serum creatinine levels stop rising. In the diuresis period, the third phase, the patient experiences gradually increasing urine output, which signals that glomerular filtration has started to recover. Laboratory values stop rising and eventually decrease. Although the volume of urinary output may reach normal or elevated levels, renal function may still be markedly abnormal. Because uremic symptoms may still be present, the need for expert medical and nursing management continues. The patient must be observed closely for dehydration during this phase; if dehydration occurs, the uremic symptoms are likely to increase.
- **Recovery phase.**

  - Usually lasts several months to 1 year.
  - Probably some scar tissue remains, but the functional loss is not always clinically significant.
  - The recovery period signals the improvement of renal function and may take 3 to 12 months. Laboratory values return to the patient's normal level. Although a permanent 1% to 3% reduction in the GFR is common, it is not clinically significant.

2.5 Pathophysiology:

Acute renal failure (ARF) is a sudden and almost complete loss of kidney function (decreased GFR) over a period of hours to days. Although ARF is often thought of as a problem seen only in hospitalized patients, it may occur in the outpatient setting as well. ARF manifests with oliguria, anuria, or normal urine volume. Oliguria (less than 400 mL/day of urine) is the most common clinical situation seen in ARF; anuria (less than 50 mL/day of urine) and normal urine output are not as common. Regardless of the volume of urine excreted, the patient with ARF experiences rising serum creatinine and BUN levels and retention of other metabolic waste products (azotemia) normally excreted by the kidneys.

2.6 Clinical Manifestations:

- Prerenal—decreased tissue turgor, dryness of mucous membranes, weight loss, hypotension, oliguria or anuria, flat neck veins, tachycardia
- Postrenal—obstruction to urine flow, obstructive symptoms of BPH, possible nephrolithiasis
- Intrarenal—presentation based on cause; edema usually present
- Changes in urine volume and serum concentrations of BUN, creatinine, potassium, and so forth, as described above

2.7 Diagnostic Evaluation:

- Urinalysis—reveals proteinuria, hematuria, casts
- Rising serum creatinine and BUN levels
- Urine chemistry examinations to distinguish various forms of acute renal failure; decreased sodium
- Renal ultrasonography—for estimate of renal size and to exclude a treatable obstructive uropathy

changes in urine:

Urine output varies (scanty to normal volume), hematuria may be present, and the urine has a low specific gravity (1.010 or less, compared with a normal value of 1.015 to 1.025). Patients with urine (below 20 mEq/L) and normal urinary sediment. Patients with intrarenal azotemia usually have urinary sodium levels greater than 40 mEq/L with casts and other cellular debris. Urinary casts are mucoproteins secreted by the renal tubules whenever inflammation is present.

change in kidney contour:

Ultrasonography is a critical component of the evaluation of both acute and chronic renal failure. Although many sonographic findings are nonspecific, their diagnostic utility is greatly enhanced by a familiarity with the clinical presentation and a thorough understanding of renal Pathophysiology.

increased bun and creatinine levels (azotemia):

The BUN level rises steadily at a rate dependent on the degree of catabolism (breakdown of protein), renal perfusion, and protein intake. Serum creatinine rises in conjunction with glomerular damage. Serum creatinine levels are useful in monitoring kidney function and disease progression.

Hyperkalemia:

With a decline in the GFR, the patient cannot excrete potassium normally. Patients with oliguria and anuria are at greater risk for hyperkalemia than those without oliguria. Protein catabolism results in the release of cellular potassium into the body fluids, causing severe hyperkalemia (high serum K□□levels). Hyperkalemia may lead to dysrhythmias and cardiac arrest. Sources of potassium include normal tissue catabolism, dietary intake, blood in the GI tract, or blood transfusion and other sources (intravenous infusions, potassium penicillin, and extracellular shift in response to metabolic acidosis).

metabolic acidosis:

Patients with acute oliguria cannot eliminate the daily metabolic load of acid-type substances produced by the normal metabolic processes. In addition, normal renal buffering mechanisms fail. This is reflected by a fall in the serum CO2-combining power and blood pH. Thus, progressive metabolic acidosis accompanies renal failure

calcium and phosphorus abnormalities:

There may be an increase in serum phosphate concentrations; serum calcium levels may be low in response to decreased absorption of calcium from the intestine and as a compensatory mechanism for the elevated serum phosphate levels.

Anemia

Anemia inevitably accompanies ARF due to reduced erythropoietin production, uremic GI lesions, reduced RBC life span, and blood loss, usually from the GI tract. With use of the parenteral form of erythropoietin (Epogen), anemia is not the major problem it once was.

2.8 Management:

The kidney has a remarkable ability to recover from insult. Therefore, the objectives of treatment of ARF are to restore normal chemical balance and prevent complications until repair of renal tissue and restoration of renal function can take place. Any possible cause of damage is identified, treated, and eliminated. Prerenal azotemia is treated by optimizing renal perfusion, whereas postrenal failure is treated by relieving the obstruction. Treatment of intrarenal azotemia is supportive, with removal of causative agents, aggressive management of prerenal and postrenal failure, and avoidance of associated risk factors. Shock and infection, if present, are treated promptly. Overall, medical management includes maintaining fluid balance, avoiding fluid excesses, or possibly performing dialysis.

1. Maintenance of fluid balance is based on daily body weight, serial measurements of central venous pressure, serum and urine concentrations, fluid losses, blood pressure, and the clinical status of the patient. The parenteral and oral intake and the output of urine, gastric drainage, stools, wound drainage, and perspiration are calculated and are used as the basis for fluid replacement. The insensible fluid lost through the skin and lungs and produced through the normal metabolic processes is also considered in fluid management.

2. Fluid excesses can be detected by the clinical findings of dyspnea, tachycardia, and distended neck veins. The lungs are auscultated for moist crackles. Because pulmonary edema may be caused by excessive administration of parenteral fluids, extreme caution must be used to prevent fluid overload. The development of generalized edema is assessed by examining the presacral and pretibial areas several times daily. Mannitol, furosemide, or ethacrynic acid may be prescribed to initiate a diuresis and prevent or minimize subsequent renal failure.

3. Adequate blood flow to the kidneys in patients with prerenal causes of ARF may be restored by intravenous fluids or blood product transfusions. If ARF is caused by hypovolemia secondary to hypoproteinemia, an infusion of albumin may be prescribed. Dialysis may be initiated to prevent serious complications of ARF, such as hyperkalemia, severe metabolic acidosis, pericarditis, and pulmonary edema. Dialysis corrects many biochemical abnormalities; allows for liberalization of fluid, protein, and sodium intake; diminishes bleeding tendencies; and may help wound healing. Hemodialysis, peritoneal dialysis, or any of the new continuous renal replacement therapies may be performed.

2.8.1 PHARMACOLOGIC THERAPY

Because hyperkalemia is the most life-threatening of the fluid and electrolyte disturbances, the patient is monitored for hyperkalemia through serial serum electrolyte levels (potassium value more than 5.5 mEq/L [5.5 mmol/L]), electrocardiogram changes (tall, tented, or peaked T waves), and changes in clinical status. The elevated potassium levels may be reduced by administering cation-exchange resins (sodium polystyrene sulfonate [Kayexalate]) orally or by retention enema. Kayexalate works by exchanging a sodium ion for a potassium ion in the intestinal tract. Sorbitol is often administered in combination with Kayexalate to induce a diarrhea-type effect (it induces water loss in the GI tract). If a retention enema is administered (the colon is the major site for potassium exchange), a rectal catheter with a balloon may be used to facilitate retention if necessary. The patient should retain the resin 30 to 45 minutes to promote potassium removal. Afterward, a cleansing enema may be prescribed to remove the Kayexalate resin as a precaution against fecal impaction.

Because many medications are eliminated through the kidneys, medication dosages must be reduced when a patient has ARF. Examples of commonly used medications that require adjustment are antibiotic agents (especially aminoglycosides), digoxin, ACE inhibitors, and medications containing magnesium. Many medications have been used in patients with ARF in an attempt to improve patient outcomes. Diuretic agents are often used to control fluid volume, but they have not been shown to hasten the recovery from ARF.

Low-dose dopamine (1 to 3 g/kg) is often used to dilate the renal arteries through stimulation of dopaminergic receptors; however, research has not definitely demonstrated that dopamine prevents ARF or improves outcome in patients with established renal failure.

Atrial natriuretic peptide (ANP), an endogenous hormone synthesized by the cardiac atria, has been shown to improve renal function in multiple animal models of ARF. It has also decreased the need for dialysis in patients with oliguric acute tubular necrosis in a multisite clinical trial of patients. Patients with nonoliguric acute tubular necrosis did not benefit. Further research on ANP use is underway.

In patients with severe acidosis, the arterial blood gases or serum bicarbonate levels (CO_2-combining power) must be monitored because the patient may require sodium bicarbonate therapy or dialysis. If respiratory problems develop, appropriate ventilator measures must be instituted. The elevated serum phosphate level may be controlled with phosphate-binding agents (aluminum hydroxide). These agents help prevent a continuing rise in serum phosphate levels by decreasing the absorption of phosphate from the intestinal tract.

2.8.2 NUTRITIONAL THERAPY

ARF causes severe nutritional imbalances (because nausea and vomiting contribute to inadequate dietary intake), impaired glucose use and protein synthesis, and increased tissue catabolism. The patient is weighed daily and can be expected to lose 0.2 to0.5 kg (0.5 to 1 lb) daily if the nitrogen balance is negative (ie, the patient's caloric intake falls below caloric requirements). If the patient gains or does not lose weight or develops hypertension, fluid retention should be suspected.

Dietary proteins are limited to about 1 g/kg during the oliguric phase to minimize protein breakdown and to prevent accumulation of toxic end products. Caloric requirements are met with high-carbohydrate meals because carbohydrates have a proteinsparing effect (ie, in a high-carbohydrate diet, protein is not used for meeting energy requirements but is "spared" for growth and tissue healing). Foods and fluids containing potassium or phosphorus (bananas, citrus fruits and juices, coffee) are restricted.

Potassium intake is usually restricted to 40 to 60 mEq/day, and sodium is usually restricted to 2 g/day. The patient may require parenteral nutrition.

The oliguric phase of ARF may last 10 to 20 days and is followed by the diuretic phase, at which time urine output begins to increase, signaling that kidney function is returning. Blood chemistry evaluations are made to determine the amounts of sodium, potassium, and water needed for replacement, along with assessment for overhydration or underhydration. After the diuretic phase, the patient is placed on a high-protein, high-calorie diet and is encouraged to resume activities gradually.

2.8.3 Nursing management:

Nursing Assessment

- Determine if there is a history of cardiac disease, malignancy, sepsis, or intercurrent illness.
- Determine if patient has been exposed to potentially nephrotoxic drugs (antibiotics, NSAIDs, contrast agents, solvents).
- Conduct an ongoing physical examination for tissue turgor, pallor, alteration in mucous membranes, BP, heart rate changes, pulmonary edema, and peripheral edema.
- Monitor intake and output.

Nursing Diagnoses

- Excess Fluid Volume related to decreased glomerular filtration rate and sodium retention
- Risk for Infection related to alterations in the immune system and host defenses
- Imbalanced Nutrition: Less Than Body Requirements related to catabolic state, anorexia, and malnutrition associated with acute renal failure
- Risk for Injury related to GI bleeding
- Disturbed Thought Processes related to the effects of uremic toxins on the central nervous system (CNS)

Nursing Interventions

Achieving Fluid and Electrolyte Balance

- Monitor for signs and symptoms of hypovolemia or hypervolemia because regulating capacity of kidneys is inadequate.

- Monitor urinary output and urine specific gravity; measure and record intake and output including urine, gastric suction, stools, wound drainage, perspiration (estimate).
- Monitor serum and urine electrolyte concentrations.
- Weigh patient daily to provide an index of fluid balance; expected weight loss is ½ to 1 lb (0.25 to 0.5 kg) daily.
- Adjust fluid intake to avoid volume overload and dehydration.
    - Fluid restriction is not usually initiated until renal function is quite low.
    - During oliguric-anuric phase, give only enough fluids to replace losses (usually 400 to 500 mL/24 hours plus measured fluid losses).
    - Fluid allowance should be distributed throughout the day.
    - Avoid restricting fluids for prolonged periods for laboratory and radiologic examinations because dehydrating procedures are hazardous to patients who cannot produce concentrated urine.
    - Restrict salt and water intake if there is evidence of extracellular excess.
- Measure BP regularly with patient in supine, sitting, and standing positions.
- Auscultate lung fields for rales.
- Inspect neck veins for engorgement and extremities, abdomen, sacrum, and eyelids for edema.
- Evaluate for signs and symptoms of hyperkalemia, and monitor serum potassium levels.
    - Notify health care provider of value above 5.5 mg/L.
    - Watch for ECG changes—tall, tented T waves; depressed ST segment; wide QRS complex.
- Administer sodium bicarbonate or glucose and insulin to shift potassium into the cells.
- Administer cation exchange resin (sodium polystyrene sulfonate [Kayexalate]) orally or rectally to provide more prolonged correction of elevated potassium.
- Watch for cardiac arrhythmia and heart failure from hyperkalemia, electrolyte imbalance, or fluid overload. Have resuscitation equipment on hand in case of cardiac arrest.
- Instruct patient about the importance of following prescribed diet, avoiding foods high in potassium.
- Prepare for dialysis when rapid lowering of potassium is needed.
- Administer blood transfusions during dialysis to prevent hyperkalemia from stored blood.
- Monitor acid-base balance.
    - Monitor arterial blood gas (ABG) levels as necessary.
    - Prepare for ventilator therapy if severe acidosis is present or respiratory problems develop.
    - Administer sodium bicarbonate for symptomatic acidosis (bicarbonate deficit).
    - Be prepared to implement dialysis for uncontrolled acidosis.

Preventing Infection

- Monitor for all signs of infection. Be aware that renal failure patients do not always demonstrate fever and leukocytosis.
- Remove bladder catheter as soon as possible; monitor for UTI.

- Use intensive pulmonary hygiene—high incidence of lung edema and infection.
- Carry out meticulous wound care.
- If antibiotics are administered, care must be taken to adjust the dosage for renal impairment.

Maintaining Adequate Nutrition

- Work collaboratively with dietitian to regulate protein intake according to impaired renal function because metabolites that accumulate in blood derive almost entirely from protein catabolism.
    - Protein should be of high biologic value—rich in essential amino acids (dairy products, eggs, meat)—so that the patient does not rely on tissue catabolism for essential amino acids.
    - Low-protein diet may be supplemented with essential amino acids and vitamins.
    - As renal function declines, protein intake may be restricted proportionately.
    - Protein will be increased if the patient is on dialysis to allow for the loss of amino acids occurring during dialysis.
- Offer high-carbohydrate feedings because carbohydrates have a greater protein-sparing power and provide additional calories.
- Weigh patient daily.
- Monitor BUN, creatinine, electrolytes, serum albumin, prealbumin, total protein, and transferrin.
- Be aware that food and fluids containing large amounts of sodium, potassium, and phosphorus may need to be restricted.

Preventing GI Bleeding

- Examine all stools and emesis for gross and occult blood.
- Administer H_2-receptor antagonist (or PPI) or nonaluminum or magnesium antacids as prophylaxis for gastric stress ulcers. If H_2-receptor antagonist is used, care must be taken to adjust the dose for the degree of renal impairment.
- Prepare for endoscopy when GI bleeding occurs.

Preserving Neurologic Function

- Speak to the patient in simple orienting statements, using repetition when necessary.
- Maintain predictable routine, and keep change to a minimum.
- Watch for and report mental status changes—somnolence, lassitude, lethargy, and fatigue progressing to irritability, disorientation, twitching, seizures.
- Correct cognitive distortions.
- Use seizure precautions—padded side rails, airway and suction equipment at bedside.
- Encourage and assist patient to turn and move because drowsiness and lethargy may prevent activity.
- Use music tapes to promote relaxation.

- Prepare for dialysis, which may help prevent neurologic complications.

Patient Education and Health Maintenance

- Explain that the patient may experience residual defects in kidney function for long period after acute illness.
- Encourage reporting for routine urinalysis and follow-up examinations.
- Advise avoidance of any medications unless specifically prescribed.
- Recommend resuming activity gradually because muscle weakness will be present from excessive catabolism.

Evaluation: Expected Outcomes

- BP stable, no edema or shortness of breath
- No signs of infection
- Food intake adequate, maintaining weight
- Stools heme negative
- Appears more alert, sleeps less during the day

2.9 Complications:

- Infection
- Arrhythmias due to hyperkalemia
- Electrolyte (sodium, potassium, calcium, phosphorus) abnormalities
- GI bleeding due to stress ulcers
- Multiple organ systems failure

2.10 Prevention of acute renal failure:

- Identify patients with preexisting renal disease.
- Initiate adequate hydration before, during, and after any procedure requiring NPO status.
- Avoid exposure to nephrotoxins. Be aware that the majority of drugs or their metabolites are excreted by the kidneys.
- Monitor chronic analgesic use—some drugs may cause interstitial nephritis and papillary necrosis.
- Prevent and treat shock with blood and fluid replacement. Prevent prolonged periods of hypotension.
- Monitor urinary output and CVP hourly in critically ill patients to detect onset of renal failure at the earliest moment.
- Schedule diagnostic studies requiring dehydration so there are “rest days,” especially in elderly patients who may not have adequate renal reserve.
- Pay special attention to draining wounds, burns, and so forth, which can lead to dehydration and sepsis and progressive renal damage.

- Avoid infection; give meticulous care to patients with indwelling catheters and I.V. lines.
- Take every precaution to make sure that the right person receives the right blood to avoid severe transfusion reactions, which can precipitate renal complications.

- Correct reversible causes of acute renal failure (eg, improve renal perfusion; maximize cardiac output, surgical relief of obstruction).
- Be alert for and correct underlying fluid excesses or deficits.
- Correct and control biochemical imbalances—treatment of hyperkalemia.
- Restore and maintain BP.
- Maintain nutrition.
- Initiate hemodialysis, peritoneal dialysis, or continuous renal replacement therapy for patients with progressive renal failure and other life-threatening complications.

3. CHRONIC RENAL FAILURE (END-STAGE RENAL DISEASE)

3.1 Definition:

Chronic renal failure (CRF, end-stage renal disease, ESRD, chronic kidney disease, CKD) is a progressive deterioration of renal function, which ends fatally in uremia (an excess of urea and other nitrogenous wastes in the blood) and its complications unless dialysis or a kidney transplantation is performed.

or

Chronic renal failure, or ESRD, is a progressive, irreversible deterioration in renal function in which the body's ability to maintain metabolic and fluid and electrolyte balance fails, resulting in uremia or azotemia (retention of urea and other nitrogenous wastes in the blood).

3.2 Incidence:

ESRD has increased by almost 8% per year for the past 5 years, with more than 300,000 patients being treated in the United States (USRDS, 2001). ESRD may be caused by systemic diseases, such as diabetes mellitus (leading cause); hypertension; chronic glomerulonephritis; pyelonephritis; obstruction of the urinary tract; hereditary lesions, as in polycystic kidney disease; vascular disorders; infections; medications; or toxic agents. Autosomal dominant polycystic kidney disease accounts for 8% to 10% of cases of ESRD in the United States and Europe. Comorbid conditions that develop during chronic renal insufficiency contribute to the high morbidity and mortality among patients with ESRD. Environmental and occupational agents that have been implicated in chronic renal failure include lead, cadmium, mercury, and chromium. Dialysis or kidney transplantation eventually becomes necessary for patient survival. Dialysis is an effective means of correcting metabolic toxicities at any age, although the mortality rate in infants and young children is greater than adults in the presence of other, nonrenal diseases and in the presence of anuria or oliguria.

3.3 Causes:

- Hypertension, prolonged and severe
- Diabetes mellitus
- Glomerulopathies (from lupus or other disorders)
- Interstitial nephritis
- Hereditary renal disease, polycystic disease
- Obstructive uropathy
- Developmental or congenital disorder

Consequences of Decreasing Renal Function

- Rate of progression varies based on underlying cause and severity of that condition.
- Stages: decreased renal reserve → renal insufficiency → renal failure → ESRD.
- Retention of sodium and water causes edema, heart failure, hypertension, ascites.
- Decreased glomerular filtration rate (GFR) causes stimulation of renin-angiotensin axis and increased aldosterone secretion, which raises BP.
- Metabolic acidosis results from the kidney's inability to excrete hydrogen ions, produce ammonia, and conserve bicarbonate.
- Decreased GFR causes increase in serum phosphate, with reciprocal decrease in serum calcium and subsequent bone resorption of calcium.
- Erythropoietin production by the kidney decreases, causing profound anemia.
- Uremia affects the CNS, causing altered mental function, personality changes, seizures, and coma.

3.4 Pathophysiology:

As renal function declines, the end products of protein metabolism (which are normally excreted in urine) accumulate in the blood. Uremia develops and adversely affects every system in the body. The greater the buildup of waste products, the more severe the symptoms. There are three well-recognized stages of chronic renal disease: reduced renal reserve, renal insufficiency, and ESRD. The rate of decline in renal function and progression of chronic renal failure is related to the underlying disorder, the urinary excretion of protein, and the presence of hypertension. The disease tends to progress more rapidly in patients who excrete significant amounts of protein or have elevated blood pressure than in those without these conditions.

3.5 Stages of Chronic Renal Disease:

Stage 1

Reduced renal reserve**,** characterized by a 40% to 75% loss of nephron function. The patient usually does not have symptoms because the remaining nephrons are able to carry out the normal functions of the kidney.

Stage 2

Renal insufficiency occurs when 75% to 90% of nephron function is lost. At this point, the serum creatinine and blood urea nitrogen rise, the kidney loses its ability to concentrate urine and anemia develops. The patient may report polyuria and nocturia.

Stage 3

End-stage renal disease **(ESRD)**, the final stage of chronic renal failure, occurs when there is less than 10% nephron function remaining. All of the normal regulatory, excretory, and hormonal functions of the kidney are severely impaired. ESRD is evidenced by elevated creatinine and blood urea nitrogen levels as well as electrolyte imbalances. Once the patient reaches this point, dialysis is usually indicated. Many of the symptoms of uremia are reversible with dialysis.

3.6 Clinical Manifestations:

- GI—anorexia, nausea, vomiting, hiccups, ulceration of GI tract, and hemorrhage
- Cardiovascular—hyperkalemic ECG changes, hypertension, pericarditis, pericardial effusion, pericardial tamponade
- Respiratory—pulmonary edema, pleural effusions, pleural rub
- Neuromuscular—fatigue, sleep disorders, headache, lethargy, muscular irritability, peripheral neuropathy, seizures, coma
- Metabolic and endocrine—glucose intolerance, hyperlipidemia, sex hormone disturbances causing decreased libido, impotence, amenorrhea
- Fluid, electrolyte, acid-base disturbances—usually salt and water retention but may be sodium loss with dehydration, acidosis, hyperkalemia, hypermagnesemia, hypocalcemia (see page 803)
- Dermatologic—pallor, hyperpigmentation, pruritus, ecchymoses, uremic frost
- Skeletal abnormalities—renal osteodystrophy resulting in osteomalacia
- Hematologic—anemia, defect in quality of platelets, increased bleeding tendencies
- Psychosocial functions—personality and behavior changes, alteration in cognitive processes

Signs and Symptoms of Chronic Renal Failure

Neurologic
Weakness and fatigue; confusion; inability to concentrate; disorientation; tremors; seizures; asterixis; restlessness of legs; burning of soles of feet; behavior changes

Integumentary
Gray-bronze skin color; dry, flaky skin; pruritus; ecchymosis; purpura; thin, brittle nails; coarse, thinning hair

Cardiovascular
Hypertension; pitting edema (feet, hands, sacrum); periorbital edema; pericardial friction rub; engorged neck veins; pericarditis; pericardial effusion; pericardial tamponade; hyperkalemia; hyperlipidemia

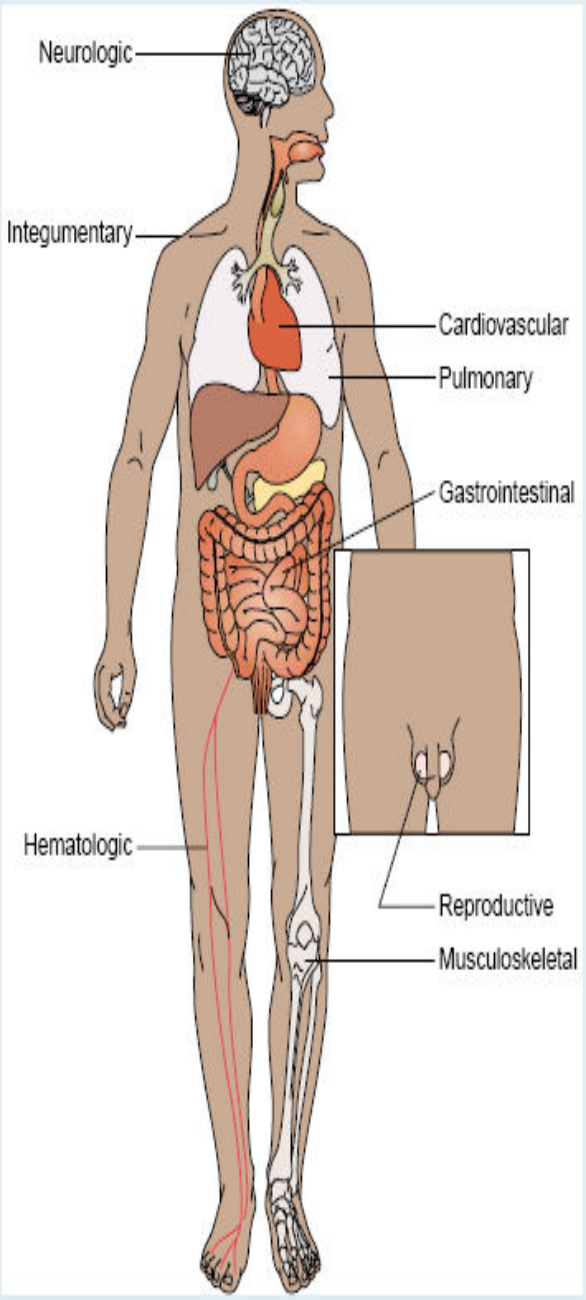
Pulmonary
Crackles; thick, tenacious sputum; depressed cough reflex; pleuritic pain; shortness of breath; tachypnea; Kussmaul-type respirations; uremic pneumonitis; "uremic lung"

Gastrointestinal
Ammonia odor to breath ("uremic fetor"); metallic taste; mouth ulcerations and bleeding; anorexia, nausea, and vomiting; hiccups; constipation or diarrhea; bleeding from gastrointestinal tract

Hematologic
Anemia; thrombocytopenia

Reproductive
Amenorrhea; testicular atrophy; infertility; decreased libido

Musculoskeletal
Muscle cramps; loss of muscle strength; renal osteodystrophy; bone pain; bone fractures; foot drop



3.7 Diagnostic Evaluation:

- Complete blood count (CBC)—anemia (a characteristic sign)
- Elevated serum creatinine, BUN, phosphorus
- Decreased serum calcium, bicarbonate, and proteins, especially albumin
- ABG levels—low blood pH, low carbon dioxide, low bicarbonate
- 24-hour urine for creatinine, protein, creatinine clearance

glomerular filtration rate:

Decreased GFR can be detected by obtaining a 24-hour urinalysis for creatinine clearance. As glomerular filtration decreases (due to nonfunctioning glomeruli), the creatinine clearance value decreases, whereas the serum creatinine and BUN levels increase. Serum creatinine is the more sensitive indicator of renal function because of its constant production in the body. The BUN is affected not only by renal disease but also by protein intake in the diet, catabolism (tissue and RBC breakdown), parenteral nutrition, and medications such as corticosteroids.

sodium and water retention:

The kidney cannot concentrate or dilute the urine normally in ESRD. Appropriate responses by the kidney to changes in the daily intake of water and electrolytes, therefore, do not occur. Some patients retain sodium and water, increasing the risk for edema, heart failure, and hypertension. Hypertension may also result from activation of the renin–angiotensin–aldosterone axis and the concomitant increased aldosterone secretion. Other patients have a tendency to lose salt and run the risk of developing hypotension and hypovolemia. Episodes of vomiting and diarrhea may produce sodium and water depletion, which worsens the uremic state.

Acidosis:

With advanced renal disease, metabolic acidosis occurs because the kidney cannot excrete increased loads of acid. Decreased acid secretion primarily results from inability of the kidney tubules to excrete ammonia (NH_3^-) and to reabsorb sodium bicarbonate (HCO_3^-). There is also decreased excretion of phosphates and other organic acids.

Anemia:

Anemia develops as a result of inadequate erythropoietin production, the shortened life span of RBCs, nutritional deficiencies, and the patient's tendency to bleed, particularly from the GI tract. Erythropoietin, a substance normally produced by the kidney, stimulates bone marrow to produce RBCs. In renal failure, erythropoietin production decreases and profound anemia results, producing fatigue, angina, and shortness of breath.

calcium and phosphorus imbalance:

Another major abnormality seen in chronic renal failure is a disorder in calcium and phosphorus metabolism. Serum calcium and phosphate levels have a reciprocal relationship in the body: as one rises, the other decreases. With decreased filtration through the glomerulus of the kidney, there is an increase in the serum phosphate level and a reciprocal or corresponding decrease in the serum calcium level. The decreased serum calcium level causes increased secretion of parathormone from the parathyroid glands. In renal failure, however, the body does not respond normally to the increased secretion of parathormone; as a result, calcium leaves the bone, often producing bone changes and bone disease. In addition, the active metabolite of vitamin D (1,25-dihydroxycholecalciferol) normally manufactured by the kidney decreases as renal failure progresses. Uremic bone disease, often called renal osteodystrophy, develops from the complex changes in calcium, phosphate, and parathormone balance

3.8 Management:

Goal: conservation of renal function as long as possible.

- Detection and treatment of reversible causes of renal failure (eg, bring diabetes under control; treat hypertension)
- Dietary regulation—low-protein diet supplemented with essential amino acids or their keto analogues to minimize uremic toxicity and to prevent wasting and malnutrition
- Treatment of associated conditions to improve renal dynamics
    - Anemia—erythropoiesis-stimulating agents (ESAs), such as epoetin alfa and darbepoetin
    - Acidosis—replacement of bicarbonate stores by infusion or oral administration of sodium bicarbonate
    - Hyperkalemia—restriction of dietary potassium; administration of cation exchange resin
    - Phosphate retention—decrease in dietary phosphorus (chicken, milk, legumes, carbonated beverages); administration of phosphate-binding agents because they bind phosphorus in the intestinal tract
- Maintenance dialysis or kidney transplantation when symptoms can no longer be controlled with conservative management

3.8.1 PHARMACOLOGIC THERAPY

Complications can be prevented or delayed by administering prescribed antihypertensives, erythropoietin (Epogen), iron supplements, phosphate-binding agents, and calcium supplements.

- **Antacids.** Hyperphosphatemia and hypocalcemia are treated with aluminum-based antacids that bind dietary phosphorus in the GI tract. However, concerns about the potential long-term toxicity of aluminum and the association of high aluminum levels with neurologic symptoms and osteomalacia have led some physicians to prescribe calcium carbonate in place of high doses of aluminum-based antacids. This medication also binds dietary phosphorus in the intestinal tract and permits the use of smaller doses of antacids. Both calcium carbonate and phosphorusbinding antacids must be administered with food to be effective. Magnesium-based antacids must be avoided to prevent magnesium toxicity.

- **Antihypertensive and Cardiovascular Agents.** Hypertension is managed by intravascular volume control and a variety of antihypertensive agents. Heart failure and pulmonary edema may also require treatment with fluid restriction, low-sodium diets, diuretic agents, inotropic agents such as digitalis or dobutamine, and dialysis. The metabolic acidosis of chronic renal failure usually produces no symptoms and requires no treatment; however, sodium bicarbonate supplements or dialysis may be needed to correct the acidosis if it causes symptoms.

- **Antiseizure Agents.** Neurologic abnormalities may occur, so the patient must be observed for early evidence of slight twitching, headache, delirium, or seizure activity. If seizures occur, the onset of the seizure is recorded along with the type, duration, and general effect on the patient. The physician is notified immediately. Intravenous diazepam (Valium) or phenytoin (Dilantin) is usually administered to control seizures. The side rails of the bed should be padded to protect the patient.

- **Erythropoietin.** Anemia associated with chronic renal failure is treated with recombinant human erythropoietin (Epogen). Anemic patients (hematocrit less than 30%) present with nonspecific symptoms, such as malaise, general fatigability, and decreased activity tolerance. Epogen therapy is initiated to achieve a hematocrit of 33% to 38%, which generally alleviates the symptoms of anemia. Epogen is administered either intravenously or subcutaneously three times a week. It may take 2 to 6 weeks for the hematocrit to rise; therefore, Epogen is not indicated for patients who need immediate correction of severe anemia. Adverse effects seen with Epogen therapy include hypertension (especially during early stages of treatment), increased clotting of vascular access sites, seizures, and depletion of body iron stores. The patient receiving Epogen may experience influenza-like symptoms with initiation of therapy; these tend to subside with repeated doses. Management involves adjustment of heparin to prevent clotting of the dialysis lines during hemodialysis treatments, frequent monitoring of hematocrit, and periodic assessment of serum iron and transferrin levels. Because adequate stores of iron are necessary for an adequate response to erythropoietin, supplementary iron may be prescribed. In addition, the patient's blood pressure and serum potassium level are monitored to detect hypertension and rising serum potassium levels, which may occur with therapy and the increasing RBC mass. The occurrence of hypertension requires initiation or adjustment of the patient's antihypertensive therapy. Hypertension that cannot be controlled is a contraindication to recombinant erythropoietin therapy. Patients who have received Epogen have reported decreased levels of fatigue, an increased feeling of well-being, better tolerance of dialysis, higher energy levels, and improved exercise tolerance. Additionally, this therapy has decreased the need for transfusion and its associated risks, including bloodborne infectious disease, antibody formation, and iron overload

3.8.2 NUTRITIONAL THERAPY

Dietary intervention is necessary with deterioration of renal function and includes careful regulation of protein intake, fluid intake to balance fluid losses, sodium intake to balance sodium losses, and some restriction of potassium. At the same time, adequate caloric intake and vitamin supplementation must be ensured. Protein is restricted because urea, uric acid, and organic acids—the breakdown products of dietary and tissue proteins—accumulate rapidly in the blood when there is impaired renal clearance. The allowed protein must be of high biologic value (dairy products, eggs, meats). High-biologic-value proteins are those that are complete proteins and supply the essential amino acids necessary for growth and cell repair. Usually, the fluid allowance is 500 to 600 mL more than the previous day's 24-hour urine output. Calories are supplied by carbohydrates

and fat to prevent wasting. Vitamin supplementation is necessary because a protein-restricted diet does not provide the necessary complement of vitamins. Additionally, the patient on dialysis may lose water-soluble vitamins from the blood during the dialysis treatment.

3.8.3 OTHER THERAPY: DIALYSIS

Hyperkalemia is usually prevented by ensuring adequate dialysis treatments with potassium removal and careful monitoring of all medications, both oral and intravenous, for their potassium content.
The patient is placed on a potassium-restricted diet. Occasionally, Kayexalate, a cation-exchange resin, administered orally, may be needed. The patient with increasing symptoms of chronic renal failure is referred to a dialysis and transplantation center early in the course of progressive renal disease. Dialysis is usually initiated when the patient cannot maintain a reasonable lifestyle with conservative treatment

3.8.4 Nursing management:

Nursing Assessment

- Obtain history of chronic disorders and underlying health status.
- Assess degree of renal impairment and involvement of other body systems by obtaining a review of systems and reviewing laboratory results.
- Perform thorough physical examination, including vital signs, cardiovascular, pulmonary, GI, neurologic, dermatologic, and musculoskeletal systems.
- Assess psychosocial response to disease process including availability of resources and support network.

Nursing Diagnoses

- Excess Fluid Volume related to disease process
- Imbalanced Nutrition: Less Than Body Requirements related to anorexia, nausea, vomiting, and restricted diet
- Impaired Skin Integrity related to uremic frost and changes in oil and sweat glands
- Constipation related to fluid restriction and ingestion of phosphate-binding agents
- Risk for Injury while ambulating related to potential fractures and muscle cramps due to calcium deficiency
- Ineffective Therapeutic Regimen Management related to restrictions imposed by CRF and its treatment

Nursing Interventions

Maintaining Fluid and Electrolyte Balance
Maintaining Adequate Nutritional Status
Maintaining Skin Integrity

- Keep skin clean while relieving itching and dryness.

    - Soap for sensitive skin, such as basis soap
    - Sodium bicarbonate added to bath water
    - Oatmeal baths
    - Bath oil added to bath water
- Apply ointments or creams for comfort and to relieve itching.
- Keep nails short and trimmed to prevent excoriation.
- Keep hair clean and moisturized.
- Administer antihistamines for relief of itching if indicated, but discourage patient from taking any OTC drugs without discussing with health care provider.

Preventing Constipation

- Be aware that phosphate binders cause constipation that cannot be managed with usual interventions.
- Encourage high-fiber diet, bearing in mind the potassium content of some fruits and vegetables.
    - Commercial fiber supplements (Fiberall, Fiber-Med) may be prescribed.
    - Use stool softeners as prescribed.
    - Avoid laxatives and cathartics that cause electrolyte toxicities (compounds containing magnesium or phosphorus).
    - Increase activity as tolerated.

Ensuring a Safe Level of Activity

- Monitor serum calcium and phosphate levels; watch for signs of hypocalcemia or hypercalcemia (see page 803).
- Inspect patient's gait, range of motion, and muscle strength.
- Administer analgesics, as ordered, and provide massage for severe muscle cramps.
- Monitor X-rays and bone scan results for fractures, bone demineralization, and joint deposits.
- Increase activity as tolerated—avoid immobilization because it increases bone demineralization.
- Administer medications as ordered:
    - Phosphate-binding medications, such as sevelamer (Renagel) or calcium carbonate (Os-Cal), with meals and snacks to lower serum phosphorus
    - Calcium supplements between meals to increase serum calcium
    - Vitamin D to increase absorption and utilization of calcium

Increasing Understanding of and Compliance with Treatment Regimen

- Prepare patient for dialysis or kidney transplantation.
- Offer hope tempered by reality.
- Assess patient's understanding of treatment regimen as well as concerns and fears.

- Explore alternatives that may reduce or eliminate adverse effects of treatment.
    - Adjust schedule so rest can be achieved after dialysis.
    - Offer smaller, more frequent meals to reduce nausea and facilitate taking medication.
- Encourage strengthening of social support system and coping mechanisms to lessen the impact of the stress of chronic kidney disease.
- Provide social work referral.
- Contract with patient for behavioral changes if noncompliant with therapy or control of underlying condition.
- Discuss option of supportive psychotherapy for depression.
- Promote decision making by patient.
- Refer patients and family members to renal support agencies.

Patient Education and Health Maintenance

- To promote adherence to the therapeutic program, teach the following:
    - Weigh self every morning to avoid fluid overload.
    - Drink limited amounts of fluids only when thirsty.
    - Measure allotted fluids, and save some for ice cubes; sucking on ice is thirst quenching.
    - Eat food before drinking fluids to alleviate dry mouth.
    - Use hard candy or chewing gum to moisten mouth.
- For further information and support, refer to the National Kidney Foundation, www.kidney.org.
- Encourage all people with the following risk factors to obtain screening for chronic kidney disease: elderly people, native Americans, Blacks, Latinos, diabetics, people with hypertension, those with autoimmune disease, and those with family history of kidney disease. More information on the National Kidney Foundation's Clinical Practice Guidelines for Chronic Kidney Disease can be obtained from www.kidney.org/professionals/kdoqi/index.cfm.

Evaluation: Expected Outcomes

- BP stable, no excessive weight gain
- Tolerates small feedings of low-protein, high-carbohydrate diet
- No skin excoriation; reports some relief of itching
- Passes small, firm stool daily
- Ambulates without falls
- Asks questions and reads education materials about dialysis

3.9 Complications:

Potential complications of chronic renal failure that concern the nurse and that necessitate a collaborative approach to care include the following:

- Hyperkalemia due to decreased excretion, metabolic acidosis, catabolism, and excessive intake (diet, medications, fluids)
- Pericarditis, pericardial effusion, and pericardial tamponade due to retention of uremic waste products and inadequate dialysis
- Hypertension due to sodium and water retention and malfunction of the renin–angiotensin–aldosterone system
- Anemia due to decreased erythropoietin production, decreased RBC life span, bleeding in the GI tract from irritating toxins, and blood loss during hemodialysis
- Bone disease and metastatic calcifications due to retention of phosphorus, low serum calcium levels, abnormal vitamin D metabolism, and elevated aluminum levels

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