ACUTE KIDNEY INJURY AND CHRONIC KIDNEY DISEASE

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OBJECTIVES

- Differentiates between acute and chronic kidney disease.
- Differentiates among the causes of pre-intra-post renal failure
- Describes the clinical course of reversible acute renal failure
- Explain the therapeutic and nursing management for a patient with olguric

- diuretic, and recovery phases of ARF
- Describe the systemic effects of CRF
- Explain the conservative management of CRF
- Explain the long term problems of CRF
- Discuss the psychological stressors of the patient with CRF and kidney transplant

ACUTE KIDNEY INJURY (ACUTE RENAL FAILURE)

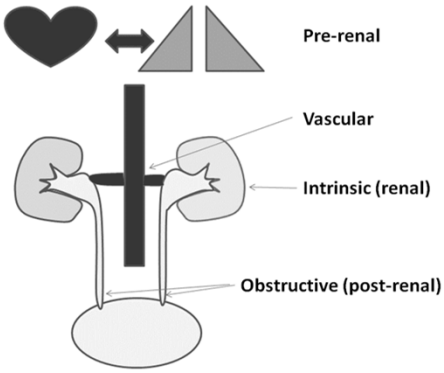
- Sudden or rapid reduction in kidney function that limits the kidney's ability to maintain the body's internal environment.
  ARF is potentially reversible, but mortality rate remains high.
- Two most common causes of renal failure are prolong ischemia (hypotension or hypovolemia) and nephrotoxic injury (exposure to nephrotoxic agents).

Pathophysiology

- Renal ischemia or nephrotoxic injury result in a disruption in the basement membrane and destroy the tubular epithelium. This decreases the GFR, causing an accumulation of nitrogenous waste products and a decrease in the urinary output.

Characteristics

- ***Azotemia***: Accumulation of nitrogenous waste products. (↑BUN & Creatinine)
- ***Uremia***: Renal function declines to the point that symptoms develop in multiple body systems.
- Urine enters the bloodstream instead of being eliminated by the body
- UOP is less than 400 ml/per day



PRERENAL FAILURE

- Due to factors external to the kidneys that reduce systemic blow flow to the kidneys which lead to decreased glomerular perfusion and filtration.
- Most common type of AKI 60-70% of all cases.

Etiologies

Decreased intravascular volume

- Hemorrhage
- Hypovolemia
- Vomiting
- Diarrhea
- Burns

Increased intravascular capacities

- Sepsis
- Vasodilation
- Anaphylaxis

Etiologies

Decreased cardiac output

- MI
- PE
- Cardiogenic shock
- CHF
- Arrhythmias
- Valvular disease

Decreased PVR / Increased intravascular capacities

- Anaphylaxis
- Neurologic injury
- Septic Shock
- HTN drugs

Etiologies

Hepatorenal Syndrome

- Liver failure

Decreased reno-vascular blood flow

- Embolism
- Renal vein thrombosis
- Renal artery thrombosis

INTRARENAL FAILURE

- Conditions that cause direct damage to the renal parenchyma, resulting in impaired function of the nephron.
- Usually due to prolonged ischemia, nephrotoxins, hemolysis of RBC, and myoglobin released from necrotic cells.

Etiologies

Nephrotoxic Injury

- Drugs
- Radiographic agents
- Hemolytic blood transfusion
- Crushing injuries
- Chemicals

Interstitial nephritis

- Allergic reaction
- NSAIDS
- ACE inhibitors

Etiologies

- Acute pyelonephritis
- Systemic Lupus erythematosus (SLE)
- Toxemia
- Acute glomerulonephritis
- **Infections**
  - Viral
  - Bacterial
  - Fungal
  - CMV

ACUTE TUBULAR NECROSIS {ATN}

- The most common cause of intrarenal acute kidney injury or acute renal failure.
- Usually caused by ischemia, sepsis nephrotoxins, or pigments.
- Ischemic and nephrotoxic acute tubular necrosis are responsible for 90% of intrarenal ARF

Pathology of ATN

Hypovolemia decreases renal blood flow and triggers the RAA system. This results in constriction, decreased blood flow, decrease GFR, tubular dysfunction, and oliguria.

Ischemia ↓ glomerular capillary permeability which reduces the GFR, ↓blood flow , and leads to tubular dysfunction.

Pathology of ATN

- Ischemia alters epithelia cells and decreases the glomerular capillary permeability. This decreases GFR.
- Tubules become damage, interstitial edema occurs, obstructing the tubules and increasing intratubular pressure.
- Glomerular filtrate leaks back into plasma through the membrane which decreases intratubular fluid flow.

POSTRENAL FAILURE

- Involves mechanical obstruction of the urinary outflow. As the flow of urine is obstructed, urine refluxes into the renal pelvis, impairing renal function.

Etiologies

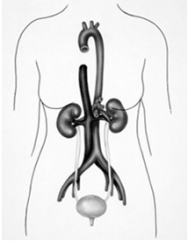
- Calculi formation
- BPH
- Trauma
- Prostate cancer/Bladder cancer
- Extrarenal tumors
- Strictures
- SCI
- Neuromuscular disorders

R.I.F.L.E

- RISK
- INJURY
- FAILURE
- LOSS
- End Stage Kidney Disease

CLINICAL COURSE

- Initiating Phase
- Oliguric Phase
- Diuretic Phase
- Recovery Phase



Oliguric Phase

- Oliguria: <400 ml urine /24 hours
- Occurs within 1-7 days of causative event
- 50% patients will not demonstrate oliguria, making the diagnosis difficult.
- Duration: 10-14 days. Can last months in some cases

Prerenal vs Intrarenal Oliguria

- It is important to distinguish pre/intra oliguria.
- **Pre renal**: Urine SG > 1.025
- Low urine sodium concentration
- **Intrarenal**: Normal or *Fixed* urine SG
- High sodium concentration
- RBC's, WBC cast cells

Diuretic Phase

- Gradual increase in UOP 1-3 L/day
- Nephrons are not fully functional
- The kidney has recovered their ability to excrete wastes, but not to concentrate urine.
- High urine volume caused by osmotic diuresis from high urea concentration.
- Duration: 1-3 weeks

Recovery Phase

- Begins when the GFR increases, allowing the BUN and creatinine levels to plateau and drop.
- Major improvement in the first 2 weeks.
- Renal function may take up to 12 months to stabilize.

Diagnostic Studies

- Labs: BUN, Creatinine, K+, Na+
- UA
- Renal Ultrasound
- Retrograde pyelogram
- CT
- MRI

MANIFESTATIONS

FUNCTIONS OF THE KIDNEY

A WET BED

A-Acid-Base Balance
W-Water Removal
E-Erythropoesis
T-Toxin Removal
B-Blood Pressure Control
E-Electrolyte Balance
D-Vitamin D Activation

MANIFESTATIONS OF AKI/ARF

- Fluid Retention
- Metabolic acidosis (HCO3)
- Sodium (Hyponatermia)
- Potassium (Hyperkalemia)
- Calcium/Phosphate
- Nitrogenous product accumulation
- Hematological (Anemia)
- Neurologic disorders

Management

- The primary goal in treatment of AKI is to maintain the body's normal state as possible while the kidney's are repairing themselves.
- Determine if there is adequate intravascular volume and cardiac output to ensure adequate perfusion.

Management

- Volume expanders may be administered along with diuretics to prevent fluid volume over load.
- Diuretics (Lasix)
- Loop diuretics (Etacynic Acid, Bumex)
- Osmotic diuretics (Manitol)

Management

- Strict I&O
- Electrolytes: Hyperkalemia
- Diet
- 30-35 kcal/kg
- Protein: 0.8 to 1.0 g/kg (0.6-2 g/kg/d)
- Fat 30-40% of total calories
- Sodium restriction 1-3 gm/day
- Potassium: 2-4 g/day (MD order 40mEq/day)
- Dialysis

GERONTOLOGIC CONSIDERATIONS

- The number of functioning nephrons decrease with age
- Comorbidities increase risk of developing AKI
- Aging kidney decrease ability to compensate for changes in fluid volume, solute load, and cardiac output.

QUESTION

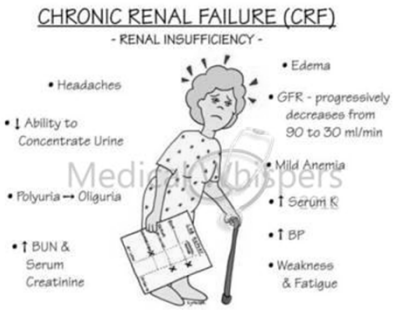
Which client has the highest risk for developing AKI?

a. A dialysis client who gets influenza
b. A teenager who had an appendectomy
c. A pregnant woman who has a fractured femur
d. A client with diabetes who has a heart catheterization

Question

A client with acute kidney injury has a serum potassium of 7.0 mEq/L. The priority for this client is to assess:

a. urine specific gravity
b. ECG results
c. mental status
d. blood pressure



CHRONIC KIDNEY DISEASE (Chronic Renal Failure)

- Progressive nonreversible destruction of both kidneys. Nonfunctional scar tissue replaces the destroyed nephrons.
- Up to 80% of the GFR may be lost with few overt changes in the functioning body
- Chronic Kidney Disease: GFR < 60 ml/min for > 3 months.

STAGES OF CKD

- Stage I: GFR ≥ 90
- Stage II: GFR 60-89 (mild)
- Stage III: GFR 30-59 (moderate)
- Stage IV: GFR 15-29 (severe)
- Stage V: GFR < 15 (kidney failure ESRD)

Etiologies

- Diabetes 43%
- HTN 23%
- Kidney disease 15%
- Others 19%

Clinical Manifestations

Uremia is a syndrome that incorporates all signs and symptoms seen in various systems throughout the body in chronic kidney disease.

Often occurs when the GFR is ≤ 10 mL/min

Urinary System

- Polyuria
- SG 1.010
- Anuria: UOP< 40 ml /24 hours

Metabolic Disturbances

- Waste product accumulation
- Increased BUN and creatinine
- Alter carbohydrate metabolism
- Elevated triglycerides

Electrolyte and Acid-Base Imbalances

- Hyperkalemia
- Hyponatermia
- Calcium and Phosphate
- Hypermanesium
- Metabolic acidosis

Hematologic System

- Anemia
- Bleeding tendencies
- Infection
- Increased incidence of cancer

Cardiovascular System

- HTN
- Accelerated Atherosclerosis
- MI
- Stroke
- CHF
- Cardiac dysrhythmias
- Uremic pericarditis

Respiratory System

- Kussmaul respiration
- Dyspnea
- Pulmonary edema
- Uremic pleuritis
- Pleural effusion
- Uremic Lung

Gastrointestinal System

- Gastritis
- Mucosal ulcerations
- Stomatitis
- Gastroparesis
- GI Bleed
- Diarrhea/constipation

Neurologic System

- CNS depression
- Seizures
- Hypertensive encephalopathy
- Dialysis encephalopathy
- Peripheral neuropathy

Musculoskeletal System

- CKD-mineral and bone disorder (CKD-MBD) develops as systemic disorder of mineral and bone metabolism.
- As kidney function deteriorates less vitamin D is converted to the active form.
- ↓ active vitamin D results in less calcium absorption from the intestines

- Hypocalcemia→ PTH →stimulates bone demineralization→ releases calcium from the bones. Phosphate is released as well→ this leads to ↑phosphate levels.

Hyperphosphatemia results from ↓ phosphate excretion by kidneys →decreased calcium levels

SKELETAL COMPLICATIONS

- Osteomalacia
- Osteitis fibrosa
- Vascular wall calcification
- Intracardiac calcification

Integumentary System

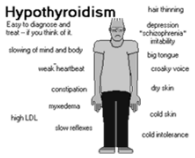
- Yellow-gray discoloration
- Pruritus
- Uremic frost
- Petechiae and ecchymoses
- Dry brittle hair and nails

Reproductive System

- Infertility
- Decreased libido
- Decrease estrogen and progesterone levels (anovulation, amenorrhea)
- Sexual dysfunction
- Decreased testosterone levels, low sperm counts

Endocrine System

- Hypothyroidism



Psychologic Changes

- Withdrawal
- Depression
- Decrease ability to concentrate
- Anxiety

DIAGNOSTIC TEST

- Urine dip for protenuria
- Microalbuminuria
- Urine albumin to creatinine ratio (300: 1)
- Urinalysis (RBC, WBC, protein, cast,glu)
- GRF

Drug Therapy for Hyperkalemia

- Kayexalate/Sorbitol
- Glucose and Insulin
- 10% Calcium gluconate

Drug Therapy for Hypertension

- Sodium and Fluid restriction
- Diuretics (Lasix)
- Beta Adrenergic Blockers
- Calcium Cannel Blockers
- ACE inhibitors
- Angiotensin receptor blockers (ARBs)

Drug Therapy for CKD-MBD

- Restrict phosphate to 1000mg/per day
- **Calcium-based phosphate binders**
  - Calcium carbonate (TUMS)
  - Calcium acetate (PhosLo)
- **Non-calcium based phosphate binders**
  - Renagel (sevelamer)
  - Fosrenol (lanthanum carbonate)

Drug Therapy CKD-MBD

- **Vitamin D Replacement**
  - **cholecaliferol**

Activated form of vitamin D used with hyperparathyroidism

- Rocaltrol (oral form)
- Calcitrol (IV)

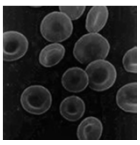
Drug Therapy for Secondary Hyperparathyroidism

Calcimimetics (Sensipar)

- Mimics calcium and increase sensitivity of the calcium receptors in the parathyroid glands.
- The glands detect calcium at a lower serum level and decrease PTH secretion

Drugs Therapy for Anemia

- Erythropoietin
  Epogen, Procrit
  Aranesp



- Iron
- Blood Transfusion

Nutritional Therapy

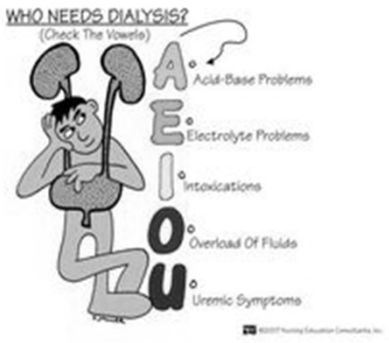
- Protein restriction 1.2 g/kg
- 40 gram/day (M.D order)
- Low phosphate diet

Dialysis Patients

- Increase protein to 1.2-1.3 g/kg/day
- Carbohydrates 35kcal/kg/day (M.D order 100 g/day)
- During PD protein intake may be higher to compensate for losses

Restrictions

- Water
- Sodium 2-4 g/day
- Potassium 2-4 g /day
- Phosphate 1000mg/day



RENAL REPLACEMENT THERAPY (RRT)

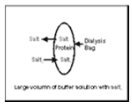
- Volume overload resulting in compromised cardiac and pulmonary status
- Elevated potassium Levels
- Metabolic acidosis (<HCO3 15 mEq)
- BUN level > 120 mg/dL
- Significant change in mental status
- Pericarditis, pericardial effusion, cardiac tamponade

DIALYSIS

- Dialysis begins when the uremic state can no longer be managed conservatively.
- Dialysis is the movement of fluid and molecules across a semi-permeable membrane from one compartment to another.

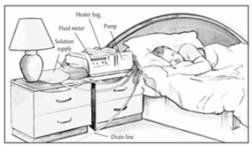
Principles of Dialysis

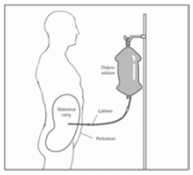
- Diffusion
- Osmosis
- Ultrafiltration



PERITONEAL DIALYSIS

- The large surface area of the peritoneum makes it a good semi-permeable membrane for performing clinical dialysis.
- Access for peritoneal dialysis is obtained by inserting a catheter through the anterior abdominal wall. The [illegible] e catheter rest in the peritonea





Dialysis Solutions

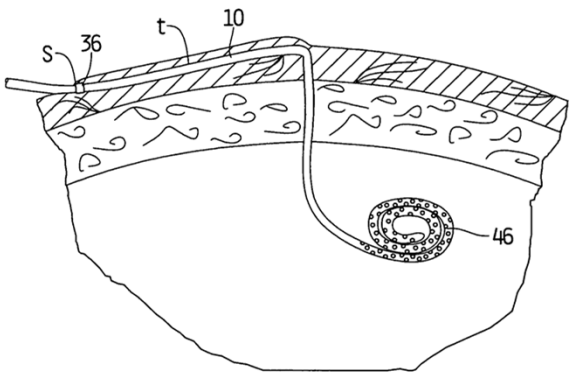
- 1-2 liter bags
- Glucose concentration
- 1.5%
- 2.5%
- 4.25%
- The electrolyte composition is similar to that of plasma

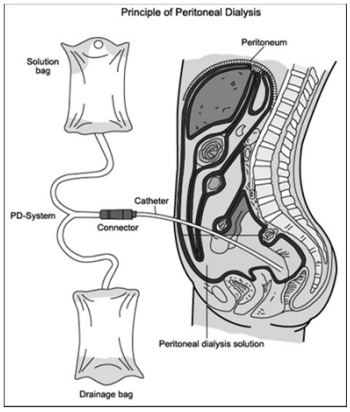
Dialysis Cycles

- Inflow
- Dwell
- Outflow

Peritoneal Dialysis Systems

- Automated
- Continuous Ambulatory





Contraindications for Peritoneal Dialysis

- Multiple abdominal surgeries
- Hernias
- Morbid obese
- Vertebral Disease
- Obstructive Pulmonary disease

Complications

- Infection {Wound, Peritonitis}
- Abdominal pain
- Hernias
- Outflow Problems
- Bleeding
- Pulmonary Complications
- Low Back Pain
- Encapsulating sclerosing Peritonitis
- Carbohydrate and lipid abnormalities
- Protein loss

HEMODIALYSIS

- **Vascular access**
- A very rapid blood flow is required, and access to a large blood vessel is essential
- *Arteriovenous fistulas (AVF)*
- *Arteriovenous Grafts (AVG)*
- *Temporary/Semipermanent cath*
- *Subcutaneous ports*
- *Shunts*

Temporary Vascular Access

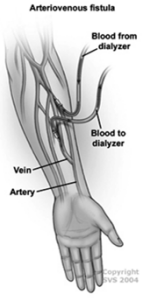
- Percutaneous cannulation of the internal jugular or femoral vein. A catheter with a double lumen. One lumen is used for blood removal and the other is used for blood return

Arteriovenous Fistulas and Grafts

- **Arteriovenous Fistula**: Anastomosis between an artery and a vein. The fistula provides for arterial blood flow through vein.
- **Arteriovenous Grafts**: Form a bridge between the arterial and venous blood supplies. Must use a synthetic graft

Shunts

- Shunts consist of a U-shaped Silastic tube divided at the midpoint, and each of the two ends are placed in an artery and a vein
- Rarely use today except for CRRT
- Continuous Renal Replacement Therapy


Arteriovenous fistula
Blood from dialyzer
Blood to dialyzer
Vein
Artery
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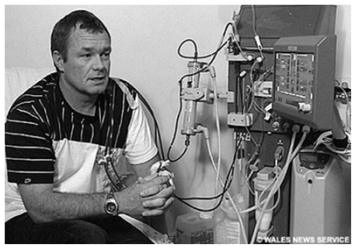
Port-a-caths


WALES NEWS SERVICE

Complications of Hemodialysis

- Infection
- Hypotension
- Blood loss
- Hepatitis
- Sepsis
- Muscle cramps
- Disequilibrium Syndrome

Continuous Renal Replacement Therapy (CRRT)

- The principles of CRRT is to dialyze patients in a more physiologic way over 24-hours, just like the kidneys
- Continuous over 24-hours
- Much slower blood flow rate compare to HD
- Requires cannulation of an artery and vein or cannulation of two veins
- Long as 30-40 days

- Use of a blood pump to propel the blood through the circuit
- Highly permeable hollow fiber hemofilter removes plasma water and nonprotein solutes (ultrafiltrates).
- Under the influence of the hydrostatic and osmotic pressure ultrafiltrates move into the extracapillary space and drain through the ultrafiltrate port into a collection bag

QUESTION

A client with CRF is hypocalcemia and hyperphosphatemia which symptoms should the nurse be assess for? (Select all that apply)

a. Trousseau's sign
b. Cardiac arrhythmias
c. Decreased clotting times
d. Fractures
e. Drowsiness and lethargy

QUESTION

The nurse notes during the out flow cycle that only 500 ml has drained. Which intervention should be done first?

a. Change the client position
b. Check the catheter for kinks
c. Call the physician
d. Clamp the catheter until the next exchange cycle.

RENAL TRANSPLANT

Over 100,400 patients are awaiting a kidney transplant, but there's only approximately only 17,800 donors available

Contraindications

- Disseminated malignancies
- Untreated or refractory cardiac diseases
- Extensive vascular diseases
- Chronic infections
- Unresolved psychological disorders
- ***The presents of Hepatitis is not a contraindication***

Types of Donors

- **Cadaver**
  Kidney are removed and preserved up to 72 hours prior to transplant.

- **Live Donors**
  1. Blood relative
  2. Emotionally related
  3. Unknown donor

Histocompatibility Studies

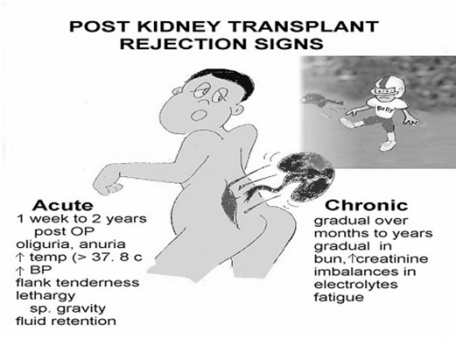
- Identify the HLA antigens for both the donor and the recipient.
- Antigen matching of 5-6 antigens have been found to have the better clinical outcome.
- Cross match studies identify if the recipient has cytotoxic antibodies to the donor and is an *absolute* contraindication for transplant

Immunosuppressive Therapy

The goal of immunosuppressive therapy is to adequately suppress the immune response to prevent rejection of the transplanted organ while maintaining sufficient immunity to prevent overwhelming infection.

Complications of Transplantation

- ❑Hyperacute rejection
- ❑Acute rejection
- ❑Chronic rejection
- ❑Infection
- ❑Cardiovascular disease
- ❑Malignancies
- ❑Corticosteroid related complications

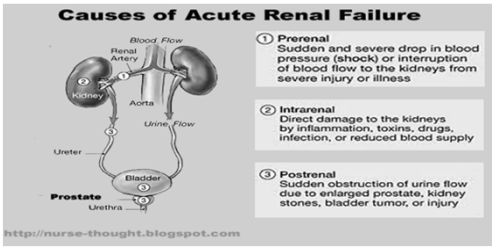


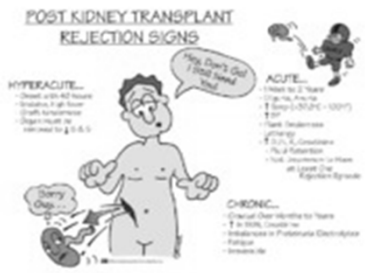
QUESTION

Which signs and symptoms have the highest priority for a client that received a renal transplant?

a. stomach discomfort or dyspepsia
b. dizziness and bleeding from the incision
c. fever, flushed feelings, or lethargy
d. signs of depression or decrease appetite

REVIEW




POST KIDNEY TRANSPLANT
REJECTION SIGNS
HYPERACUTE...
ACUTE...
CHRONIC...