AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
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Life-Threatening Emergencies in the Renal Failure and Dialysis Patient

Patients with renal failure and those undergoing dialysis may present to the emergency department with a variety of life-threatening conditions. The lecturer will review the major complications associated with these disorders and outline their recognition and management. Topics to be discussed include electrolyte abnormalities, pulmonary edema, pericarditis, infections of the dialysis access site, and uremic encephalopathy.

- Identify life-threatening presentations in the patient with renal failure.
- Explain the management of each presentation in the emergency department.

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FACULTY

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Life Threatening Emergencies in the Renal Failure and Dialysis Patient

John F. O'Brien, M.D.

Renal Failure and Dialysis: History

1943 Kolff designed artificial kidney
1946 Peritoneal dialysis first used
1960 Quinton/Scribner developed external AV shunt
1966 Brescia/Cimino developed internal AV fistula
1968 Tenckhoff peritoneal catheter developed
1973 ESRD program of Medicare begun
1976 Continuous ambulatory peritoneal dialysis developed
1990 Over 100,000 hemodialysis patients in US (HCFA)
1998 Over 250,000 ESRD patients in the US (Medical costs: $45,000 per patient per year)

Major Causes of Chronic Renal Failure*

- Diabetes mellitus (35%)
- Hypertension (30%; 40% in African Americans)
- Glomerulopathies
- Tubulointerstitial diseases
- Hereditary diseases
- Renal vascular diseases
- Obstructive uropathy

* Up to 38% of hemodialysis patients HIV positive in many inner city dialysis centers

Progression to Renal Failure

- May be acute or slow progression
- Fall in glomerular filtration rate (GFR) below 25 ml/min characterized by further progressive deterioration of renal function
- Deterioration continues despite resolution of primary problem
  - ♦ New pathogenic mechanisms
  - ♦ Progression occurs at constant rate

Problems Associated with Renal Insufficiency

- Inability to excrete excess water
- Inability to excrete toxic metabolites
- Inability to control electrolyte levels
- Failure of hematopoiesis
- Failure of vitamin D activation

Mortality Related to Onset of End-Stage Renal Disease (ESRD)

- 5 year survival is only 18-38%; mortality rate: 25.4%/year in 1988; 22.3%/year in 1996
- Diabetics have poorest prognosis, while those with glomerulonephritis have best relative prognosis
- Average cost of dialysis patient is $45,000/year (93% on Medicare)
- Co-morbid disease processes complicate management of dialysis patients greatly
- Number of ESRD patients growing at 7-10%/year
- Presently 71% of ESRD patients receive dialysis (83% hemodialysis, 17% peritoneal dialysis)
- Malnutrition present in up to 50% of patients with ESRD, and is independently associated with increased morbidity and mortality

Causes of Death in End-Stage Renal Disease Patients*

Myocardial infarction**	14.5%
Other cardiac cause	29.9%
Septicemia	10.6%
Withdrawal from dialysis	9.9%

Cerebrovascular disease	5.6%
Malignancy	3.4%
Pulmonary infection	2.4%
Unknown cause	7.6%
Other known cause	16.1%

USRDS 1994 Annual Data Report

*Over 50% of deaths in dialysis patients are cardiovascular related

**Poorer long-term survival present in dialysis patients after acute myocardial infarction

Therapy for Renal Failure

- Dietary:
  - Modest protein and fluid restriction
  - Restrict K intake
  - Phospate binders
  - Vitamin D and Ca supplements
- Hemodialysis
- Peritoneal dialysis

Indications for Initiating Dialysis

- Uremia
  - Blood urea nitrogen (BUN) >125 mg/dl common decision point
  - Clinical manifestations more important: nausea, vomiting, anorexia, fatigue, altered mental status
- Severe hyperkalemia
- Volume overload (unresponsive to diuretics)
- Severe acidosis
- Pericarditis (uremic)

Hemodialysis

- Employs diffusion across semipermeable membrane
  - Remove unwanted substances
  - Add desirable components
- Process similar to glomerular filtration
- Three components of hemodialysis equipment
  1. Blood delivery system
  2. Composition and delivery system of dialysate
  3. Dialyzer

Blood Delivery System in Hemodialysis

- Access options
  - Percutaneous venous catheters (Clotting, low flow rate and infection limit utility)
  - Arteriovenous fistula
    - External arteriovenous fistula
    - Endogenous fistula
    - Prosthetic (Graft) fistula
- Blood pumped to dialyzer by roller pump
- Appropriate monitors
  - Measure flow and pressure
  - Recognize air leak (system shutdown)
- Blood flow approximately 300-450 mL/min

Dialysate Composition and Delivery in Hemodialysis

- Dialysate composition similar to plasma water
  - K varied most often
  - Na, Ca, Mg, Cl, acetate or HCO_3 variable
- Dialysate delivery

- Storage tank
- Proportioning system (Manufactures dialysate on-line)

Water Contamination in Dialysate

- Reverse osmosis device prevents most contamination
- Main contaminants:
  - Bacteria
  - Chloramine (antiseptic)
  - Aluminum
  - Fluoride
- Daily monitoring for bacteria and chloramine levels recommended

Dialyzer in Hemodialysis

- Dialyzer
  - Hollow fiber (Capillary) - most common type
    - Membrane material spun into thousands of fine capillaries packed into bundles
    - Blood flows through capillaries with dialysate circulated outside
    - Countercurrent dialysate flow at about 500 ml/min
  - Most common membrane material: Cellulose and synthetic noncellulose membranes
  - Reduced mortality/morbidity with modified cellulose or synthetic membranes (certain centers)
  - In USA, over 55% of outpatient dialysis done with synthetic noncellulose membranes (which are expensive and reused to cover costs)
- Ultrafiltration
  - Negative hydrostatic pressure on the dialysate side manipulated to achieve desired fluid removal
  - Dialysis membranes have different ultrafiltration coefficients (fluid removed/mmHg/min)

Time Requirements for Hemodialysis

- Most patients require 9-12 hours of dialysis/week broken into several sessions
- Dialysis time depends on:
  - Body size
  - Residual renal function
  - Dietary intake
  - Complicating illnesses
  - Degree of catabolism/anabolism
- Ultrafiltration followed by dialysis:
  - Faster, more efficient fluid removal
  - Less hypotension, muscle cramps, nausea and vomiting
- Morbidity/mortality reduced by adequate dialysis efficiency, and correlates positively with long duration of dialysis sessions. High efficiency and high flux dialyzers further reduce mortality in some studies

Dialysis Variables in Hemodialysis

- Duration
- Frequency of treatments
- Size/type of dialyzer
- Dialysate composition
- Blood flow rate
- Dialysate flow rate and hydrostatic pressure
- Membrane type and surface area (Determinants of clearance, ultrafiltration)

Electrolyte and Solute Abnormalities in Dialysis Patients:
Hyperkalemia

- Causes:
  - Missed/ineffective dialysis
  - Excessive dietary K intake
  - Severe acidosis

- Acute infection with marked catabolism
- Rhabdomyolysis
- Acute hemolysis
- Drug effects

- Diagnosis
  - Suspect in any dialysis patient with weakness, dysrhythmia or hypotension
  - Electrocardiogram remains best guide to determining need for immediate therapy:
    - Tall, narrow-based T waves (Best seen II, III, V_2, V_3)
    - PR interval prolongation
    - ST segment changes (depression)
    - QRS widening
    - Atrial standstill
    - Sine wave QRS complex
    - Ventricular fibrillation or asystole
  - ECG may be normal even with high K
    - Rate of K increase most important factor
    - Chronic renal failure patients relatively tolerant of high K
  - Serum K measurement
    - Can be run rapidly: "code labs"
    - Beware delays in reporting (recheck)
- Emergency Treatment
  - Indication: K >6.5 mEq/L, ECG changes
  - Three main approaches to therapy:
    - Antagonize K effects on the heart
      Ca chloride (1.4 mEq/ml) or gluconate (0.4 mEq/ml)
      - Dose: 10 cc slow IV
      - Acts in 2-3 minutes, lasts < 1 hour (temporizing)
      - Caution: Can potentiate digitalis cardiotoxicity
    - Drive K into cells
      Na bicarbonate
      - Dose: 1-2 50 cc ampules (50-100 mEq) IV
      - Caution: May cause volume overload

      Glucose/insulin
      - Dose: D_{50} 1-2 ampules (50-100 cc); Regular insulin 10 units
      - Caution: Hypoglycemia

      Albuterol nebulized treatment
      - Dose: 1 cc (5 mg) in saline inhaled (Doses of 10-20 mg used in emergency)
      - Effective even if no prompt IV access

      Hyperventilation if intubated
    - Remove K from body
      Hemodialysis (rapid)
      Peritoneal dialysis (slower)
      Kayexylate
      - Dose: 25-50 gm in 70% sorbitol
      - PO more effective than enema
      - Exchanges 2 Na for 1 K
      - Caution: Volume overload

      Loop diuretic (if urine produced)

Hypokalemia

- K levels immediately at end of hemodialysis session 30% less than 5 hour steady state level
- True hypokalemia usually due to:
  - Alkalosis during hemodialysis
  - Prolonged poor intake
  - Recurrent vomiting and/or diarrhea

- Hypokalemia during dialysis may lead to arrhythmias (also increases digoxin toxicity)
- Dialysate K concentration of 1-3 mEq/L used for most (May be varied from 0-4 mEq/L)
- Optimal K elimination by hemodialysis accomplished by short daily treatments (Maximum K that can be removed in a single session is 70-90mEq, because K shifts slowly from intracellular to extracellular speed)

Sodium

- High dialysate Na concentration causes:
  - Hypertension
  - Exaggerated thirst
  - Excessive weight gains
- Hyponatremic dialysate causes:
  - Intradialysis hypotension
  - Muscle cramps
  - Headache
  - Nausea and vomiting
  - Provocative for dialysis disequilibrium
- Most patients: dialysate Na 140-145 mEq/L
- Abnormal sodium levels in hemodialysis patients due to abnormal water consumption or dialysate composition error
- Sodium modeling proportioning system - gradually adjusts dialysate and thus serum sodium levels throughout the dialysis process (from Na of 150-160 mEq/L early to normal levels late)

Glucose

- Dialysates are glucose free, normoglycemic (0.20-0.25% dextrose), or mildly hyperglycemic (>0.25%)
- Glucose free dialysate
  - Net loss of 30 gm glucose/treatment
  - Stimulates gluconeogenesis, ketogenesis
  - Increases loss of amino acids
  - Enhances K clearance
- Most dialysates normoglycemic

Calcium

- End-stage renal disease naturally presents with:
  - Hypocalcemia
  - Hyperphosphatemia
  - Hypovitaminosis D
  - Hyperparathyroidism (secondary)
- Only ~40% of Ca protein bound in dialysis patients
- Dialysate Ca concentration 2.5-3.0 mEq/L
- Ca salts used as dietary phosphate binders (Have replaced aluminum containing antacids)
- Hypercalcemia
  - Causes:
    - ★ Excessive synthetic vitamin D
    - ★ Excessive calcium
    - ★ Malignancies
    - ★ Secondary hyperparathyroidism
  - Symptoms:
    - ★ Nausea, vomiting, constipation
    - ★ Altered mental status, weakness
    - ★ Seizures, coma
- Hypocalcemia
  - Causes:
    - ★ Phosphate retention
    - ★ Decreased active vitamin D
    - ★ Acute alkalosis (bicarbonate) may precipitate

- Symptoms:
  - Tetany, seizures
  - Dysrhythmias

Phosphate
- Retention occurs in renal failure
- Not well removed by dialysis
- Treatment of hyperphosphatemia
  - Dietary restriction
  - GI binding agents (Ca carbonate or acetate)
  - Vitamin D analogues (calcitrol)

Hypermagnesemia
- Almost always, body can excrete huge magnesium loads with normal renal function
- Hypermagnesemia requires external Mg load with renal insufficiency
  - Antacids
  - Laxatives
  - Phosphate binders
- Only 1% of total body Mg in extracelluar stores
- Symptoms:
  - >4 mEq/dl: Hyporeflexia, flaccidity, vasodilation
  - >10 mEq/dl: Bradyarrhythmias, asystole, depressed cardiac conduction
- Mg in dialysate 0.5-1.0 mEq/L
- Treatment
  - Calcium chloride or gluconate will temporarily antagonize membrane effects
  - Dialysis allows rapid removal
  - Dietary modification to prevent recurrence

Acidosis
- Due to failure of excretion of metabolic acids
  - 50-100 mEq/day excreted normally
  - Increased load:
    - Ketoacidosis
    - Lactic acidosis (eg. hypoperfusion, sepsis)
    - Drug toxicity (eg. ethylene glycol toxicity)
- Dialysate buffers
  - Bicarbonate
    - Used in >90% of hemodialysis centers
    - Solution subject to bacterial contamination
    - Mixing error risks (Usually HCO_3 30-35 mEq/L)
- Acetate
  - Used in <10% of dialysis centers
  - Very bacteriostatic
  - Metabolized by Krebs cycle to HCO_3
  - Influx in hemodialysis may exceed capacity to metabolize - Hyperacetatemia
    - Nausea and vomiting
    - Headache
    - Peripheral vasodilation (hemodynamic instability)
    - Decreased myocardial contractility (hemodynamic instability)
    - Metabolic acidosis (hemodynamic instability)
    - Arterial hypoxemia (hemodynamic instability)
  - Problems occur especially with high surface area, high-efficiency dialyzers

Acute Hemodialysis Patient Complications
- Hypotension

- Infection
- Uremic pericarditis and pericardial tamponade
- Muscle cramps
- Anemia
- Platelet and hemostatic abnormalities
- Arrhythmias
- Hypersensitivity reactions (Anaphylaxis)
- Pyrogen reactions
- Hypoxemia
- Hemolysis
- Volume overload (Acute pulmonary edema)
- Dialysis dysequilibrium syndrome
- Technical mishaps (e.g. Air embolus)
- Angioaccess dysfunction

Intradialysis Hypotension in Hemodialysis

- Occurs in 10-50% of treatments
- Most frequent complication of dialysis
- Most commonly due to excessive rate or amount of ultrafiltration
- Other factors in intradialysis hypotension
    - Left ventricular dysfunction/ischemia/arrhythmias
    - Pericardial tamponade
    - Air embolism
    - Autonomic dysfunction (Reflex sympathetic inhibition)
    - Inappropriate vasodilation (eating, sepsis, warm dialysate)
    - Medications (antihypertensives, narcotics, anxiolytics, etc.)
    - Bleeding
    - Acetate toxicity (Vasodilation and cardiac toxicity)
    - Electrolyte disorders (K, Ca, Mg)
    - Hypoxemia
    - Hypothermia
    - Dialysis membrane hypersensitivity
    - Vomiting/diarrhea/decreased oral intake
    - Decrease in plasma osmolality

Evaluation and Treatment of Intradialysis and Post-Dialysis Hypotension

- Volume challenge with repetitive 250-500 cc NS IV
- If no response, consider especially:
    - Hyperkalemia
    - Pericardial tamponade
    - Occult bleeding
    - Infection

Infectious Complications in Dialysis Patients

- Infection second leading cause of death in dialysis patients (up to 20%)
- Procedural antibiotic prophylaxis recommended (dental, GI, GU, etc.)
- Abnormalities in immunity:
    - Depressed T and B lymphocyte function
    - Impaired neutrophil function (chemotaxis, phagocytosis, etc)
- Frequent skin breaches during vascular access common port of infection entry
- Vascular access infection often subtle
- Urinary tract infection, pneumonia also common
- Tuberculosis 10 times more frequent in hemodialysis patients

Approach to Febrile Hemodialysis Patients

- Antipyretics
- Careful history, PE to evaluate source
- Laboratory studies
  - Chest radiograph
  - Urinalysis and culture (if urine produced)
  - Blood culture x 2-3
    - 10 cc blood volumes improve yield
    - One from each catheter port
  - CBC?
- Aggressive antibiotic coverage
  - Access infection most common source
    - Staph aureus, followed by Staph epidermidis and gram negative organisms most common
    - Vancomycin (1 gm IV) + an aminoglycoside (2.5 mg/kg) reasonable initial regimen
  - Alternate antibiotic regimen if other source
- Possible catheter removal

Uremic Pericarditis and Pericardial Tamponade:

Uremic Pericarditis

- Pathophysiology unclear (Up to 20% of dialysis patients develop)
- Consider other etiologies: viral, idiopathic, etc.
- Pericardial friction rub present on initial presentation in 90%
- Uremic pericarditis tends to occur during two periods:
  - Just before dialysis required (Does not correlate with level of catabolic metabolites)
  - First few months of dialysis therapy
- Two-thirds of uremic pericarditis resolves with dialysis intensification. About one-third requires operative drainage of the pericardium
- May present as cardiac tamponade with volume depletion of dialysis

Pericardial (Cardiac) Tamponade

- Exists if buildup of pericardial fluid critically reduces diastolic cardiac filling
- Consider other etiologies: viral, idiopathic, malignancy, etc.
- Major complaint is dyspnea due to progressive development of interstitial fluid in the lungs
- Diagnosis
  - Physical exam
    - Tachycardia and tachypnea with signs of low cardiac output suggestive
    - Pulse paradoxus (Inspiratory decrease in systolic BP > 10 mmHg)
    - Jugular venous distension
  - Electrocardiogram
    - Reduced QRS voltages suggestive of large effusion
    - Beat to beat variability of cardiac voltages due to heart swinging inside large pool of pericardial fluid (electrical alternans) suggestive
  - Echocardiography
    - RA + RV diastolic collapse
    - Inspiratory bulge of intraventricular septum into LV
    - Lack of pericardial effusion excludes diagnosis
  - Pulmonary artery or cardiac catheterization (equalization of diastolic intracardiac pressures in all chambers)
- Treatment
  - Aggressive volume support may temporize hemodynamic compromise
  - Pericardial drainage techniques
    - Percutaneous pericardiocentesis (May be both diagnostic and therapeutic)
      Risk of complications as high as 5% if no fluoroscopic or echocardiographic guidance
      Subxiphoid approach most effective
    - Pericardial window (Subxiphoid percardiotomy)
    - Partial pericardiectomy

Muscle Cramps

- At least 20% report occurrence during dialysis
- Typically involve legs, feet, hands
- Occur during later half of and post-dialysis
- Usually self-limited in 5-10 minutes
- Pathogenesis: Intradialysis skeletal muscle ischemia
- Occur most frequently with excessive ultrafiltration
- Prevention:
  - Avoidance of aggressive ultrafiltration
  - Dialysis with hyperosmolar or HCO_3 buffered dialysate
  - Quinine (260-325 mg) - unproven
  - Vitamin E (400 IU) - unproven
- Treatment: Hyperosmolar agents
  - Dextrose 50% (50 ml)
  - Mannitol 25% (100 ml)
  - Salt solution 23.5% (10ml)

Anemia

- Causes
  - Diminished erythropoietin production due to decreased renal mass
  - Increased blood loss
  - Shortened RBC survival (1/2-2/3 of normal)
  - Iron/folate deficiency
- Treatment
  - Transfusion if emergency
  - Recombinant human erythropoietin (Given 1-3 times/wk at end of dialysis; Goal Hct 30-35. Erythropoietin increases graft patency with no increased incidence of thrombosis or thrombophlebitis)
  - Folate and iron (as indicated) supplements

Platelet and Hemostatic Abnormalities

- Serious abnormality in all dialysis patients
- Platelet dysfunction
  - Decreased binding of von Willebrand factor to receptor
  - Increased endothelial generation of nitric oxide
  - Central platelet vascular streaming in anemia
- Heparin anticoagulation
  - Probably major cause of bleeding in dialysis patients
  - May cause heparin-induced thrombocytopenia
- Bleeding prevention
  - Regional anticoagulation or heparin-free hemodialysis
  - Prevent/treat anemia (transfusion, erythropoietin)
  - Adequate dialysis
- Treatment:
  - Gentle local pressure if external bleeding
  - If heparin is problem:
    - ★ PTT prolonged
    - ★ Give 1 mg protamine sulfate to neutralize 100 units of heparin (Empiric 20 mg protamine sulfate dose reasonable if prolonged PTT and unknown heparin dose)
  - If platelet dysfunction:
    - ★ Bleeding time prolonged
    - ★ Cryoprecipitate
      Rich in von Willebrand factor
      10 units q 12-24^o
      Onset 30 min, lasts up to 36^o
    - ★ Desamino-8-D-arginine vasopressin (DDAVP)

Induces endothelial release of factor VIII von Willebrand multimers
Give IV (0.3 mcg/kg) or intranasally 3 mcg/kg)
Maximal effect at 2^{o}, lasts up to 8^{o}

- ★ Conjugate estrogens
  Premarin (25 mg/day for 7 days)
  Initial effect at 6^{o}, persists up to 21 days

Arrhythmias

- Common
- High concomitant incidence of:
  - Hypertensive, ischemic and hypertrophic cardiomyopathy
  - Conduction system disease
  - Coronary artery disease
  - Pericardial disease
  - Medication toxicity (eg. digitalis)
- Provocative dialysis factors:
  - Reduction in plasma K concentration (most important)
  - Transient hypercalcemia
  - Transient acidosis (acetate-buffer) and alkalosis (HCO_3 buffer)
  - Hypotension/hypoperfusion
  - Hypoxemia
  - Direct acetate cardiotoxicity
- Treatment
  - Address primary arrhythmia
  - Evaluate and correct electrolytes, especially K, Ca, Mg
  - Optimize dialysis regimen to prevent hypoxia, hypoperfusion, acid/base disorder or electrolyte imbalance)

Hypersensitivity Reactions

- More common in hemodialysis patients
- Etiology
  - Ethylene oxide sensitization
    - ★ Sterilizing agent in dialyzers
    - ★ Most common hypersensitivity cause in hemodialysis patients
    - ★ Causes allergic rxn in first 10-30 minutes
    - ★ Rare with thorough dialyzer rinsing
  - Angiotensin converting enzyme (ACE) inhibitor use and polyacrylnitrile dialyzer membrane exposure can precipititate
  - First use syndrome (cellulosic dialyzer)
  - Toxin exposure (eg. formaldehyde)
- Reaction types:
  - Urticaria/angioedema
  - Wheezing/respiratory collapse
  - Hypotension/hypertension
  - Fever and chills
  - Back/abdominal pain
- Treatment:
  - Epinephrine
  - Antihistamines
  - Corticosteroids
  - Saline infusion

Pyrogen Reactions

- Intradialysis or post-dialysis febrile event 2° to bacterial or bacterial product exposure w/o infection
- Basically, a historical diagnosis of exclusion
- Presentation: Fever, chills, rigors, myalgias, hypotension

- Incidence < 1 per 1000 dialysis treatments
- Etiology:
  - Contaminated dialysate (esp. HCO_3 solution)
  - Use and reuse of high-flux dialyzers
- Treatment
  - Antipyretics
  - Antibiotics after appropriate cultures until bacterial infection excluded

Hypoxemia
- Prevalence: up to 90% of dialysis treatments
- Absolute PO_2 decline 5-35 mmHg (mean 15 mmHg)
- May contribute to other dialysis complications
- Etiology:
  - Membrane effects (complement activation, leukocyte microemboli)
  - Dialysate effects (predominantly acetate)
- Oxygen saturation monitoring during dialysis important
- Treatment
  - Avoidance measures
  - Use HCO_3 buffered dialysate (>35 mEq/L)
  - Use more biocompatible membrane
  - Supplemental O_2 during dialysis

Hemolysis
- Occurs most commonly from passage of blood through kinked dialysis tubing
- Water-borne dialysate contaminant causes:
  - Chloramines
  - Copper
  - Nitrates
  - Formaldehyde
- Overheated or hypo-osmolal dialysate may cause

Acute Dyspnea
- Multiple potential etiologies
  - Anxiety
  - Reactive airway disease
  - Pleural or pericardial effusions/tamponade
  - Pneumonia
  - Cardiac dysfunction
  - Hypotension
  - Acute blood loss/severe anemia
  - Volume overload (Acute pulmonary edema)
  - Others
- Acute pulmonary edema in the hemodialysis patient
  - Common clinical problem
    - ★ Dietary indiscretion (salt and/or water)
    - ★ Missed dialysis
    - ★ Myocardial dysfunction/arrhythmia
  - Diagnosis
    - ★ Physical exam
    - ★ Chest radiograph
    - ★ Excess weight over dry weight
  - Treatment
    - ★ Dialysis (actually ultrafiltration) best
    - ★ Sitting position

        - ★ Oxygen supplementation
        - ★ Morphine
        - ★ Furosemide (venodilation vs diuresis)
        - ★ Nitroglycerin SL or IV vs nitroprusside
        - ★ Oral sorbitol

Air Embolism

- Rare, but life-threatening event
- Suspect if acute cardiorespiratory or neurologic complication
- Signs/Sx: dyspnea, tachypnea, chest pain, hypotension, confusion, cardiac arrest
- "Mill wheel" murmur on cardiac auscultation
- Management:
    - ♦ Clamp access lines
    - ♦ Lt lateral decubitus, Trendelenburg position
    - ♦ 100% oxygen
    - ♦ Aspiration of RV outflow tract if arrest (percutaneous)
    - ♦ Hyperbaric oxygen therapy

Dialysis Disequilibrium

- A constellation of neurologic symptoms and signs produced during or up to 12^{o} after hemodialysis
- Most common in new dialysis patients
- Pathophysiology thought to be rapid decrease in total body osmolality by hemodialysis, while brain stays relatively hyperosmolar and swells, causing increased intracranial pressure
- Symptoms/signs
    - ♦ Headache, malaise, nausea, vomiting, muscle cramps, hypertension
    - ♦ More severe cases: altered mental status, seizures, coma, death
- A diagnosis of exclusion: Differential diagnosis of altered mental status in hemodialysis patients:
    - ♦ Structural
        - ★ Cerebrovascular accident
        - ★ Subdural/epidural hematoma
        - ★ Intracerebral abscess
        - ★ Malignancy/metastasis
    - ♦ Metabolic
        - ★ Disequilibrium syndrome
        - ★ Uremia
        - ★ Drug toxicity/withdrawal
        - ★ Meningitis/encephalitis
        - ★ Hypertensive encephalopathy
        - ★ Hypoperfusion/hypovolemia
        - ★ Sepsis
        - ★ Glucose/electrolyte abnormality
        - ★ Dialysis dementia
- Treatment
    - ♦ Rule out other causes
    - ♦ Treat seizures (benzodiazepines, phosphenotoin)
    - ♦ Mannitol (increases serum osmolality - decreases brain edema)
    - ♦ Hyperventilation (decreases cerebral blood flow - decreases ICP)
- Prevention: Begin early hemodialysis with short, frequent sessions

Complications of Hemodialysis Access (Angioaccess Dysfunction)

- After initial placement, about $4000.00 and 3 hospital days per year spent on hemodialysis access maintenance (Radiologic access placement much less expensive than surgical placement with equal success)
- Bleeding - discussed previously
- Infection - discussed previously
- Aneurysms and pseudoaneurysms

- Thrombosis
- Distal vascular compromise

Shunt Aneurysms and Pseudoaneurysms

- Aneurysms
  - Caused by repeated venopuncture of same site
  - May rupture through skin
  - Severe bleeding: local compression, immediate vascular surgery consult
- Pseudoaneurysms
  - Pulsating extravascular hematomas
  - More common with AV prosthetic grafts
  - Causes:
    - Repeated access or back wall punctures
    - Use before adequate maturation (Usually about 6 weeks after placement)
  - More likely than true aneurysm to present with bleeding or infectious complications
  - May also require surgical intervention

Thrombosis of Vascular Access Sites

- Most common vascular access-related problem, usually due to stenosis proximal to the venous anastomosis, caused by progressive intimal hyperplasia
- Presents w/ lost, diminished site thrill/bruit
- Etiology
  - Direct trauma
  - Prolonged compression (eg. during sleep)
  - Prolonged low flow states (eg. hypotension, stenosis)
  - Hypercoagulable state
- Common post-dialysis
  - Hypovolemic state
  - Prolonged compression due to access site bleeding
- Evaluation*
  - Early intervention important in salvage?
  - Angiography to define underlying anatomy
  - Prompt vascular surgery consultation

  * Color Doppler ultrasound very effective to screen for vascular access stenosis before occlusion
- Treatment options
  - Thrombolysis
    - Effective only if established access site occlusion (Not used if first few days post access creation)
    - Most effective technique: Arterial and venous catheter infusion w/ urokinase
    - Contraindicated if suspected infection
    - Success rate 60-90%
    - Hematoma formation main complication
    - Angioplasty of stenotic site usually also required
  - Angioplasty
    - Stenosis of venous anastomosis causes late access thrombosis (58-81%)
    - Intimal hyperplasia causes occlusion
    - Angioplasty successful in up to 90%
    - Stents now often used
  - Surgical thrombectomy and shunt revision

Vascular Insufficiency of Hemodialysis Vascular Access

- Etiology
  - Steal syndrome due to reversal of distal arterial flow
  - Venous hypertension may also contribute
- Incidence < 1%
- Presentation

- Pain of distal musculature with exercise
- Pain/ulceration of extremity
- Swelling, cyanosis, hyperpigmentation

- Treatment
  - Elevation (intermittent)
  - Distal compression bandage?
  - Vascular surgical consult

High Output Cardiac Failure Due to Hemodialysis Vascular Access

- Rare
- Usually requires >20% of cardiac output through AV fistula
- Presents with congestive heart failure unexplained by other factors
- Diagnosis:
  - Branham's sign (Temporary fistula occlusion results in reduced heart rate)
  - Doppler ultrasonic flow meter or isotope dilution
- Treatment: Shunt revision

Peritoneal Dialysis (PD)

- Less common technique compared to hemodialysis (16% of dialysis patients in USA, but 38% in Canada, 50% in United Kingdom and over 90% in Mexico undergo peritoneal dialysis)
- Mortality advantage over hemodialysis if matched for pretreatment prognostic factors (exception: elderly diabetics)
- Technique:
  - Dialyzing fluid introduced through implanted abdominal percutaneous catheter
  - Fluid equilibrates with plasma water (dwell time)
  - Fluid drained and replaced
- Advantages:
  - Avoid vascular access/complications
  - Avoid need for anticoagulation during dialysis
  - Fewer fluid and diet restrictions
  - In-center dialysis not required
  - Less day-to-day biochemical fluctuations
  - Less hemodynamic stresses
- Disadvantages
  - Complications of peritonitis/peritoneal catheter malfunction
  - Requires daily treatments
- Technique options:
  - Intermittent peritoneal dialysis (IPD)
    - ★ Rapid cycling of dialysate ($\leq$ 60 min)
    - ★ Accomplished several times daily
  - Continuous cycling peritoneal dialysis (CCPD)
    - ★ Rapid cycling during the night
    - ★ Uses automated cycler
  - Continuous ambulatory peritoneal dialysis
    - ★ Most widely used PD technique
      1-2 L warm dialysate from collapsible bag
      Dwell time 4-6 hours
      Fluid drained back to bag
      Repeated 4 times/day
- Equipment for Peritoneal Dialysis
  - 1-2 L bags with 1.5, 2.5, or 4.25% dialysate (percentages refer to dextrose concentration)
    - ★ Small volumes if pain or difficulty breathing w/ large volumes
    - ★ High dextrose (4.25%) - more ultrafiltration
  - Peritoneal access catheter
    - ★ Tenckoff catheter most common:

Straight silastic tube with multiple intraperitoneal perforations
One or two Dacron felt cuffs
External connector

- ★ Multiple variations exist
  - Discs to prevent catheter displacement
  - Devices to prevent omental plugging
- ♦ Connector (Y connector allows optimal peritoneal dialysis technique and reduces complications compared to straight connector)

Peritoneal Dialysis-Related Complications:
Mechanical Complications: Inadequate outflow

- Etiology
  - ♦ Occurs in 7% early after catheter insertion
  - ♦ Tip migration (epigastric, hypochondrial)
  - ♦ Constipation
  - ♦ Omental plugging (more common in children; reduced by partial omentectomy at insertion)
  - ♦ Luminal blood or fibrin clots (thrombolytics may resolve)
- Treatment: Etiology dependent

Mechanical Complications: Leaks

- Incidence
  - ♦ 5-10% immediately post-operative
  - ♦ 2-4% late
- Etiology
  - ♦ Exit site leaks
    - ★ Present with clear fluid from cutaneous catheter ostium (high Accucheck)
  - ♦ Internal cuff leaks
    - ★ Present with abdominal wall edema
- Treatment
  - ♦ Prophylactic antibiotics indicated
  - ♦ May resolve with supine PD
  - ♦ Short term cessation of PD (hemodialysis)
  - ♦ Surgical correction

Infectious Complications of Peritoneal Dialysis

- Peritonitis most common complication of PD
  - ♦ Risk greatest with CAPD
  - ♦ Two major infection routes
    - ★ Transluminal (Break in technique)
    - ★ Contiguous spread (tunnel or exit site infection)
  - ♦ Rare causes
    - ★ Direct intestinal tract contamination
    - ★ Hematogenous seeding
  - ♦ Symptoms (often mild)
    - ★ Abdominal pain/tenderness
    - ★ Cloudy dialysate
    - ★ Nausea/vomiting
    - ★ Fever
  - ♦ Diagnosis
    - ★ Aspirate catheter, freshly drained bag
    - ★ Send for gm stain, culture, cell count + diff

      Dx if > 100 WBC/mm^3 w/ >50% PMN's
      Gram stain positive in 30-40%
  - ♦ Microbiology
    - ★ Vast majority due to single organism (Polymicrobial - think bowel perforation)

50-70% gram positive organisms
- Staph aureus
- Staph epidermidis

~20% gram negative organisms
- Enterobacteriaceae
- Acinetobacter
- Pseudomonas

<5% fungal or mycobacterial

10-20% culture negative

- Treatment
  - Most patients effectively treated as outpatients
  - Indications for admission:
    - Severe illness/severe abdominal pain
    - High fever
    - Recurrent vomiting
    - Hypotension
    - Inability to administer antibiotics at home
    - Inability/unreliable 24-48° followup
  - Dialysate gram stain directs initial therapy
    - Gram positive organisms:
      - 1st generation cephalosporin IM/IV, often same intraperitoneal (IP)
      - Vancomycin if cephalosporin allergy or suspected methacillin-resistant Staph.
    - Gram negative organisms
      - Aminoglycoside IM/IV, then IP doses
    - Negative gram stain
      - Empiric 1st generation cephalosporin or vancomycin and an aminoglycoside
  - Other antibiotic regimens
  - Treatment for 10-14 days

Peritoneal Catheter Infections

- Exit-site infections
  - Presentation:
    - Exudative drainage around catheter
    - Erythema
    - Local tenderness
  - Treatment
    - Culture area around catheter (?)
    - Local cleansing/antimicrobial ointment
    - Oral 1st generation cephalosporin
- Tunnel infection
  - Usually stems from exit-site infection
  - May cause SC abscesses along tunnel
  - Hints to tunnel infection
    - Exit-site infection or peritonitis fails to respond to appropriate therapy
    - More than minor tenderness, erythema or fluctuance around catheter
    - Recurrent recent bouts of peritonitis
  - Like exit-site infection, most common causes Staph epidermidis and aureus
  - Ultrasound may assist in diagnosis
  - Treatment
    - Abscess incision and drainage
    - Frequently, catheter removal required

Other Complications in Peritoneal Dialysis

- Hernias
  - Occur in 10-25% of CAPD patients

- Incisional and inguinal most common
- May incarcerate

- Hemoperitoneum
  - Fairly common
  - Usually 2° to minor irritation
  - Send fluid to R/O peritonitis
  - Causes of Hemoperitoneum in PD Patients
    - Retrograde menstruation/ovulation/ectopic pregnancy
    - Catheter-induced trauma (omental abrasion, repositioning)
    - Bowel disease (ischemic, inflammatory)
    - Peritonitis
    - Cysts (ovarian, polycystic kidney, acquired cystic kidney)
    - Abdominal trauma
    - Strenuous exercise (including sexual activity)
    - Systemic bleeding (thrombocytopenia, anticoagulants)
    - Hypertonic exchanges (hyperemia)
    - Pancreatitis
    - Vasculitis (systemic lupus erythematosus)
    - Sclerosing peritonitis
    - Adhesions
    - Granulosa cell tumor
    - Cholecystitis
    - Colonoscopy
    - Dissection from adjacent sites (femur hematoma, spleen, colon)
    - Previous hepatitis
    - Enema/constipation
    - Extracorporeal lithotripsy
    - Splenic infarction
- Pneumoperitoneum
  - Occurs in ~5% of CAPD patients
  - May be 2° to visceral perforation
- Acute hydrothorax
  - Fairly rare (1.6% of 3200 CAPD pts in one study)
  - Most (88%) rt-sided, associated with dyspnea
  - Glucose high in pleural fluid (2-3 x blood)
  - Therapy is temporarily discontinuing PD

Pseudoemergencies in Dialysis Patients

- Constipation
  - Common with aluminum-containing antacids
  - Treatment:
    - Oral Sorbitol (1-2 oz 70% soln)
    - Colace or Metamucil to prevent
    - Avoid: Mg-containing laxatives
- Nausea and vomiting
  - Post-dialysis usually due to acute volume depletion
  - Must rule out treatable causes (e.g. drug side effect)
  - Treat with antiemetics (Metoclopramide for diabetics)
  - Recommend less aggressive volume removal with subsequent dialysis
- Itching
  - Common - multiple causes
    - Unclear etiology
    - Dry skin - try oil-based topical ointments (eg. Vaseline Intensive Care)
    - Treatment:
      Diphenhydramine or hydroxyzine, cholestyramine and/or oral charcoal
      Topical menthol, camphor and phenol
    - Parathyroidectomy if severe and 2° to hyperPTH
- Musculoskeletal problems
  - Night muscle cramps - try quinine (325 mg PO) or diazapam (5-10 mg PO)
  - Frequent fractures/tendon ruptures
  - Calcific periarthritis

Summary:

- Dialysis patients challenging to emergency physicians
  - Many possible complications
  - Associated co-morbid disease processes
- Awareness of potential problems assists diagnosis:
  - Hemodialysis issues
  - Peritoneal dialysis issues
- Engage nephrologist, surgeon early in diagnostic evaluations and treatment

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