Content Description

This session will provide a review of the most common renal disorders seen in critically ill patients. This information will serve to prepare the participant for the CCRN and/or PCCN certification examinations.

Learning Objectives

At the end of this session, the participant will be able to:

1. Discuss the pathophysiology of acute kidney and chronic renal failure.

2. Describe the collaborative management of patients with acute and chronic renal disorders.

3. Describe the life-threatening electrolyte abnormalities associated with acute and chronic kidney disease and their management and prevention.

Summary of Key Points/Outline

I. Renal failure

   A. Acute kidney injury
      1. Etiology
      2. Management and prevention

   B. Chronic renal failure in the ICU patient
      1. Etiology
      2. Complications
      3. Management strategies

   C. Electrolyte abnormalities in acute and chronic kidney disorders
      1. Potassium
      2. Sodium
      3. Calcium
      4. Acid-base disturbances
Bibliography/Webliography


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Anatomy of the kidneys

Description
Nephron is the basic functional unit of kidney
Renal corpuscle (glomerulus and Bowman’s capsule)
Kidneys receive 20-25% of cardiac output, 1200 ml/min

Functions of the kidney

Regulation of acid-base balance
Reabsorption and production of bicarbonate
Excretion of small amounts of hydrogen ions (acid)
Regulation of blood pressure
Activation of rennin-angiotensin-aldosterone system
Antidiuretic hormone (ADH) stimulation
Sodium and water reabsorption
Electrolyte regulation
Sodium, potassium, calcium, phosphate, magnesium, chloride
RBS synthesis
Excretion of hormone erythropoietin by endocrine cells in kidney

Acute kidney injury (AKI)

Defined
No commonly accepted definition
Best: A syndrome that develops when there is a sudden decline in the glomerular filtration rate
Description
Most common renal disorder in critically ill
Occurs in 1% of all hospitalized patients
High morbidity and 50% mortality rate
Often accompanied by multiple organ dysfunction syndrome (MODS)
Pathophysiology
Deterioration of renal function
Poor excretory function
Poor homeostatic function of fluid and electrolytes
Retained nitrogenous water (urea) – azotemia
Oliguria commonly develops
   Urine output of less than 400 ml/24 hours
Anuria – urine output less than 100 ml/24 hrs
Uremia-toxic urea increase in ARF

A. Etiology of AKI
   1. Related to type of renal failure
   2. Three categories related to the location and nature of renal dysfunction
      a Prerenal
      b Postrenal
      c Intrarenal

B. Prerenal failure
   Etiology
   Impaired renal perfusion results in ARF
      1) Fluid volume loss – bleeding, dehydration
      2) Decreased cardiac output – heart failure
      3) Vasoconstriction of renal vessels
   If prolonged or severe can lead to acute tubular necrosis (ATN)
   Reversible if treated within 24 hours
   Laboratory values
      Elevated serum blood urea nitrogen (BUN)
      Creatinine levels rise more slowly
      BUN: creatinine ratio of 15:1

C. Postrenal AKI
   3. Obstruction to the flow of urine
      a Occurs at any point along the urinary tract
   4. Rapidly reversible once obstruction is alleviated
   5. Etiology
      a Renal stones
      b Tumors
      c Edema R/T surgery or trauma
      d Urinary catheter obstruction
      e Blood clots
      f Injury to ureter R/T surgery or trauma

D. Intrarenal AKI
   1. Etiology
      a Inflammation
      b Prolonged ischemia
      c Nephrotoxic insult to renal tubules or glomerulus
   2. Decreased GFR and shunting of blood from medulla to renal cortex
   3. Leads to hypoxia of tubules of nephron
      a Tubules obstructed with dead cells and debris
      b Back flow of urine in system to glomerulus
4. Types of intrarenal AKI
   a. Cortical involvement
      1) Vascular or infectious process alters outer renal layer
         (a) Lupus, Goodpastures, infections
   b. Medullary involvement – most common
      1) Nephrotoxic insults
         (a) Antibiotics (aminoglycosides), analgesics
         (b) ACE inhibitors
         (c) Contrast media
   c. Ischemic causes
      1) Hypotension
      2) Trauma
      3) Shock, sepsis
      4) Transfusion reactions

E. Course of AKI
   1. Initiation phase
      a. Begins at the time of the precipitating event
      b. Renal function worsens
      c. ARF is potentially reversible at this stage
   2. Oliguric/Anuric Phase
      a. Intrinsic renal damage has occurred
      b. Urine output at its lowest
      c. Some patients are nonoliguric (> 400 ml/24 hours)
      d. Spans 7 to 14 days but may last months

3. Diuretic Phase
   a. Renal tissue is recovering from the insult & healing
   b. Gradual increase in urine output
   c. Related laboratory indices improve
   d. Some have a notable diuresis
   e. Avoid dehydration & pre-renal condition

4. Recovery Phase
   a. Improving renal function
   b. May span 3 to 12 months
   c. Laboratory results return to patient’s normal

F. Patient Assessment
   1. Signs of uremia
      a. Fatigue
      b. Confusion
   2. Assess for edema
   3. JNVD
   4. Skin turgor, mucous membranes
   5. Note admission weight & changes

G. Diagnosis of AKI
   6. Serum BUN & creatinine elevation
7. Elevated potassium
8. Sodium level-variable
9. Hgb & Hct decreased
10. Urine osmolality decreased
11. Fractional excretion of urine (FeNa)
   a. Prerenal failure FeNa < 1%
   b. ATN FeNa > 1%
12. Prerenal failure-urine is concentrated
13. Intrarenal failure-dilute urine
14. KUB
15. Renal ultrasound
16. IV pyelogram
17. Angiography
18. CT scan
19. Radionucleotide renal scan
20. Renal biopsy

H. Systemic Effects of AKI

1. Cardiovascular
   a. Fluid overload
   b. Pulmonary edema
   c. HTN-renin, volume overload
   d. Dysrhythmias- electrolyte abnormalities & acidosis
   e. Pericarditis due to uremia

2. Respiratory
   a. Pneumonia

3. Gastrointestinal
   a. Anorexia, nausea & vomiting
   b. Bleeding & gastritis

4. CNS & Neuromuscular
   a. Lethargy, confusion, twitching

5. Hematologic
   a. Anemia and thrombocytopenia
   b. Bleeding

6. Hypocalcemia

7. Metabolic Acidosis
   a. Oliguria- unable to eliminate acids
   b. Renal buffers fail

8. Hyperkalemia
   a. Potassium cannot be excreted
   b. Higher risk in the oliguric and anuric patientRelease of intracellular potassium to extracellular
   c. Dysrhythmias and cardiac arrest

I. Prevention Strategies for Acute Kidney Injury
1. Prevent precipitating events
2. Acetylcysteine in prevention of CIN
   a One day before and on day of contrast
   b Decreases renal injury in high risk patients (DM)
   c Prevents toxic effects of contrast on tubules
   d Bicarbonate & IV hydration also protect kidney

J. Management Strategies for Acute Kidney Injury
1. Postrenal failure
   a Insertion of a urinary catheter
   b Suprapubic tube
   c Percutaneous or cystoscopically placed stent
2. Diuretics
   a Controversial
   b Promote change from oliguria to nonoliguria
   c Increase renal perfusion & GFR
   d Must correct hypovolemia before use of diuretics
   e Types and uses of diuretics

   (1) Loop diuretics (furosemide) improves renal blood flow and results in solute diuresis
      (a) Large or trial of escalating doses (200 mg furosemide) may be associated with complications such as ototoxicity
   (2) Mannitol- osmotic diuresis and protects against further injury especially in myoglobinuria
3. Dopamine- still controversial
   a May use if still oliguric after fluids & diuretics
   b Low doses may increase renal perfusion and GFR but do not prevent ARF or improve outcomes

K. Management of Hyperkalemia
1. Arrhythmia monitoring- (lethal ventricular)
2. Administer cation exchange resins (Kayexalate)
   a Exchanges sodium for potassium ion in GI tract
3. Intravenous glucose and insulin
   a Drive potassium into the cell temporarily lowering high serum potassium & protecting heart
4. Intravenous sodium bicarbonate given to increase plasma pH
   a Increased pH forces potassium intracellular
5. Intravenous calcium for cardio-protection
6. Eliminate other sources of potassium

L. Fluid & Electrolyte Disturbances in AKI
1. Other electrolytes in ARF
   a Hypernatremia in oliguric patients
   b Managed by sodium restriction
   c Judicious fluid management – highly variable
M. Renal Replacement Therapy

1. Indications
   a. Extracellular fluid volume overload
   b. Acidosis or Electrolyte abnormalities
   c. Uremia with pericarditis or encephalopathy
   d. Timing for initiation varies

2. Types of Renal Replacement Therapy
   a. Hemodialysis
      (1) Extracorporeal therapy - toxins removed from blood by diffusion across semipermeable membrane
      (2) Vascular access needed
      (3) High flow rates result in efficient & rapid removal of small molecules and extracellular fluid
      (4) Continuous renal replacement therapy (CVVH)
      (5) Principles of Hemodialysis
         (a) Osmosis
            (i) Water movement from area of lower to higher concentration of particles
         (b) Diffusion
            (i) Particles move from high concentration to low
         (c) Convection
            (i) Solute transfer with flow of water across a pressure gradient
   
   b. Peritoneal Dialysis
      (1) Performed continuously
      (2) Clearance of solutes from diffusion across peritoneal membrane
      (3) Fluid removal with hypertonic glucose solution
      (4) Little clinical applicability in ARF

   c. Continuous Renal Replacement Therapy
      (1) Extracorporeal therapy to remove toxins from the blood via a semipermeable membrane - performed continuously
      (2) Continuous arteriovenous hemofiltration (CAVH)
      (3) Continuous venovenous hemodialysis (CVVHD)
      (4) Continuous venovenous hemofiltration (CVVH)
      (5) Continuous venovenous hemodiafiltration (CVVHDF)

N. Nutrition in Acute Renal Failure

1. Pre-existing or hospital acquired malnutrition contributes to mortality
2. Increased protein catabolism & excess release of amino acids from skeletal muscle
3. Sustained negative nitrogen balance
4. Calorie needs of 30 kcal/kg/day to reduce protein catabolism
5. Oral/enteral route preferred
6. Critically ill may require parenteral nutrition
7. Restrict sodium, water, potassium

II End-Stage Renal Disease (ESRD)

- Progressive decrease in number of functioning nephrons
- May occur over months to years with decreasing GFR
- Primary renal disease or secondarily due to systemic disease or by acute damage
- "End-stage" when dialysis or transplantation needed for survival

A. Causes of ESRD
   - Diabetes
   - Hypertension
   - Glomerulonephritis

B. Diagnosis of ESRD
   - Elevated creatinine & BUN
   - Creatinine clearance is decreased
   - Metabolic acidosis
     (1) Accumulation of hydrogen ions
     (2) Lack of erythropoietin production by kidneys

C. Management of ESRD
   - Fluid volume excess (sodium & H2O retention)
     - Fluid restriction
     - Dialysis
   - Hypertension
     - Antihypertensive therapy
   - Fatigue due to anemia
     - Erythropoietin therapy subcutaneously
   - Depression, anxiety

   Hemodialysis
   - 3 to 4 times per week
   - Peritoneal dialysis
     - For those who meet strict criteria
     - Cycled overnight
     - Continuous ambulatory (CAPD)
   - Renal transplantation

     (1) Living (related or unrelated)
     (2) Cadaveric
     (3) Best outcomes with HLA antigen matching
     (4) Lifelong immunosuppressive therapy
D. Management of Electrolyte Disturbances in ESRD

A. Potassium excess
   1. Diminished excretion
   2. Redistribution to extracellular fluid due to acidosis
   3. May result in life-threatening dysrhythmias
   4. Control with diet restriction
   5. Management as with ARF

B. Phosphate excess
   1. Adverse effect on bone mineral content
   2. Elevate parathyroid levels
   3. May be associated with low serum calcium
   4. Decreased excretion, excess intake
   5. Administer phosphate binders

C. Magnesium excess
   1. Impaired excretion of the ion
   2. Rarely symptomatic
   3. May depress cardiac conduction
   4. Avoid magnesium-containing antacids and laxatives such as maalox & milk of magnesia
   5. Administer magnesium--containing antacids and laxatives such as maalox & milk of magnesia

D. Hypocalcemia
   1. Associated with hyperphosphatemia & Vitamin D deficiency
   2. May suppress cardiac function
   3. Reduce dietary phosphorus
   4. Calcium & Vitamin D replacement
   5. Aluminum-containing antacids to bind phosphate
   6. Monitor serum concentrations

E. Nutritional concerns
   1. Sodium restriction of 2 to 3 grams per day
   2. Restrict potassium-containing foods
   3. Many need protein restriction
   4. Phosphate restrictions

Management of ESRD

1. Avoid potential for complications related to use of drugs with poor renal excretion
2. Careful administration of lanoxin & monitor serum drug levels

C. Complications of ESRD
   1. Monitor for and treat metabolic acidosis
      a. Accumulation of hydrogen ions (retained acid)
      b. Renal bicarbonate wasting
      c. Compensatory hyperventilation induces fall of pCO2
      d. No treatment is usually required but acutely ill hospitalized patient at risk
2. Monitor for and treat cardiovascular complications commonly associated with ESRD
   a. Cardiovascular disease- most common cause of death in patients with ESRD
   b. Pericarditis associated with uremia

Certification Questions
1. A 58-year-old continuous ambulatory peritoneal dialysis (CAPD) patient is admitted to the ICU following a small bowel resection. The patient is hemodynamically stable and has a 3L excess fluid balance. For these reasons, the critical care nurse anticipates the patient’s end stage renal disease (ESRD) will be managed using:
   A. Peritoneal dialysis (PD)
   B. Slow continuous ultrafiltration (SCUF)
   C. Hemodialysis (HD)
   D. Continuous venovenous hemofiltration (CVVH)

2. In planning the care of a patient in acute renal failure, the critical care nurse ascertains the patient is at risk for infection owing to:
   A. Excessive carbohydrate intake
   B. Fluid overload
   C. Protein-calorie malnourishment
   D. fluid restriction

3. While assessing the patient, the nurse notices spasms of the patient’s hand when the blood pressure cuff inflates on her arm. This carpopedal spasm is due to
   A. Hyperphosphotemia
   B. Hyperkalemia
   C. Hypocalcemia
   D. Hypernatremia

4. Excessive volume loss associated with hypotension is associated with
   A. Prerenal ARF
   B. Intrarenal ARF
   C. Postrenal ARF
   D. Acute glomerulonephritis