BAPTIST HEALTH School of Nursing
NSG 4017: Critical Care Nursing
Nursing Management for Patients with Altered Renal Functioning

OBJECTIVES

At the close of this lecture, the student will be able to:

1. Apply the knowledge of basic anatomy and physiology to the patient with acute renal failure.
2. Compare and contrast the etiologies of acute renal failure: prerenal, intrarenal, and postrenal.
3. Understand and compare the different phases of acute renal failure.
4. Recognize the manifestations of acute renal failure, including the symptoms for each of the different phases.
5. Know the medical management of acute renal failure and nursing’s role in achieving this.
6. Prepare a comprehensive nursing plan of care for the patient with acute renal failure.
7. Define dialysis and hemodialysis.
8. Prepare a teaching plan for the patient undergoing hemodialysis.
9. Recognize the complications that may result from the use of hemodialysis and know the nursing care required for the significant complications.
10. Know the survival rates for the types of kidneys used in transplants.
11. Know the legal requirements for nurses in relation to death and organ donation.
12. Recognize the complications of renal transplants.
13. Prepare a comprehensive nursing plan of care for the renal transplant patient.
14. Know the symptoms of renal carcinoma.
15. Prepare a comprehensive nursing plan of care for the nephrectomy patient.
16. Prepare a nursing plan of care for the patient in each category of renal trauma.
17. Educate the patient regarding the nursing care surrounding the patient undergoing plasmapheresis.
18. Implement the necessary nursing management for a patient recovering from a plasmapheresis procedure.

TERMS TO KNOW:
1. Glomerulus
2. Juxtaglomerular apparatus
3. Nephron
4. Prerenal
5. Intrarenal
6. Postrenal
7. Anuria
8. Oliguria
9. Polyguria
LECTURE OUTLINE

I. Assessment
   A. Bleeding - flanks. Abdominal distention, abdominal guarding.
   B. Volume depletion/overload
      1. Neck veins
      2. Hand veins - while hand in dependent position, if venous filling takes longer than 5 seconds, hypovolemia is suggested. After elevating the hand, if hand vein distention does not disappear in 5 seconds, fluid overload is suspected.
      3. Skin turgor
      4. Oral cavity
   C. Edema - may indicate
      1. Fluid volume overload
      2. Disorder in another body system
      3. Loss of albumin
      4. Circulatory difficulties
      5. Poor venous return
      6. Indentation made by fingertip should disappear in 15 seconds. See pitting edema guideline in Black and Hawks.
   D. Auscultation
      1. Heart -
         a. Fluid overload - 3rd heart sound
         b. Friction rub - pericardial, from uremia.
      2. Blood pressure - Orthostatic hypotension - drop of more than 20 mm Hg or rise in pulse of more than 20 BPM
      3. Lungs
         a. Fluid overload - crackles
         b. Dyspnea
   E. Percussion - fluid wave can be created in ascites by placing hand on one side of abdomen and tapping opposite flank with other hand. (Protuberant, rounded abdomen and abdominal striae are also symptoms.)
   F. Urine
      1. glucose, protein - should NOT be present
      2. Lytes - requires 24 hr. specimen. Change may indicate intrarenal ARF
3. Sediment - casts and epithelial cells may indicate intrarenal ARF
4. Hematuria - damage somewhere in renal system

II. Acute renal failure
A. Definition - abrupt loss of kidney function. May occur over a period of hours to a few days.
B. Etiology - most common causes are hypotension and prerenal hypovolemia.
1. Prerenal causes - the kidneys depend on an adequate blood supply being delivered to the glomeruli!!
   a. Circulatory volume depletion, including diarrhea, vomiting, hemorrhage, excessive use of diuretics, burns renal salt-wasting conditions, and glycosuria. (Hypovolemia is associated with a high rate of mortality.)
   b. Volume shifts - 3\textsuperscript{rd} spacing, vasodilation, gram-negative sepsis.
   c. Decreased cardiac output (pump failure, pericardial tamponade, acute pulmonary embolism).
   d. Decreased peripheral vascular resistance (spinal anesthesia, septic shock, anaphylaxis).
   e. Vascular obstruction (bil. renal artery stenosis or other bil. renal artery obstruction, dissecting aneurysm).
2. Intrarenal causes
   a. Kidney diseases
   b. Acute tubular necrosis (ATN) - defined as cell destruction due to impaired renal perfusion. The most frequent cause of ARF.
      (1) hypotension, which is associated with high rate of mortality
      (2) direct damage from nephrotoxins - antibiotics, heavy metals, poisons such as mushrooms or insecticides, anesthetics, contrast dyes, organic solvents such as gasoline, other drugs such as acetaminophen, NSAIDS, salicylates. (refer to p. 854.)
      (3) rhabdomyolysis (see p. 852).
      (4) rejection of transplanted kidney.
      (5) glomerulonephritis, thrombosis, scleroderma, trauma, tumor invasion, and cortical necrosis.
3. Postrenal causes - only 5\% of cases - obstruction of the urinary tract within the tubules or the ureters or the urethra. Common causes include prostatic hypertrophy, calculi, invading tumors, surgical accidents, ureteral or urethral strictures or stenosis, and retroperitoneal fibrosis. Spinal cord injury may lead to decreased bladder emptying and a functional obstruction.
C. Pathophysiology
1. Biochemical theory - decreased renal blood flow leads to decreased oxygen delivery to the proximal tubules. The result is cell death and tubular necrosis. Vasospasms may result in tubular damage. Mortality
rate may be as high as 50%; the highest mortality rates occur when failure is caused by trauma or surgery.

2. Onset phase - the period from the precipitating event to the development of renal manifestations. May last a period of minutes or hours or even a week.

3. Oliguric-anuric or nonoliguric phase - 1 to 8 weeks, usually. Dialysis may be required during this phase.

4. Diuretic phase - a gradual or abrupt return to glomerular filtration and leveling of the BUN. Urine output may be 1000 to 2000 ml/day, which may lead to dehydration; 25% of the deaths from ARF occur during this phase.

5. Recovery phase - lasts 3 to 12 months. During this time, the client often returns to the pre-renal failure activity level. Mild tubular abnormalities, including glycosuria and decreased concentrating ability, may continue for years, and the client will continually be at risk of fluid and electrolyte imbalance, especially during times of stress.

6. Consequences
   a. Fluid and electrolyte imbalances, resulting in fluid overload or depletion, hyperkalemia, hyponatremia, hypocalcemia, and hypermagnesemia.
   b. Acidosis
   c. Increased susceptibility to secondary infections
   d. Anemia
   e. Platelet dysfunction
   f. GI complications: anorexia, nausea, vomiting, diarrhea or constipation, and stomatitis
   g. Increased incidence of pericarditis
   h. Uremic encephalopathy characterized by apathy, defective recent memory, episodic obtundation, dysarthria, tremors, convulsions, and coma
   i. Impaired wound healing

D. Clinical manifestations and diagnostic findings
   1. Alteration in expected urine output - most common symptom
   2. Non-oliguric (polyuric) renal failure - may excrete as much as 2 L/dy. Urine is dilute and nearly isomolar - low urine specific gravity (a few nephrons are still working). Hypertension and tachypnea with symptoms of fluid overload. Also, symptoms of fluid depletion, such as dry mucous membranes, poor skin turgor, and orthostatic hypotension.
   3. Oliguric renal failure - urine less than 400 ml/day (because of the changes from aging, the elderly may have oliguria at 600-700 ml/day.
      a. Symptoms from prerenal causes (history of precipitating event, such as hemorrhage or MI, etc.) Urine has high specific gravity and osmolality; little or no proteinuria. Very little urinary sodium

b. Symptoms from intrarenal (intrinsic) renal failure - edema, weight gain, hemoptysis from elevated left ventricular end-diastolic pressure, weakness from anemia, and hypertension. Urine has fixed specific gravity, high sodium concentration, and definite proteinuria. If glomerulonephritis was the cause, there will be hematuria and red blood cell and hemoglobin casts. ATN will cause muddy-brown granular casts and there may be elevated levels of serum creatinine, phosphokinase, and potassium.

c. Postrenal failure symptoms - fixed specific gravity, elevated sodium concentration and little or no proteinuria.

4. Diagnostic tests - urinalysis, urine specific gravity and sodium levels, serum creatinine, BUN, intake and output ratio. Also, urine culture and sensitivity.

E. Medical management

1. Intensive treatment includes physiologic monitoring - most interventions center on maintenance of fluid and electrolyte balance and nutrition.
   a. Dialysis: indications include significant volume overload, uncontrolled hyperkalemia or acidosis, progressive uremia as evidenced by risking BUN and creatinine concentrations, altered central nervous system function, and pericarditis.
   c. Pericarditis: assess for pleuritic pain that is relieved by sitting upright, a pericardial friction rub, tachycardia, and fever. Treatment includes steroids or NSAIDS and, possibly, pericardiocentesis.
   d. Seizures: occur due to elevated BUN. Treated with phenytoin and phenobarbital.
   e. Anemia: treat with transfusions or erythropoietin.
   f. Bleeding tendencies: vitamin K. Lower BUN, since BUN interferes with platelet aggregation.

2. Fluid replacement - given carefully. Replacement volumes are usually calculated on the basis of some fraction of previous day’s urine output plus an amount to calculate for insensible body loss over previous 24 hours. Some physicians may use body weight as a guide.

3. Diuretics - Furosemide and mannitol most common. Diuretics are most likely to affect the outcome of non-oliguric ARF.

4. Electrolyte replacement - based primarily on urine and serum electrolyte concentrations.
   a. Hyperkalemia - the result of inadequate excretion. Released from body cells when acidosis present and rapid tissue catabolism occurs. Most dangerous due to possibility for cardiac arrhythmias
and arrest. ECG monitors used. Kayexelate, an exchange resin, may be administered with sorbitol, an osmotic cathartic, through the NG tube, or orally. Potassium-containing foods and medications should be avoided. In an emergency, such as an extremely high serum potassium, a bolus of 50% glucose with regular insulin, followed by sodium bicarbonate, can be given to avoid cardiac arrest.

b. Hyponatremia - due to too much IV fluid, causing serum dilution. Treat with fluid restriction and, perhaps, careful administration of hypertonic saline solutions.

c. Hyperphosphatemia - treat with reduced dietary intake and phosphate binders such as aluminum-based antacids.

d. Hypocalcemia - treat hyperphosphatemia

e. Metabolic acidosis - results from accumulation of end waste products. Treated with sodium bicarb. Dialysis may help.


F. Nursing management

1. Recognizing risk factors - age, dehydration, elevated creatinine levels, contrast dye, nephrotoxic meds.


3. Skin integrity - at considerable risk due to poor nutrition and edema. Use meticulous skin care frequently, special mattresses when the client has limited movement. Use ROM policies if necessary.

4. Infection - avoid catheters, to include urethral catheter. If one is present, provide meticulous cath care. Watch for symptoms of infection. Pulmonary hygiene.


6. Anxiety - frequent, careful explanations needed. Provide support for family as necessary. Be aware that client may be mechanically ventilated and unable to communicate well.

7. Teaching - teach family and client to understand the disease and its implications and need for long-term follow-up care.
III. Dialysis - peritoneal or hemodialysis. Both may be used temporarily or permanently. Works through ultrafiltration (the removal of fluid from the blood through osmosis or hydrostatic pressure) and diffusion. Both processes use a semipermeable membrane.

A. Goals
   1. Removal of end products of protein metabolism, such as urea and creatinine.
   3. Correction of acidosis and replenishment of bicarbonate buffer system.
   4. Removal of excess fluid from blood.

B. Peritoneal dialysis - see p. 352 in Urden.

C. Hemodialysis - first developed in 1943 - Medicare started paying for treatments in 1973. Treatment of choice for barbiturate overdose. Most hemodialysis done for CRF and is performed at outpatient dialysis center. Performed in hospital center or ICU as treatment for acute renal failure (BUN approx 90 or creatinine approx 9) or CRF with excessive potassium.
   1. Procedure - blood diverted from client through dialyzer. Machine is the pump. Large tubing goes from client’s artery to machine to client’s vein. See picture p. 345 in Urden. In the hemodialysis machine, blood runs on one side of the semipermeable membrane, dialysate on the other side. Toxins diffuse across. Strict asepsis is necessary. A vital aspect is an adequate blood supply. Access to this blood supply is via an external shunt, a fistula, a subclavian catheter (Quentin) or, in an emergency, a Quentin catheter placed in the femoral vein. An internal fistula is the access of choice for CRF. Infection at the site of insertion and clotting of fistula or hunts are complications that necessitate creation of new fistula or creation of new shunt.
   2. Schedules
      a. Continuous renal replacement therapy - ideal for the hemodynamically unstable pt because abrupt fluid losses and solute changes don’t happen. Usually venous blood. MAP of at least 70 mm. Hg is desirable. See table 20-8 for ultra-filtration rates. Critical care nurse is responsible for calculating fluid replacement necessary. Formula is drainage in bag (called ultrafiltrate) - all fluids taken in by pt. = output. Exact replacement fluid depends on pt’s clinical condition and plan of care. Ordered by physician.
      b. For CRF - 3-4 hrs. of treatment 3 days per week.
   3. Therapeutic effects - clear waste products from the body; restore fluid, electrolyte, and acid-base balances; and reverse some of the untoward manifestations of irreversible renal failure. Unfortunately, hyperlipidemia seems to increase by renal osteodystrophy usually improves. Pruritus is a problem. Dosage schedules of ROUTINE medications must be altered to prevent loss of medications through dialysis. Supplemental doses of some
medications may be required.

4. Complications
   a. Technical problems - blood leaks, overheating of dialysate, insufficient loss of fluid, improper concentration of salts in the dialysate, and clotting.
   b. Hypotension or hypertension, cardiac arrhythmias from potassium imbalance, air embolus, hemorrhage from heparinization - may develop subdural, retroperitoneal, pericardial or intraocular bleeding. For clients with ulcer disease, GI bleeding is an issue.
   c. Significant complications
      (1) infection - hep B is most common.
      (2) dialysis equilibrium syndrome - occurs due to rapid removal of some ions from the blood. Symptoms: mental confusion, deterioration of the level of consciousness, headache, and seizures. May last several days and usually occurs in patients just being started on dialysis treatments, but also may be seen in critical patients.

IV. Renal transplant - first successful transplant completed in the 1950s. A successful transplant prolongs life and markedly improves the quality of life. Transplants have been made possible by immunosuppressive medications and by the availability of donor kidneys. One year survival rates for the transplanted kidney is 85 to 90% in living donor transplants and 75-80% with cadaver kidneys. The PRIMARY limiting factor to the number of transplants done is the availability of kidneys, although more have been available since the passage of “request” legislation in 1992. The program in Arkansas is managed by ARORA (Arkansas Regional Organ Recovery Association). Recipients are usually less than 70 years of age who have an estimated life expectancy of 2 years or more and in whom the transplant will improve the quality of life. The psychosocial concerns include the client’s (1) feelings about the transplant, (2) understanding and acceptance of the risks and chances of graft survival, and (3) family and social obligations. Infection and active malignancy are the only absolute contraindications to transplantation. Clients with liver disease, psychological disorders, advanced atherosclerosis, hypertension, respiratory disease, and gastrointestinal bleeding need particular consideration.
   A. The most desirable source of kidneys for transplant is a living related donor whose tissue matches the client closely.
   B. Cadaver donors - must have been declared brain dead, be under 60 years of age, have normal renal function, no malignant disease outside the CNS, no generalized infection, no significant hypertension, no abdominal or renal trauma, negative hepatitis B antigen and human immunodefiency (HIV) virus antibody, and continuous ventilation and heartbeat until the kidneys are surgically removed from the body. The kidneys can only be allowed to be warm and ischemic for 1 hour total but may be cool and ischemic for 24-48 hours.
C. Surgery.- p. 899 or 968 - grafted kidney usually begins to function immediately. Sometimes adequate functioning is delayed a few days and hemodialysis may be performed until good function is established.

D. Complications
1. Graft rejection - the major postoperative complication is graft rejection. The reaction is stimulated by foreign histocompatibility antigens. Rejection may be hyperacute (within 48 hours of graft), acute - 6 wks. to 2 yrs later, or chronic. Chronic manifestations mimic CRF. Acute manifestations include fever, graft tenderness at the site of the transplanted kidney, anemia, and malaise. Urography, renal scan, echo, and CT scan are used for diagnosis. Azathioprine, cyclosporine and prednisone are used for maintenance.
2. Immunosuppressive therapy consequences
   a. Increased susceptibility to infection
   b. Increased risk of malignancy
   c. Degenerative bone disease
3. Infection - a potential problem and is the most life-threatening in the early transplant period. Usually urinary infections, pneumonia, sepsis.
5. Cardiovascular - hypertension in 50-60%.
6. Respiratory - caused by fungi or bacteria. CMV and Pneumocystis very serious.
8. Integumentary - skin carcinomas. Wound healing may be slow (steroids).
9. Death - cardiovascular deaths remain the leading cause of death in the late transplant period. MI, stroke, heart failure.

E. Nursing management
1. Assessment pre-procedure, including psychological.
2. Support donor as well as recipient if donor living.
3. Ensure no infection in either person pre-op. Bil. nephrectomy may be performed first to remove infected or polycystic kidneys.
4. Post-procedure, a major task is observing for, recognizing, and treating infections rapidly. Preventive respiratory treatment is essential. Coughing and deep-breathing exercise are started immediately. Wound care is done using strict aseptic technique. Oral hygiene important - (oral bugs). Immunosuppressive medications may be expensive!!
5. Peritonitis - watch for post-op infection, to include sepsis - frequently monitor blood pressure, pulse, respiration, CVP, weight hourly intake and output. Ensure lab studies drawn. Manage IV fluids carefully.
V. Renal cancer - at least 85% of all renal tumors are malignant.
   A. Pathophysiology - renal cell carcinoma is the most frequent type of tumor; it accounts for 90% of all kidney neoplasms. Tumor growth begins in the renal cortex and usually continues for some time before it produces symptoms. Can grow very large and compress renal parenchyma. The lungs and mediastinum are the most frequent sites for metastasis.
   B. Staging - prognosis partially depends on the stage at time of treatment. Five year survival rates for stage I are about 65%; stage II, 40%; stage III or IV - rare. 10 year survival rates: stage I, 40%; stage II, 35%.
      1. Stage I: encapsulated
      2. Stage II: tumor has invaded perineal fat
      3. Stage III: tumor extends into renal vein or regional lymphatics
      4. Stage IV: tumor has metastasized.
   C. Clinical manifestations and diagnostic findings - as many as 35% have metastasis when diagnosis is made.
      1. Common symptoms - hematuria, flank pain, palpable abdominal or flank mass. Episode of hematuria may occur as much as 9 mos. before pain noted and 14 mos. after pain noted before diagnosis actually made. This is partly because the symptoms are episodic and may not occur again for some time.
      2. Other symptoms - fever, weight loss and cachexia, fatigue, hypertension, amyloidosis, thrombophlebitis, anemia, erythrocytosis, hypercalcemia, abnormal serum liver profile and an elevated sed rate.
      3. Diagnosis - IVP most helpful. Echo and CT some help. Renal biopsy provides most definitive info.
   D. Surgical management - radical nephrectomy. Radiation and chemo may also be used. If tumor is in renal pelvis, nephroureterectomy done. Fol bilateral disease, partial nephrectomy performed. If bilateral total nephrectomies performed, pt. placed on dialysis. More than a year transpires before pt. considered for renal transplant. Tumor may be shrunken preop with radiation or renal artery embolization (reduces tumor’s blood supply and risk for hemorrhage). Embolization may be used to control hemorrhage in inoperable kidney. Preop preparation also includes adequate hydration to ensure excretion of waste products. Patient and family emotional support necessary.
   E. Nursing management of surgical client
      1. One of the biggest challenges is reestablishing effective breathing pattern postoperatively!!! Incision is so close to diaphragm that deep breathing and coughing are difficult. Liberal use of narcotics to relieve pain and external mechanical support such as a pillow to support chest and abdomen for deep-breathing and coughing may help. Incentive spirometer provides measurable information on effectiveness of deep-breathing.
      2. Urinary output must be carefully monitored. Meticulous cath care.
3. Paralytic ileus - begin oral intake only after adequate bowel function reestablished. Encourage early ambulation.
4. Keep client and family informed as much as possible to reduce anxiety.

F. Medical management
1. Radiation
2. Chemotherapy - vinblastine seems best but only 25% effective.
3. Immunotherapy - such as interleukin-2.

VI. Renal trauma - traffic accidents and falls are the most common cause of injury. Penetrating injuries from bullets and knives also occur.
A. Clinical manifestations and diagnosis - 1st clue - type of injury client has suffered. Hematuria is a cardinal manifestation - found in 80% of cases. Other findings include shock, flank pain, and the development of palpable mass in the affected flank. Paralytic ileus may also occur. Diagnosis is by KUB, IVP, retrograde pyelography, renal scan, echo, CT scan, and renal arteriography.
B. Classifications - picture p. 867 or 931 - Black.
1. Contusion with intrarenal hemorrhage: hematoma confined to parenchyma
2. Minor laceration - rupture with subcapsular hemorrhage
3. Major laceration - rupture into the renal pelvis
4. “Fractured” kidney - shattered rupture - hemorrhage throughout kidney
5. Vascular (pedicle) injury - kidney may not survive.
C. Complications - high risk of sepsis. Also hypertension from fibrosis, renal artery thrombosis, arteriovenous aneurysms.
D. Care
2. Categories 3,4,5 - surgery probably necessary - nephrectomy or repair depending on whether repair possible. Vascular injuries must be corrected within 18 hours if kidney to survive.
E. Nursing management - as necessary