Endocrine Alterations

Imad T. Asmar
BSN, MSN Clinical Nursing Specialist

23-5-2021

Hyperglycemic Crises

- DKA and HHS are endocrine emergencies.
- Historically, DKA was described as the crisis state in type 1 DM, whereas HHS was thought to occur in type 2 DM.
- Now ,DKA and HHS are increasingly being seen concurrently in the same patient

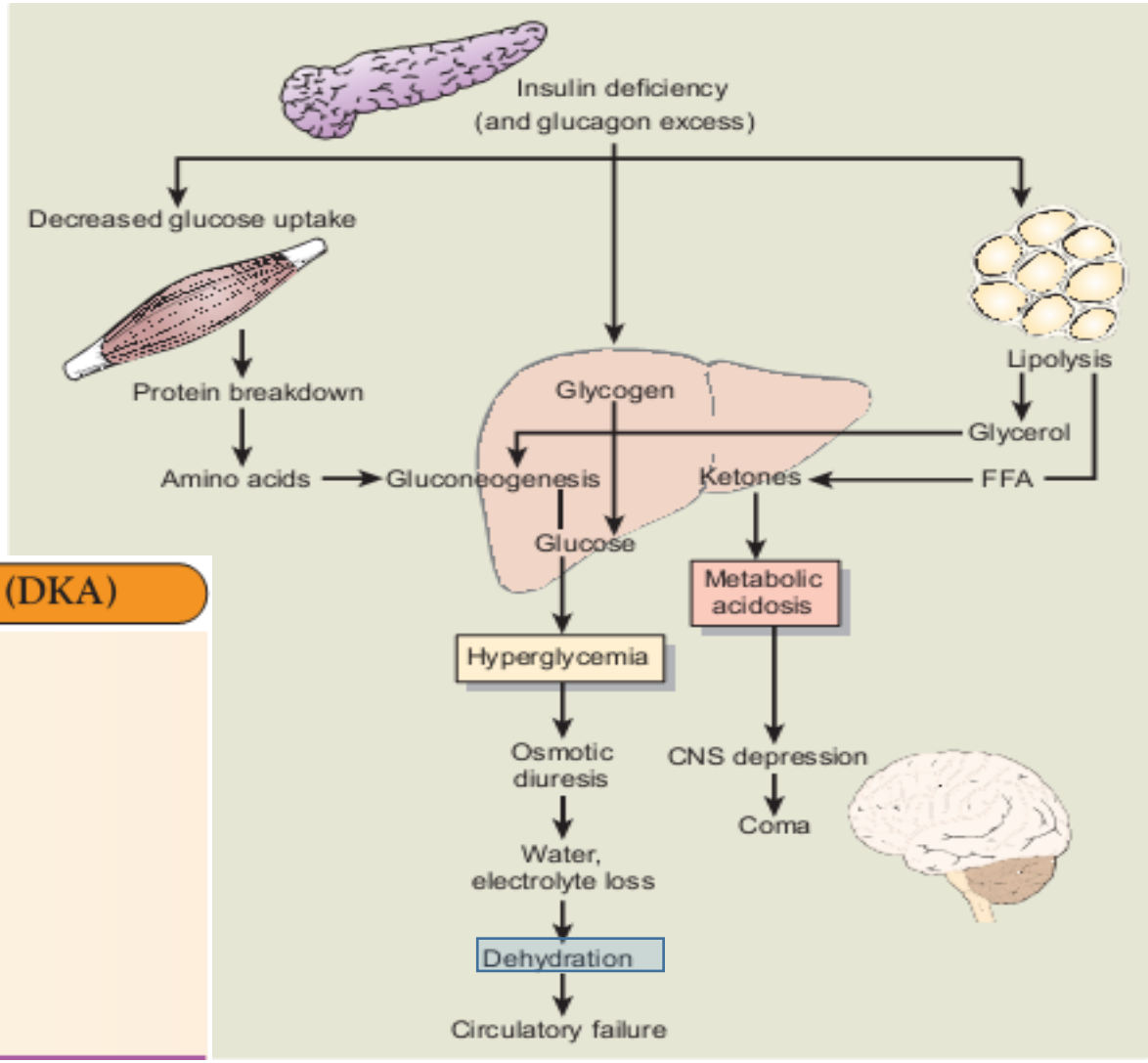
Diabetic Ketoacidosis

- Diabetic ketoacidosis (DKA) is a critical illness that manifests with severe hyperglycemia, metabolic acidosis, and fluid and electrolyte imbalances.

- DKA results from severe insulin deficiency that leads to the disordered metabolism of proteins, carbohydrates, and fats.

- The concomitant elevation of counter-regulatory hormones such as growth hormone (GH), cortisol, epinephrine, and glucagon exacerbates the condition, leading to further hyperglycemia and hyperosmolality, ketoacidosis, and volume depletion.

Pathophysiology of DKA



BOX 44-9 Signs of Diabetic Ketoacidosis (DKA)

- Hyperventilation
- Kussmaul's respirations and "fruity" breath
- Lethargy, stupor, coma
- Hyperglycemia
- Glycosuria
- Volume depletion
- Hyperosmolality
- Increased anion gap (>7 mEq/L)
- Decreased bicarbonate (<10 mEq/L)
- Decreased pH (<7.4)

Laboratory studies

Possible findings include:
hyperosmolality,.
increased anion gap (>7 mEq/L).
decreased bicarbonate (<10 mEq/L) and decreased pH (<7.4).
The serum glucose may range from 300 to 800 mg/dL or higher.
Sodium, potassium, creatinine, and BUN levels are all elevated.
Magnesium and phosphate may also be high.
The key diagnostic feature of DKA is the presence of serum ketones

Management

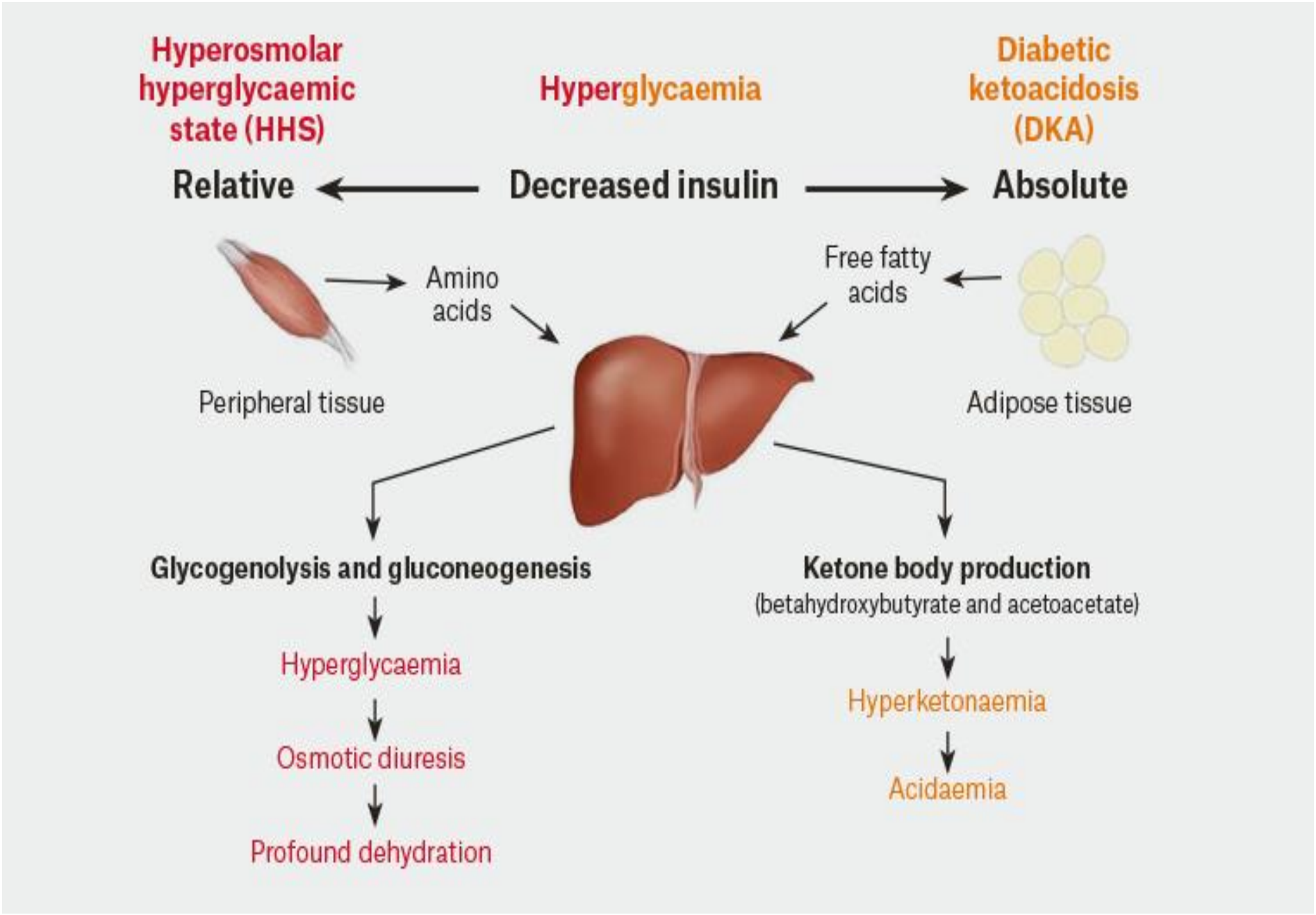
Treatment goals for the patient with DKA include the following:

- Improve circulatory volume and tissue perfusion.
- Correct electrolyte imbalances.
- Decrease serum glucose concentration.
- Correct ketoacidosis.
- Determine precipitating events.

Hyperosmolar Hyperglycemic State

- A marked hyperglycemia and hyperosmolality without ketoacidosis
- HHS has a higher mortality rate than any other complication of diabetes.
- the mechanisms of disease are the same as for DKA.

➢ A reduction in circulating insulin coupled with the effects of counter-regulatory hormones such as cortisol and epinephrine leads to the development of hyperglycemia and the extreme hyperosmolar state.


Hyperosmolar hyperglycaemic state (HHS)
Hyperglycaemia
Diabetic ketoacidosis (DKA)
Relative
Decreased insulin
Absolute
Amino acids
Free fatty acids
Peripheral tissue
Adipose tissue
Glycogenolysis and gluconeogenesis
Hyperglycaemia
Osmotic diuresis
Profound dehydration
Ketone body production
(betahydroxybutyrate and acetoacetate)
Hyperketonaemia
Acidaemia

Laboratory studies

- A blood glucose level greater than 600 mg/dL. Glucose can be in excess of 2,000 mg/dL.
- Serum osmolality is extremely high (>310 to 320 mOsm/kg)
- Acidosis is not present or is very mild. In HHS, the anion gap attributable to ketoacidosis usually is less than 7 mEq/L. The patient may present with azotemia, hyperkalemia, and lactic acidosis

Management

- Therapy for HHS is directed at correcting the volume depletion, controlling hyperglycemia, identifying the underlying cause of HHS and treating it.
- The volume depletion is usually greater in HHS than in DKA.
- Rapid rehydration is more cautiously carried out because of the fragile state of the patient, who often has comorbidities.
- It is necessary to give low-dose insulin by continuous infusion
- Investigation of the underlying cause of HHS

	DKA	HHS
Glucose, mg/dl	250-600	600-1200
Sodium meq/L	125-135	135-145
Potassium	Normal to↑	Normal
Osmolality mosm/ml	300-320	330-380 (>350)
Plasma ketones	++++	+/-
Serum bicarbonate	<15meq/L	Normal to slightly ↓
Arterial pH	6.8-7.3	>7.3
Arterial pCO_2	20-30	Normal
Anion gap	↑	Normal to slightly↑

Harrison's Principles of Internal medicine 19th edition

key differences between hyperosmolar hyperglycemic nonketotic state vs. diabetic ketoacidosis

	HHS	DKA
Primary physiologic abnormality	Hypertonicity	Ketoacidosis
Primary treatment	Controlled rehydration	Insulin & glucose
Risks encountered during treatment	Hypokalemia Hypophosphatemia Hypoglycemia Younger patients: cerebral edema	Hypokalemia Hypophosphatemia Hypoglycemia
Key parameter to monitor during tx	Serum osmolality	Anion gap
Time required for disease to develop	Days-weeks	Hours-Days
Patients affected	Usually type-2 DM Often older	Usually type-1 DM Often younger
Epidemiology	True HHS is relatively rare (often over-diagnosed)	Extremely common

Fluid replacement

- In DKA, the typical water deficit approximates 100 mL/kg, and it may be as high as 200 mL/kg in HHS
- Fluid replacement usually starts with an initial bolus of 1 L of 0.9% NS. This is followed by an infusion of 15 to 20 mL/kg during the first hour.
- IV fluids are rapidly infused until the patient's blood pressure and serum sodium level normalize

Insulin therapy

- An initial IV bolus of 0.1 units/kg of regular insulin is administered, followed by a continuous infusion of 0.1 units/kg per hour to achieve a steady decrease in serum glucose levels of 50 to 75 mg/dL per hour

Hypoglycemia

- A hypoglycemic episode is defined as a decrease in the plasma glucose level to less than 70 mg/dL and is sometimes referred to as insulin shock or insulin reaction
- Patients receiving insulin therapy must be closely monitored for hypoglycemia

TREATMENT OF HYPOGLYCEMIA

- Mild Hypoglycemia-Patient is completely alert. Symptoms may include pallor, diaphoresis, tachycardia, palpitations and hunger .Patient is able to drink

Treatment: 15 g of carbohydrate by mouth

- Moderate Hypoglycemia- Patient is conscious, cooperative, and able to swallow safely. Symptoms may include difficulty concentrating, confusion, slurred speech, or extreme fatigue. Blood glucose is usually less than 55 mg/dL. Patient is able to drink.
- Treatment: 20 to 30 g of carbohydrate by mouth

- Severe Hypoglycemia -Patient is uncooperative or unconscious. Blood glucose is usually less than 40 mg/dL or patient is unable to drink
- Treatment with intravenous access: 12.5 g of DW 50%
- Treatment without intravenous access: 1 mg of glucagon subcutaneously

ADRENAL INSUFFICIENCY

- In adrenal insufficiency (AI), the cortex does not make enough steroid hormones
- Primary AI, also called Addison's disease. In this rare condition, the adrenal glands do not work properly and cannot make enough cortisol (a “stress” hormone)
- Secondary AI ,results when the pituitary gland, does not signal the adrenal glands to make cortisol.

Signs and symptoms

- Extreme fatigue
- Weight loss and decreased appetite
- Darkening of your skin (hyperpigmentation)
- Low blood pressure, even fainting
- Low blood sugar (hypoglycemia)
- Nausea, diarrhea or vomiting (gastrointestinal symptoms)
- Abdominal pain
- Muscle or joint pains
- Irritability
- Depression or other behavioral symptoms

Adrenal crisis

- Is a life-threatening absence of cortisol and aldosterone (mineralocorticoid). A deficiency of cortisol results in decreased production of glucose, decreased metabolism of protein and fat, decreased appetite, decreased intestinal motility and digestion, decreased vascular tone, and diminished effects of catecholamines.

Nursing and Medical Interventions

- Treatment of adrenal crisis include identifying and treating the precipitating cause, replacing fluid and electrolytes and replacing hormones

Diabetes Insipidus / DI

Definition

- Conditions R/T decrease production of ADH, or decrease renal response to ADH, that could be transient or chronic.

Etiology and Pathophysiology

- Central DI occurs when any organic lesion of the hypothalamus, or posterior pituitary interferes with ADH synthesis, transport, or release.
- Brain tumors, pituitary or other cranial surgery, closed head trauma, central nervous system (CNS) infections, and vascular disorders may cause DI.

Classifications

- Neurogenic/central = lesions of hypothalamus.
- Nephrogenic = decrease response of ADH by kidney.
- Dipsogenic = psychologic increase water intake.

Clinical manifestations

- Polyurea = 5-20 L/d + nocturia
- Polydepsia

Hypovolemic shock

- Hypernatremia

Clinical manifestations

- Weight loss, poor tissue turgor, hypotension, tachycardia, constipation, shock, irritability and mental dullness and coma.
- These symptoms are related to rising serum osmolality and hypernatremia.

Diagnosis

- Identification of the cause, A complete history and physical exam
- Urine osmolality < 100 mmol/kg
- SG < 1.005
- Serum osmolality > 295 mmo/kg
- A water deprivation test is usually done to confirm the diagnosis of central DI

Management of DI

- Fluid replacement
- Hormonal replacement of ADH =DI central
- Treatment of the cause in NDI = diuretics + Na intake, NSAID (Indocin) increase response of kidney to ADH

NURSING MANAGEMENT

- Provide fluids orally or IV
- I &O charting + Wt measurements
- Administer desmopressin acetate & assessment of S.E
- For chronic DI long term therapy of DDAVP (oral/intranasal)
  - teach signs of overdose- headache, nasal irritation, nausea
  - daily Wt
  - follow-up

Syndrome of Inappropriate ADH SIADH

- Over production of ADH
- ADH (vasopressin) is synthesized in the hypothalamus and stored in the posterior pit. Gland. Plays a major role in regulation of water balance and osmolarity.

Characterized by

- Fluid retention, sudden Wt gain
- Serum hypoosmolality
- Dilutional hyponatremia, Na below 125 mEq/L

Syndrome of Inappropriate ADH SIADH

Causes:

1. Malignant tumors
2. CNS disorders= head injuries, CVA, brain tumors, meningitis, SLE ….
3. Drugs = antiepileptics, opioids, diuretics
4. Other conditions: hypothyroidism, lung infection, COPD, mechanical ventilation

Syndrome of Inappropriate ADH SIADH

Clinical manifestations

- Hypervolemia+hyponatremia = cerebral edema= seizure = coma

Diagnostic studies

- Na < 134 mEq/L , urea + cr., Hb, Hct
- Serum osmolality < 280 mmol/kg
- Urin SG > 1.005

Syndrome of Inappropriate ADH SIADH

Management and nursing care

- Treat the cause
- Restrict fluids (800-1000ml/d) + diuretics
- Flat head of bed to increase venous return = decrease ADH production
- Provide safety measures and seizure precautions
- I&O, wt, k-replacement
- Frequent oral hygiene

Thyroid Storm

- A complication of preexisting hyperthyroidism (thyrotoxicosis)
- Excessive amount of thyroid hormone
- Cause:

  - toxic diffuse goiter (Graves' disease) it is an autoimmune disease
  - amiodarone causes thyroid dysfunction in 14% of patients
  - excessive ingestion of thyroid hormone
  - excessive pitutary TSH
  - thyroiditis

Thyroid Storm

- Thyroid storm also called thyroid crisis.
- It is a life threatening condition.
- Major stressors can precipitate thyroid storm in the hyperthyroid patient

Thyroid Storm

- Pathophysiology

  - thyroid hormone increases cellular oxygen consumption,

  - excess metabolism generate heat.

  - temperature rise to as high as 41.

  - cellular oxygen demand increased

  - cardiac response to increase CO, hypertension and tachycardia, tremors, fatigue

Thyroid Storm

Pathophysiology

- Catabolism and a negative nitrogen balance occur. Metabolic acidosis developed, intestinal peristalsis increases resulting in diarrhea, N, V, dehydration and WT loss.
- Muscular contraction increases (hyper-reflexia of hyperthyroidism)

Thyroid Storm

- Clinical manifestations

  - tachycardia, PVCs, palpitation, CHF, pulmonary edema, cardiogenic shock.

  - nervousness, muscle weakness, confusion, convulsion, heat intolerance, diaphoresis, fine tremor.

  - N, V, diarrhea, weight loss, increased appetite

  - hyperthermia, hypercalcemia, hyperglycemia, hypoalbuminemia

Thyroid Storm

- Medical management
  - To prevent cardiovascular collapse
    - beta-blockers
  - To reduce hyperthermia
    - hypothermic measures
    - aspirin is contraindicated because they prevent protein binding of T_3 to T_4, increasing the free active thyroid hormone
  - To reverse dehydration

Pharmacological management

- Drugs that block thyroid synthesis

  - propylthiouracil and methimazole, administered orally or via NGT.

  - propylthiouracil also block the conversion of T_4 to T_3
- Drugs that block release of thyroid hormone.

  - inorganic iodine
- Drugs that block catecholamine effect.

  - beta adrenergic such as propranolol

Thyroid Storm

- Nursing management
  - safe adminstration of drugs
  - monitor the effect of drugs
  - normalize body temp
  - rehydration, and balance electrolytes
  - patient education

Hypothyroidism

- One of the most common medical disorders in the U.S.

- Affects 8% of women and 2% of men over 50

Hypothyroidism
Etiology and Pathophysiology

- Results from insufficient circulating thyroid hormone
- Can be primary or secondary
- May also be transient to thyroiditis or discontinuance of thyroid hormone therapy

Hypothyroidism
Etiology and Pathophysiology

- Iodine deficiency is the most common cause worldwide and is most prevalent in iodine-deficient areas

- In places where iodine intake is adequate, the primary cause in the adult is atrophy of the gland

Hypothyroidism
Etiology and Pathophysiology

- Atrophy is the end result of Hashimoto's thyroiditis and Graves' disease

- Also may develop as a consequence of treatment for hyperthyroidism, specifically the removal of the thyroid glands, or radioactive iodine therapy

Hypothyroidism
Clinical Manifestations

- Cardiovascular System
  - Increased capillary fragility
  - Decreased rate and force of contraction
  - Cardiac hypertrophy
  - Muffled heart sounds

Hypothyroidism
Clinical Manifestations

- Cardiovascular System (cont.)
  - Anemia
  - Tendency to develop CHF, angina, and MI

Hypothyroidism
Clinical Manifestations

- Respiratory System
  - Dyspnea
  - Decreased breathing capacity

Hypothyroidism
Clinical Manifestations

- GI System
  - Decreased appetite
  - Nausea and vomiting
  - Weight gain
  - Distended abdomen

Hypothyroidism
Clinical Manifestations

- Integumentary System
  - The composition of skin changes as hyaluronic acid deposits (gel-like substance capable of holding large amounts of fluid) giving rise to a full, puffy apperance of face, hands, and feet
  - The facial expression is dull and mask-like
  - Dry, thick, elastic, cold skin
  - Thick, brittle nails
  - Dry, sparse, course hair
  - Pallor
  - Puffy face

Hypothyroidism
Clinical Manifestations

- Musculoskeletal System
  - Fatigue
  - Weakness
  - Muscular aches and pains
  - Slow movements
  - Arthralgia

Hypothyroidism

Clinical Manifestations

- Nervous System
  - Apathy, Lethargy, Fatigue, Hoarseness
- Pulmonary system:

  - pleural effusion, increse PCO2, respiratory acidosis, sleep apnea
- Kidney and electrolyte balance

  - renal blood flow is reduced and reduced GFR, decreased urin SG, and urine osmolality, Na decreased

Hypothyroidism

Clinical Manifestations

- Nutrition and elemination

  - decreased gastric motility, paralytic illius, decreased absorption

- Thermoregulation

  - heat production decreased, inability to maintain body heat, sweating diminish.

- anemia

Hypothyroidism

Clinical Manifestations

- Nervous System
  - Slowed mental status
  - Slow, slurred speech
  - Stupor, coma
  - Paresthesia
  - Anxiety and depression

Hypothyroidism
Clinical Manifestations

- Reproductive System
  - Prolonged menstrual periods or amenorrhea
  - Decreased libido
  - Infertility

Hypothyroidism

Clinical Manifestations

- Increases susceptibility to infection
- Sensitivity to narcotics, barbiturates, anesthesia
- Cold intolerance
- Goiter

Hypothyroidism

Complications

- Mental sluggishness
- Drowsiness
- Lethargy progress gradually or suddenly to impairment of consciousness or coma

Hypothyroidism
Complications

- Myxedema coma
- Can be precipitated by infection, drugs, cold, or trauma
- Characterized by subnormal temperature, hypotension, and hypoventilation

Hypothyroidism
Diagnostic Studies

- Laboratory tests that measure TSH and free T_4
  - Low T4, T3, and high TSH
- Other abnormal findings are elevated cholesterol and triglycerides, anemia, and increased creatine kinase

Hypothyroidism/ Myxedema

- Medical management

  - levothyroxine 100-500 mcg IV then followed with a loading dose of 75-100 mcg daily.