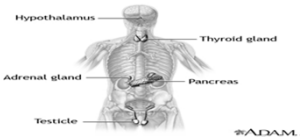
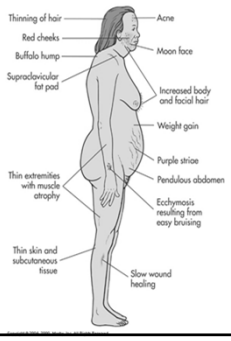


ENDOCRINE DISEASE

Dr. Leonece D. Myers, DNP, MSN, RN
APRN, CFNP, FNP-BC, CCRN

- Hypoparathyroidism
- Hyperparathyroidism
- Cushing Syndrome
- Addison's Disease
- Pheochromocytoma



HYPERPARATHYROIDISM

- Increase secretion of parathyroid hormone (PTH). PTH regulates calcium, phosphate levels, renal tubular reabsorption of calcium, and activation of vitamin D.
- Oversecretion of PTH is associated with increase serum calcium levels

TYPES OF HYPERPARATHYROIDISM

- Primary
- Secondary
- Tertiary

MANIFESTATIONS

- Decreased bone density
- Cyst formation
- General weakness
- Hypercalciuria
- Calculi formation
- Osteoporosis
- Fractures

MANIFESTATIONS

- Cardiovascular symptoms
- Neurologic
- Musculoskeletal
- Gastrointestinal
- Renal
- Integumentary

DIAGNOSTIC STUDIES

- PTH
- Serum calcium
- Elevated urine calcium
- Serum phosphate
- Electrolytes (Na/Cl/Cr/amylase)
- Bone density measurements
- X-rays, UTZ, MRI, thalium scan

TREATMENT

Surgical
a. Parathyroidectomy (partial/complete)
b. Autotransplantation

Nonsurgical
a. Monitor electrolytes
b. Diet
c. Phosphorus supplements (Bisphosphonates)
d. Drugs (Estogen, Progestin,Phosphate, Calcimimetic)
e. Ambulation

COMPLICATIONS

- Tetany
- Hemorrhage
- Laryngospasms

HYPERCALCEMIC CRISIS

- Serum calcium level > 15 mg/dL
- Results in neurologic, cardiovascular, and renal symptoms
- Treatment involves rehydration with large volumes
- Diuretics: promote renal excretion of Ca++
- Phosphate therapy
- Cytotoxic agents: Mithramycin
- Calcitonin
- Dialysis

INTERVENTIONS

- I&O
- IV calcium gluconate
- Monitor electrolytes
- Frequent monitoring for Chvostek' Trousseau's signs.
- Mobility to promote bone calcification

HYPOPARATHYROIDISM

- Inadequate circulating PTH
- Hypocalcemia due to lack f PTH to maintain serum calcium levels.
- Pseudohypoparathyroidism due to resistance of PTH a the cellular level. There is a genetic defect resulting in hypocalcemia in spite of normal or high levels .
- Most common cause is iatrogenic

MANIFESTATIONS

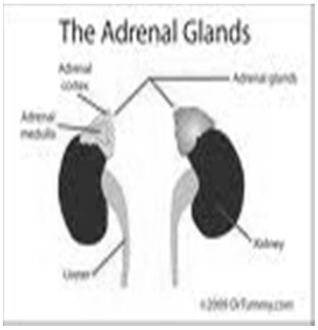
Cardiovascular symptoms
Neurologic
Gastrointestinal
Renal
Musculoskeletal
Integumentary

MANIFESTATIONS

- Numbness and tingling (lips, fingers, feet)
- Tonic spasms of smooth muscles and skeletal
- Dysphagia
- Laryngospasm
- Decreased serum calcium and PTH levels
- Increased serum phosphate

TREATMENT

IV calcium chloride, gluconate, or gluceptate
ECG monitoring
Rebreathing
Lowering pH
Oral calcium supplements (1.5-3 g/day)
Vitamin D
Diet (high calcium)
EKG monitoring during calcium administration



DISORDERS OF THE ADRENAL CORTEX

- Glucocorticoids: Cortisol
- Mineralocorticoids: Aldosterone
- Androgens

CUSHING SYNDROME

- Clinical abnormalities caused by an excess of corticosteroids, particularly glucocorticoids.

ETIOLOGY

- Iatrogenic administration of exogenous corticosteroids. (Most common cause)
- ACTH-secreting pituitary tumor
- Adrenal cortisol secreting tumor
- Excess secretion of ACTH from carcinoma of the lung or other malignant growth outside the pituitary or adrenal glands

MANIFESTATIONS

- Weight gain
- Truncal obesity
- Moon face
- "Buffalo hump"
- Hyperglycemia
- Protein wasting
- Muscle weakness
- Osteoporosis
- Pathologic c fractures
- Bone and back pain
- Thin fragile skin
- Purplish strae
- Hypervolemia, HTN
- Edema
- Inhibition of immune response
- Na+ and water retention
- Hirsutism
- Menstrual irregularities

DIAGNOSTIC STUDIES

- 24 hour urine for free cortisol
- Dexamethsone suppression test
- Plasma cortisol
- CT scan, MRI
- Plasma ACTH levels
- CBC, electrolytes
- Eye exam

TREATMENT

Surgery

1. Transsphenoidal for pituitary tumor
2. Adrenalectomy for malignant tumors

Radiation

Drug therapy

1. Mitotane : "Medical adrenalectomy"
2. Ketconazole, Metyrapone, Cytaden

TREATMENT

- Gradual discontinuance of corticosteroids therapy
- Reduction of the corticosteroid dose
- Conversion to an alternate-day regimen

COMPLICATIONS

- Cardiovascular disease
- Diabetes Mellitus
- Infection
- Thromboembolic phenomena (polycythemia)
- Changes in appearance (Hisutism, gynecomastia)

INTERVENTIONS

- Monitoring vital signs
- Daily weights
- Monitor glucose
- Emotional support
- Control HTN, hyperglycemia, hypokalemia
- Diet (high protein)

POSTOPERATIVE

- Monitor for postoperative hemorrhage
- Monitor BP, fluid balance and electrolyte
- High doses of corticosteroids (SoluCortef)
- Monitor for infection
- Monitor I&O
- Monitor urine cortisol levels

EDUCATION

- Medical alert bracelet
- Avoid exposure to extremes of temperatures, infections, and emotional disturbances
- Adjustment corticosteroid replacement therapy
- Lifetime replacement

ADRENOCORTICAL INSUFFICIENCY

- Hypofunction of the adrenal cortex
- Primary: Addison's disease
- Secondary: Lack of ACTH secretion, may be due to pituitary disease or suppression of the hypothalamic-pituitary axis.

ADDISON's DISEASE

The most common cause is due to an autoimmune response.

Adrenal tissue is destroyed by antibodies against the patient own adrenal cortex.

ETIOLOGY

- Infarction
- Fungal infections
- Acquired Immunodeficiency Syndrome
- Metastatic cancer
- Adrenal hemorrhage (anticoagulant therapy)
- Chemotherapy
- Ketoconazole therapy
- Bilateral adrenalectomy

MANIFESTATIONS

- Weight loss
- Hyperpigmentation
- Vitilligo, alopecia
- Hypotension
- Hyperkalemia
- Hyponatremia
- Anorexia
- Fatigability
- Diarrhea
- Irritability
- Depression
- Decreased axillary and pubic hair
- Decrease muscle size and tone
- Shock
- Anemia

DIAGNOSTIC STUDIES

- ACTH-stimulation test
- Electrolytes
- Glucose
- CBC
- BUN, creatinine
- EKG (Peaked T waves)
- CT scan, MRI

TREATMENT

- Replacement therapy (Hydrocortisone)
- During stress need to increase glucocorticoid (cortisol) must be increased to prevent Addisonian crisis.
- Mineralocorticoid replacement (Floinef)
- Increase salt intake
- Patient education

MANIFESTATION OF ADDISONIAN CRISIS

- Hypotension
- Tachycardia
- Dehydration
- Hyponatemia, hyperkalemia, hypoglycemia
- Fever, confusion
- Weakness
- Shock
- Circulatory collapse

COMPLICATIONS ADDISONIAN CRISIS

- A life threatening emergency caused by insufficient adrenocortical hormones or a sharp decrease in these hormone.
- Triggered by stress
- After adrenal surgery
- Pituitary gland destruction
- Sudden withdrawal of corticosteroid hormone replacement therapy.

TREATMENT OF ADDISONIAN CRISIS

- High dose hydrocortisone replacement
- Large volumes of normal saline and D5W
- VS q 30 minutes-4 hour for first 24 hrs
- I&O
- Daily weights
- Protect from light and environmental extremes

COMPLICATIONS OF CORTICOSTEROIDS

- Hypertension
- Suppression of inflammation and immune response
- Inhibit the antibody response to vaccines.
- Predisposition to PUD
- Mood and behavior changes
- Redistribution of fat
- Healing delayed
- Osteoporosis

PHEOCHROMOCYTOMA

- A catecholamine-secreting neoplasm associated with hyperfunction of the adrenal medulla.
- A rare condition characterized by a tumor of the adrenal medulla that produces excessive catecholamines (epinephrine, norepinephrine)
- 90% tumors are benign

MANIFESTATIONS

- Hypertension
- Hypermetabolism
- Postural hypotension in a hypertensive patient
- Hyperglycemia (polyuria)
- Headaches and visual disturbances
- Tremors, pallor, perspiration, face flushing
- Diarrhea, vomiting, abdominal pain
- Emotional changes, psychotic behavior

DIAGNOSTIC STUDIES

- VMA and metanephrine
- Elevated epinephrine and norepinephrine in serum and urine
- CT scan and MRI of the adrenal gland
- Clonidine suppression test (used to distinguish essential HTN from phenochromocytoma)

TREATMENT

- Alpha-adrenergic blocking agents (Retgitine,
- Phenoxybenzamine, Terazosin, Prazosin)
- Catecholamine synthesis inhibitor (Metyrosine)
- Calcium channel blocker (Cardene)
- Surgery : adrenalectomy

INTERVENTIONS

- Bed rest, elevate HOB 45 degrees
- Calm quiet environment
- Eliminate stimulants(caffeine)
- Sedatives to promote relaxation
- Encourage oral fluids and maintain IV infusion
- Monitor vital sign, ECG, Neurologic status
- I&O, labs (BUN, creatinine, and glucose)