Endocrine

Jennifer MacDermott, MS, RN, ACNS-BC, NP-C, CCRN
Clinical Nurse Specialist
Surgical Intensive Care Unit

Objective
- Identify abnormal assessment finding in a patient with endocrine dysfunction.
- Describe types of endocrine dysfunction.
- Describe the standards in caring for a patient undergoing endocrine surgery.

Endocrine Facts
- Endocrine organ/gland manufactures and secretes hormones
- Body functions: reproduction; growth and development; maintenance of homeostasis; and energy production, utilization, and storage during metabolic processes
- Negative feedback loop

Assessment of the Patient with Endocrine Dysfunction

Physical Assessment
- Changes in skin texture (hypothyroidism/hyperthyroidism)
- Exophthalmos (hyperthyroidism)
- Changes in physical appearance
  - Facial hair
  - ‘Moon face’ (Cushing's disease)
  - ‘Buffalo hump’ (Cushing's disease)
  - Obesity of the trunk with thinness of extremities (Cushing's disease)
  - Increased size of feet and hands (Cushing's disease)
- Edema
- Blood pressure
  - Elevated (hyperfunction of adrenal cortex/hyperparathyroidism)
  - Decreased (hypofunction of adrenal cortex)
Serum Laboratory Studies

• ADH
  – Normal: 1 – 5 pg/ml
  – Controls water resorption in kidney
• Glucose
  – Normal: 70 – 105 mg/dL
  – Controlled by insulin and glucagon release
  – Abnormal results
    • Hyperglycemia
    • Hypoglycemia

Serum Laboratory Studies

• Sodium (Na+)
  – Normal: 136 – 145 mg/dL
  – Result of balance between dietary sodium intake and renal excretion; aldosterone, natriuretic hormone, and antidiuretic hormone all play a role
  – Abnormal results
    • Hypernatremia
    • Hyponatremia
• Osmolality
  – Normal: 285 – 295 mOsm/kg
  – Measure of concentration of dissolved particles in blood
  – Abnormal results
    • Hyperosmolality
    • Hypoosmolality

Urine Laboratory Studies

• Glucose
  – Normal: negative (random specimen)
  – Abnormal result is a consequence of blood glucose levels that exceed the capability of renal threshold and results in glucose spilling into the urine
• Ketones
  – Normal: negative
  – Abnormal result occurs as a result of fatty acid catabolism
• Osmolality
  – Normal: 50 – 1200 mOsm/kg (random specimen)
  – Evaluation of ability of kidney to concentrate urine
  – Abnormal results
    • Hyperosmolality
    • Hypoosmolality

Endocrine Dysfunction

Acute Hypoglycemia

• Serum glucose <50 mg/dL
• High risk populations
  – Diabetic patients taking insulin
  – Children and pregnant women with type 1 diabetes
  – Elderly patients with diabetes
• Causes
  – Excess insulin or oral hypoglycemics
  – Decreased exogenous insulin requirement
  – Underproduction of glucose
  – Rapid utilization of glucose

Acute Hypoglycemia

Clinical Manifestations

<table>
<thead>
<tr>
<th>Activation of Sympathetic Nervous System</th>
<th>Inadequate Glucose Supply to Neural Tissues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nervousness/apprehension</td>
<td>Headache</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Restlessness</td>
</tr>
<tr>
<td>Palpitations</td>
<td>Difficulty speaking/thinking</td>
</tr>
<tr>
<td>Pallor</td>
<td>Visual disturbances</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>Paresthesia</td>
</tr>
<tr>
<td>Dilated pupils</td>
<td>Difficulty walking</td>
</tr>
<tr>
<td>Tremor</td>
<td>Altered consciousness</td>
</tr>
<tr>
<td>Fatigue</td>
<td>Coma</td>
</tr>
<tr>
<td>Weakness</td>
<td>Convulsions</td>
</tr>
<tr>
<td>Headache</td>
<td>Catatonia</td>
</tr>
<tr>
<td>Hunger</td>
<td>Acute paranoia</td>
</tr>
</tbody>
</table>
**Acute Hypoglycemia Management**

- Prolonged hypoglycemia may lead to irreversible brain damage and coma
- Mild hypoglycemia
  - Blood glucose 60 – 80 mg/dL
  - Treatment: 15g carbohydrate by mouth (4 oz sweetened carbonated beverage, 4 oz fruit juice, 1 cup skim milk, 3-4 glucose tablets)
- Moderate hypoglycemia
  - Blood glucose <60 mg/dL
  - Treatment: 20g – 30g carbohydrate by mouth
- Severe hypoglycemia
  - Blood glucose <60 mg/dL
  - Treatment: ½ amp D50 IV or 1mg glucagon SQ

**Diabetic Ketoacidosis (DKA) Pathophysiology**

- High glucose levels → shift fluid out of cells to extracellular space (osmotic diuresis) → urinary losses of water/electrolytes
- Serum glucose exceeds renal threshold → glucose excreted via kidneys (glucosuria)
- Hyperosmolar state further impairs insulin secretion and promotes insulin resistance
- GFR ↓ as result of fluid volume deficit leading to reduced glucosuria → ↑ serum glucose

**Diabetic Ketoacidosis (DKA) Clinical Manifestations**

- Flushed, dry skin
- Dry mucous membranes
- ↓ skin turgor
- Tachycardia
- Hypotension
- Kussmaul respirations
- Acetone breath
- Altered level of consciousness
- Visual disturbances/blurred vision
- Polydipsia (↑ thirst)
- Polyuria (↑ urination)
- Nausea and vomiting
- Anorexia
- Abdominal pain
- Weakness
- Headache

**Diabetic Ketoacidosis (DKA) Laboratory Studies**

- Serum glucose 300 – 800 mg/dL
- ↑ urine glucose level
- Arterial pH <7.30
- ↓ serum bicarbonate (0 – 15 mEq)
- Positive serum and urine ketones
- ↑ serum creatinine and azotemia
- Electrolytes vary with state of hydration
- Hyperkalemia/hypokalemia
- Serum hyperosmolality (average 330 mOsm/kg)
**Hyperglycemic Hyperosmolar Nonketotic Coma (HHNK)**

- Inadequate insulin secretion or action and/or decreased utilization and/or increased production of glucose
- Most commonly seen in Type 2 Diabetes, but can be seen in patients without diabetes
- Mortality 10 – 40%
- Factors leading to HHNK
  - High calorie parenteral or enteral feedings
  - Decreased thirst mechanism
  - Difficult access to fluids
  - Medications

**HHNK Pathophysiology and Clinical Manifestations**

- **Pathophysiology**
  - Develops over weeks to months
  - Hyperglycemia → shift water to extracellular space → profound dehydration
  - Insulin levels prevent lipolysis and ketone formation
- **Clinical manifestations**
  - Flushed, dry skin
  - Dry mucous membranes
  - ↓ skin turgor
  - Tachycardia
  - Hypotension
  - Shallow respirations
  - Altered level of consciousness

**HHNK Laboratory Studies**

- Serum glucose 600 – 1200 mg/dL
- Arterial pH >7.30
- Serum bicarbonate >15 mEq/L
- Azotemia
- Electrolytes vary with state of dehydration, often hypernatremic
- Serum hyperosmolality > 350 mOsm/kg
- Absence of significant ketosis

**DKA and HHNK Management**

- **Insulin therapy**
  - IV bolus 0.15 units/kg regular insulin
  - IV infusion 0.1 units/kg/hr
  - Goal: steady decrease serum glucose 50 – 75 mg/dL per hour
  - Monitor blood glucose q1hr
- **Electrolyte replacement**
  - Potassium replacement
  - Phosphorus replacement

- **Sodium bicarbonate administration**
  - Only use when serum pH <7.0
  - Only correct to serum pH 7.0
  - If necessary, should be added to hypotonic saline infusion and administered slowly
- **Patient and family education**
### Antidiuretic Hormone (ADH)
- Also known as vasopressin
- Synthesized by hypothalmus and stored in the posterior pituitary
- Release mediated by osmoreceptors in the hypothalmus
- ADH stimulation → kidneys reabsorb water → reduced urine output
- ADH suppression → water excreted via kidneys → increased urine output

### DKA and HHNK Management
- Complications
  - Cerebral edema
    - Result of rapid fluid administration
    - Result of reduction in serum glucose too quickly
  - Seizures
  - Coma

### Diabetes Insipidus
- Disorder of posterior lobe of pituitary gland
- Pathophysiology
  - Deficiency of ADH
  - Results in large volumes of water to be excreted via kidneys
- Causes
  - Idiopathic
  - Intracranial tumors, metastatic disease, lymphoma
  - Head trauma
  - Surgical ablation or irradiation of pituitary gland
  - CNS infections
  - Chronic renal disease
  - Medications causing suppression of ADH release: phenytoin, alcohol, α adrenergic agents

### Diabetes Insipidus Management
- Identify and correct underlying pathology
- Adequate fluid replacement
- Replace ADH
  - Vasopressin (Pitressin)
  - Desmopressin (DDAVP)
  - ADH (vasopressin tanate in oil)
  - Thiazide diuretics, mild salt depletion, prostaglandin inhibitors (ibuprofen, aspirin) are used to treat nephrogenic DI

### DI Clinical Manifestations and Laboratory Studies
- Clinical manifestations
  - Polyuria (excessive urination) and polydipsia (excessive thirst)
  - Excessive ingestion of fluids
  - Nocturia
  - Dry mucous membranes, poor skin turgor, decreased saliva and sweat production
  - Disorientation
  - Seizures
- Laboratory studies
  - Serum Na+ >145 mEq/L
  - Urine specific gravity 1.001 to 1.005
  - Low serum ADH level

### Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH)
- Disorder of posterior lobe of pituitary gland
- Pathophysiology
  - Excessive ADH secretion from pituitary gland
  - Unable to dilute urine → retain fluids → dilutional hyponatremia
- Causes
  - Malignant lung tumors
  - Tuberculosis or severe pneumonia
  - Head injury
  - Brain surgery or brain tumor
  - Adrenal insufficiency
  - Medications
Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH)

- Clinical manifestations
  - Reduced urine output
  - Weight gain
  - Weakness, muscle cramps
  - Fatigue
  - Difficulty breathing
  - Confusion, hemiparesis, seizures, coma
- Laboratory studies
  - Na+ <135 mEq/L
  - Low hematocrit
  - Low BUN
  - Elevated urine osmolality
  - Reduced serum osmolality

SIADH Management

- Identify and correct underlying cause
- Monitoring intake/output, daily weight, urine and serum chemistries, neurological status
- Fluid restriction
- IV saline
- Diuretics
- Correction of hyponatremia (slowly)

Endocrine Related Surgeries

- Pre-procedure care
  - Informed consent
  - Clotting studies
  - Blood typing
  - IV access
- Post-operative care
  - Vital sign assessment
  - Respiratory effort
  - Pain assessment and analgesic administration if necessary
  - Assessment of surgical dressing, reinforcement if necessary
  - Encourage ambulation
  - Patient education

Endocrine Surgery

- Hypophysectomy
  - Pituitary gland removal
  - Patient education – replacement therapy with corticosteroids and thyroid hormones
- Thyroidectomy or parathyroidectomy
  - Thyroid removal or parathyroid removal
  - Post-operative assessment for hypocalcemia and respiratory difficulties
  - Support of head during positioning to avoid tension on sutures
  - Patient should avoid talking to reduce edema to vocal cords; MD should be notified of any voice changes
- Adrenalectomy
  - Adrenal gland removal
  - Post-operative assessment for hypotension and hypoglycemia
  - Patient education – replacement therapy with corticosteroids if bilateral adrenalectomy

Questions?