Endocrine Emergencies

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Endocrine Pathophysiology

- Ductless glands-secrete hormones directly into bloodstream
- Hormones are chemical messengers-target oriented and specifically regulate cellular metabolism and function
- Function of Endocrine System-regulate growth and development, reproduction, metabolism, stress response, F&E balance, acid-base and energy balance
Negative Feedback

- When physiological effect of hormone is achieved, rate of secretion stops increasing or decreases
- Nervous and Endocrine systems strive for homeostasis
- Hypothalamus integrates both systems by initiating endocrine responses from neurotransmitter stimuli
Adrenal Glands

- Cortex Hormones
  - Glucocorticoids
    - Control utilization of carbohydrates, proteins and fats
    - Increase gluconeogenesis
    - Anti-inflammatory effects
    - Secretion controlled by ACTH
  - Mineralocorticoids
    - Increases sodium and water reabsorption
    - Release controlled by renin angiotensin system, decreased sodium and ACTH
Adrenal Glands

- Medulla Hormones
  - Effect sympathetic receptor sites
  - Epinephrine
    - Stimulates alpha and beta receptors
    - Increases rate and force of myocardial contraction, bronchodilation, tachypnea, decreased peristalsis, increases serum glucose and causes peripheral vasoconstriction
  - Norepinephrine
    - Greater effect on alpha than beta receptors
    - More severe peripheral vasoconstriction
ADH and Water Regulation

- ADH is manufactured in hypothalamus; acts on distal renal tubules and collecting ducts to increase permeability to water -> water is retained in interstitial fluid to balance water volume
ADH and Water Regulation

- Osmoreceptors shrink when ECF is hypertonic and plasma osmolality increases -> osmoreceptors are stimulated to release ADH from post pituitary -> water reabsorbed in kidney
- ECF volume deficit leads to stimulation of stretch receptors in carotid sinus, periphery and aortic arch archstimulating release of ADH
Diabetes Insipidus

- Permanent or transient deficiency in ADH

- Etiology
  - Neurogenic:
    - Hypothalamic or pituitary tumor
    - Closed head injury
    - Neurosurgery
    - Infection-meningitis, encephalitis
    - Aneurysm
Diabetes Insipidus

Etiology (continued)

Nephrogenic
- Renal diseases-polycystic kidneys, pyelonephritis
- Multiple myeloma, amyloidosis

Psychogenic
- Intake of large amounts of water

Drugs which inhibit ADH secretion and action
- Ethanol, lithium and dilantin
Diabetes Insipidus

- Clinical signs and symptoms
  - UOP 4-20 liters/24 hours
  - Dry mucous membranes
  - Dehydration, seizures HA lethargy, coma
  - Tachycardia, hypotension
  - Thirst, weight loss
  - Decreased CO and PCWP
Diabetes Insipidus

- Lab data
  - Increased serum osmolarity > 295
  - Decreased urine osmolarity < 500
  - Hypernatremia
  - Specific gravity 1.001-1.005
  - Elevated BUN and HCT
  - Water deprivation test: stimulus for ADH release (dehydration) and kidneys cannot concentrate urine—no response in neurogenic and nephrogenic DI
  - ADH Test: kidneys cannot concentrate urine with exogenous ADH—corrects neurogenic, but not nephrogenic DI
Diabetes Insipidus

Complications
- Dehydration, hypoperfusion and shock
- Dilation and hypertrophy of bladder

Goals of Therapy
- Prevent tissue hypoperfusion
- Restore circulating fluid volume and electrolyte balance
Diabetes Insipidus

**Treatment**
- Aggressive fluid replacement based on UOP with .45 NS or D5W
- Strict I and O, monitor urine specific gravity and urine and serum osmolarity
- Determine and treat cause
- Neurogenic-Vasopressin IV,IM subq, DDAVP-monitor for hypertension and chest pain
- Monitor electrolytes and signs of dehydration and shock
Syndrome of Inappropriate ADH (SIADH)

- Increased production or secretion of ADH unrelated to plasma osmolarity → increase in water reabsorption in renal tubules and increase in Na excretion → increase in total body water → hyponatremia and hypervolemia
- Failure of the negative feedback system
SIADH

Etiology
- Aneurysm or CVA
- Neoplasms-lung, brain, prostate, ovary, pancreas, leukemia, lymphoma
- Head injury, skull fracture, subdural hematoma, subarachnoid hemorrhage, post neuro surgery
- Pneumonia, lung abscess, TB, COPD, ARF
- CNS infection-meningitis, encephalitis, Guillain-Barre syndrome, AIDS
Syndrome of Inappropriate ADH (SIADH)

**Etiology**

- Hypoxia or low left atrial pressures stimulating release of ADH-asthma, pulmonary infections, heart failure, positive pressure ventilation
- Meds which increase ADH secretion or potentiate ADH
  - Chemo, thiazide diuretics, acetaminophen, amitriptyline
Syndrome of Inappropriate ADH (SIADH)

Clinical Signs and Symptoms

- Weight gain with n/v and diarrhea
- Hyponatremia → lethargy, weakness, confusion, irritability, diminished reflexes, seizures and coma
- Increased BP, HR, CVP, PCWP, S3 (CHF)
- Decreased urine output
Syndrome of Inappropriate ADH (SIADH)

Lab Data

- Hyponatremia
- Decreased serum osmolarity <275
- Increased urine osmolarity
- Increased urine specific gravity >1.030

Complications

- Fluid Overload and heart failure
- Seizures
- Coma
- Death
Syndrome of Inappropriate ADH (SIADH)

- **Treatment**
  - Restrict fluid intake and prevent water intoxication - 1000-1500 milliliters per day
  - Diuretics
  - Replace UOP and insensible loss ~ 1 liter/day
  - Goal: Water loss to exceed intake until normal osmolarity
  - Identify and treat cause
  - Correct electrolyte imbalances and K replacement
  - Seizure precautions
Syndrome of Inappropriate ADH (SIADH)

- **Treatment**
  - **Diuretics**
  - **Hypertonic saline (3%)** if severe s/s - confusion, seizures. Calculate Na deficit and replace specific volume. May give with Lasix
  - **Daily weights and strict I/O**
  - **Demeclocycline** interferes with normal ADH action on renal tubules
Acute Hypoglycemia

- Defined: Serum Glucose less than 70 mg/dl
- Symptomatic hypoglycemia usually occurs at serum glucose <50 mg/dl or a sudden drop in glucose

Symptoms include:
- Headache
- Sweating
- Impaired vision
- Dizziness
- Fast heartbeat
- Hunger
- Shaking
- Irritability
- Anxiety
- Weakness/fatigue
Hypoglycemia Etiology

- Insufficient intake
- Inadequate glucose production: excessive exercise, excessive alcohol intake
- Excessive Insulin dose
- Drugs: sulfonylurea therapy, alpha blockers, salicylates, haldol, quinidine, norpace, ethanol
Hypoglycemia: Etiology

- Insulin dose greater than current need or site absorption
- Oral hypoglycemic meds
- Insufficient oral intake-skipped meals or n/v, anorexia
- Strenuous exercise without caloric intake
- Excessive alcohol intake (inhibits gluconeogenesis)
- Weight loss decreases insulin resistance
Hypoglycemia Etiology

- Tumors: malignant or benign
- Pregnancy and immediate postpartum
- Severe liver disease
- Adrenal Insufficiency
- Post gastrectomy surgery
- Beta blocker therapy - may not exhibit s/s of hypoglycemia and BB may impair recovery from hypoglycemia by inhibiting glycogenolysis
Hypoglycemia Signs & Symptoms

- SNS leads to tachycardia, palpitations, tachypnea, diaphoresis, pallor, cool skin, tremors, anxiety, weakness, & nausea
- Other s/s: hunger, paresthesia, blurred vision, HA, irritability, fatigue, confusion
- Serum glucose < 50
  - 20 - 40 -> seizures
  - < 20 -> coma
Hypoglycemia Interventions

- D 50% if patient unable to swallow
- Glucagon 0.5 to 2 mg IV, IM or SQ
- PO 10 grams carbohydrates (4 oz OJ) followed by protein
- Re-measure serum glucose level 20-30 minutes after treatment
- Treat cause
- Patient education!!!
Hypoglycemia

Potential complications:

- Seizures - treat hypoglycemia
- Arrhythmias and extension of MI - treat according to ACLS protocol

Management

- Maintain serum glucose 80-110 mg/dl
- Evaluate patient for signs/symptoms of hypoglycemia
- Patient education and identification (alert bracelet)
Glucose Control in ICU

- Intensive glucose control has been recommended by American Diabetes Association, American Society of Clinical Endocrinologists and IHI; it is widely practiced in ICUs.

- NICE-SUGAR study (2009) demonstrated lowering glucose to 81-108 increased mortality in critically ill patients.
Glucose Control in ICU

- NICE study-standard control group glucose level of <180
- Over 6000 subjects from 42 hospitals
- 90 day mortality was 2.6% higher in the tight glucose control group
- Severe hypoglycemia (<40) occurred in 6.8% of tight glucose control group vs. 0.5% in the standard control group
Glucose Control in ICU

- Based on these studies, clinical practice guidelines developed by American Assoc of Clinical Endocrinologists and American Diabetes Assoc recommend insulin infusions to maintain glucose in critically ill patients between 140-180 to minimize risk of hypoglycemia.
- Society of Critical Care Medicine Surviving Sepsis guidelines recommends glucose <150.
- AHA recommends target glucose 90-140.
- Current trend is more liberal glucose control in critically ill patients.
DKA Pathophysiology

- Severe metabolic complication of Insulin dependent Diabetes Mellitus
- Insulin deficiency and increase in insulin antagonistic hormones (glucagons, cortisol and catecholamines)
- Altered metabolism of carbohydrate, fat, and protein and hyperglycemia
**DKA Pathophysiology**

- Decreased insulin level and gluconeogenesis and increased insulin resistance result in exaggerated hepatic glucose production

- Lipolysis leads to increased fatty acids and ketonemia and ketonuria -> metabolic acidosis
DKA Pathophysiology

- Hyperglycemia leads to increased osmolarity of ECF and cellular dehydration which leads to reduced tissue perfusion -> lactic acidosis -> metabolic acidosis
- Glycosuria and osmotic diuresis leads to F&E imbalance. Lactic acidosis and ketosis leads to metabolic acidosis and altered mental status
- Dehydration leads to hypotension, hypovolemic shock, coma and cellular death
- Hyperkalemia leads to arrhythmias
DKA

- **Etiology**
  - Insufficient insulin dose or missed doses
  - Infection or trauma
  - Poor compliance with disease management
    - Educational needs
    - Psychosocial stressors
    - Alcohol or drug use
    - Adolescence
  - Medication side effects
DKA Signs and Symptoms

- Neuro: change in LOC, lethargy, visual changes
- Cardiac: hypotension, tachycardia, tall, peaked T waves, widening QRS
- Pulm: Kussmaul respirations, acetone breath
- GI: n/v, diarrhea, abdominal pain
- Renal: polyuria
- Skin: warm, flushed, dry and dry mucous membranes
DKA Lab Values

- Elevated serum glucose
- Elevated ketones, metabolic acidosis
- Presence of glucose and ketones in urine
- Hypokalemia or serum potassium >5.5mEq/l
- Metabolic acidosis pH < 7.3, HCO3 < 18
- Elevated Hct
- Anion gap: Na - (Cl + HCO3) > 10
Goals of Therapy:
- Correct dehydration and electrolyte balance
- Correct hyperglycemia
- Restore tissue perfusion
- Restore acid base balance
- Resolve infection if present
DKA Interventions

- 0.9% NS if serum sodium is low or 0.45% NS if sodium normal
- D5W with 0.45% NS when glucose < 250 mg/dl
- IV Insulin infusion until blood glucose <250 mg/dl
- Hourly serum glucose monitoring and urine ketone monitoring
- Strict I & O
- Monitor ABGs
- Administer bicarb if pH < 7.0 for cerebral and myocardial protection
DKA Interventions

- Monitor for hypoglycemia
- Administer antibiotics if infection is present
- Patient teaching with Diabetes educator
- Address psychosocial issues - new diagnosis, lifestyle changes, poor compliance, identify precipitating factors
Hyperosmolar Hyperglycemic Non-Ketosis (HHNK)

- Life threatening hyperglycemic episode with hyperosmolality, severe dehydration, and altered mental status without ketosis.
- Relative insulin deficiency impairs glucose transport, but sufficient insulin to prevent ketosis.
- Hyperglycemia leads to osmotic diuresis, dehydration, severe electrolyte losses and compromised glomerular filtration reducing urinary release of glucose.
- Cellular dehydration may lead to coma.
HHNK Etiology

- Newly diagnosed non-Insulin dependent diabetes
- Advanced age and severe dehydration
- CVA, MI, pancreatitis, sepsis, GI bleed in patient with preexisting non-Insulin dependent DM
- Drug interactions: dilantin, steroids, thiazide diuretics
- TPN or tube feedings without adequate hydration
HHNK Signs and Symptoms

- Change in mental status, lethargy or coma
- Cardiac: hypotension, tachycardia, decreased cardiac output, CVP and PCWP
- Pulm: deep, rapid respirations without acetone breath
- GI: n/v, polydipsia
- Renal: polyuria
- Skin: warm, flushed, dry and dry mucous membranes
HHNK Lab Values

- Severely elevated serum glucose > 1000 mg/dl
- No ketosis present
- Elevated serum osmolality
- Sodium and potassium levels depend on hydration but are often depleted and may require potassium replacement
- Tissue hypoperfusion may lead to lactic acid production and acidosis
Goals of Therapy:
- Correct dehydration and electrolyte balance
- Correct hyperglycemia
- Restore tissue perfusion
- Resolve infection if present
HHNK Interventions

- 0.9% IV fluids to correct hydration
- IV Insulin infusion
- Hourly serum glucose monitoring
- Strict I & O
- Monitor for heart failure with fluid replacement
- Monitor for hypoglycemia
- Monitor for thromboembolic event from dehydration and decreased tissue perfusion
Disseminated Intravascular Coagulation

- Hypercoagulable state
- Occurs when normal coagulation cascade is overstimulated
- Thrombosis and hemorrhage
- Coagulation occurs as many sites and normal inhibitory mechanisms are overwhelmed
- Clotting factors and platelets are depleted
DIC

- Thrombi continue to form on damaged epithelial walls occluding vessels leading to tissue ischemia and end organ dysfunction
- Clot formation activates fibrinolytic system which breaks down fibrin clots releasing fibrin split products which act as anticoagulants -> excessive bleeding!
- End result is shock and end organ dysfunction and failure if cycle is not interrupted!
DIC Etiology

- Shock
- Sepsis
- Extensive surgery
- Trauma, burns
- Malignancy
- Obstetric complications
- Snake bite
- Heat stroke
DIC Signs and Symptoms

- Sudden onset of bleeding for no apparent reason
- Petechiae, ecchymoses, hematomas, epistaxis, conjunctival bleeding, uncontrollable bleeding from venipuncture sites, drains, lines and incisions
- Labs: elevated PT, PTT, INR: reduced platelets, reduced fibrinogen level
DIC Patient Management

- PREVENTION!!!
- Optimize oxygen delivery
- Reverse clotting mechanism
- Replace coagulation factors
- Prevent complications—acute respiratory failure, end organ dysfunction, acute kidney injury
Patient Management

- Oxygen and mechanical ventilation
- Blood and blood product administration
- Avoid venipuncture or other invasive procedures
- Assess for multiple organ dysfunction and limb ischemia
Good Luck!!!