ELECTROLYTE IMBALANCE

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(2013/01/24)
Pathophysiology, Clinical Features, Aetiology and Management of.....

A. Hyponatraemia

B. Hyperkalaemia
HYPONATRAEMIA

- The most common electrolyte disorder (occur in up to 6% of hospitalized patients)
- Serum sodium concentration of <135 mEq/L
- Due to an excess of body water relative to body sodium content
- Changes in total body water is regulated by thirst, arginine vasopressin (AVP) and the kidney
HYPONATRAEMIA

- Three types:
  1. Hypovolaemic Hyponatraemia
  2. Hypervolaemic Hyponatraemia
  3. Euvolaemic Hyponatraemia
HYPONATRAEMIA

**Hypovolemic**
- Gastrointestinal solute loss (diarrhea, emesis)
- Third-spacing (ileus, pancreatitis)
- Diuretic use
- Addison disease
- Salt-wasting nephritis
HYponatraemia

**Euvolemic**
- Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- Diuretic use
- Glucocorticoid deficiency
- Hypothyroidism
- Psychogenic polydipsia
HYPONATRAEMIA

Hypervolemic with decreased effective circulating blood volume
- Decompensated heart failure
- Advanced liver cirrhosis
- Renal failure
CARDIAC FAILURE

- Non-osmotic vasopressin stimulation
- Activation of ventricular and arterial receptors
- Stimulation of sympathetic nervous system
- Activation of the Renin-angiotensin-aldosterone system

- RENAL WATER RETENTION
- ↑ SYSTEMIC AND RENAL ARTERIAL VASCULAR RESISTANCE
- RENAL SODIUM RETENTION
- MAINTENANCE OF ARTERIAL CIRCULATORY INTEGRITY
CIRRHOSIS

- Systemic arterial vasodilation
  - Activation of arterial baroreceptors
    - Non-osmotic AVP stimulation
    - SNS stimulation
    - Activation of RAAS

- Cardiac output
- Water retention
- Systemic arterial vascular and renal resistance
- Sodium retention
- Maintenance of arterial circulatory integrity
CLINICAL FEATURES

- CNS symptoms – Sodium <125 mEq/L
  - Disorientation
  - Restlessness and agitation
  - Apathy
  - Psychosis
  - Seizures
- Others – nausea, vomiting, headache, muscle cramps
COMPLICATIONS OF SEVERE HYponatraemia

Due to hyponatraemia induced cerebral oedema,

I. Respiratory arrest
II. Coma
III. Brainstem herniation
IV. Death
Rapid correction of hyponatraemia can cause OSMOTIC DEMYELINATION

Goal - to raise the serum sodium level by 1.5 to 2 mEq/L/hour (<= 12 mEq/L for 24 hours)
OSMOTIC DEMYELINATION

- Characterized by confusion, quadriplegia, pseudobulbar palsy, and ‘locked-in syndrome’
TREATMENT OPTIONS

- Hypertonic saline (3%)
- Loop diuretics e.g. Frusemide
- For hypervolemic and euvolemic hyponatremia:
  - Fluid restriction
  - Demeclocycline
  - AVP antagonists e.g. Conivaptan
TREATMENT OPTIONS

- **Demeclocycline:**
  - Inhibits AVP action at the distal renal tubules
  - Render AVP ineffective even in the presence of increased AVP levels
  - Loss of free water in urine
TREATMENT OPTIONS

- Arginine vasopressin receptor antagonists:
  Act as an antagonist of vasopressin receptors
  Block the effect of vasopressin
  Excretion of free water in urine
POTASSIUM BALANCE

- The ratio of ICF:ECF $K^+$ concentration $\sim$38:1
- Maintained by basolateral $Na^+$, $K^+$-ATPase pump
- To maintain the steady state, $K^+$ ingestion should be matched with excretion
- $K^+$ secretion at distal convoluted tubule and cortical ducts – main contributor to $K^+$ excretion
HYPERKALAEMIA

- Plasma K\(^+\) concentration >5.0 mmol/L
- Chronic hyperkalemia - always due to decreased renal K\(^+\) excretion
- Hyperkalemia partially depolarizes the cell membrane and prolonged depolarization impairs membrane excitability
Impaired cell membrane excitability causes, 

- Nervous system:
  Weakness and flaccid paralysis

- Heart:
  ECG changes $\rightarrow$ ventricular fibrillation/asystole
HYPERKALAEMIA - CAUSES

- Renal failure
- Decreased $K^+$ secretion
  - Adrenal insufficiency, drugs (ACE inhibitors, NSAIDs, heparin)
- Resistance to aldosterone:
  - Tubulointerstitial disease, drugs ($K^+$-sparing diuretics,)
TREATMENT

1. Discontinuing exogenous K\(^+\) intake and drugs reducing K\(^+\) excretion
   e.g. Angiotensin Converting Enzyme Inhibitors

2. Minimizing membrane depolarization and excitability
   - Calcium gluconate
TREATMENT

3. Shift $K^+$ into cells
   - Insulin (with glucose to prevent hypoglycemia)
   - $\text{NaHCO}_3$
   - $\beta_2$-adrenergic agonists e.g. Salbutamol

4. Promoting $K^+$ loss
   - Diuretics
   - Cation-exchange resin e.g. Sodium polystyrene sulfonate
   - Dialysis
SUMMARY

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