Diabetes Insipidus

Absence of vasopressin due to impaired production of ADH (neurogenic) or failure of renal tubules to respond to ADH (nephrogenic); classically manifesting as polydipsia and high volume of poorly concentrated urine despite increased serum osmolarity

ANESTHETIC CONSIDERATIONS:

- Etiology of DI / Co-morbid Disease
  - Neurogenic
    - Posterior pituitary surgery, tumor
    - Head injury / trauma
  - Nephrogenic
    - Renal disease / obstruction
    - Drug induced
    - Fluoride toxicity (volatiles…methoxyflurane)
- Volume depletion / hemodynamic instability
- Hypernatremia
  - Altered neurological status
  - Risk of intracranial hemorrhage with acute, severe hypernatremia
- Complications of Management of DI:
  - iatrogenic SIADH, volume overload

ANESTHETIC GOALS:

- Correct hypernatremia (no more than 0.5 mEq/h)
- Maintain hemodynamic stability and electrolyte balance
  - May require ADH / DDAVP treatment +/- volume, chlorpropamide, clofibrate, HCTZ

HISTORY

- Urine output > 100 mL/hr with hypernatremia requires investigation for DI
- Serum Na, acuity of change
- Presence of symptoms of hypernatremia:
  - Thirst
  - Lethargy, altered mental status
  - Convulsions, coma
  - Hypovolemia / shock
- Localizing signs associated with acute and severe hypernatremia
  - Brain shrinkage and tearing of meningeal vessels → intracranial hemorrhage
- Management strategies employed (e.g. fluid administration, diuretic therapy, DDAVP etc.)
- Etiology of DI: Neurogenic (central) vs. Nephrogenic (lack of response to ADH at kidney)
  - Neurogenic
    - Posterior Pituitary injury
      - Head injury, basal skull fracture
      - Postoperative / intraoperative esp. transphenoidal pituitary surgery (often transient)
    - Infiltration with Sarcoidosis
  - Nephrogenic
    - Renal disease / obstruction
    - Drugs: Lithium, Glyburide, Amphotericin B, Methoxyflurane
    - Systemic disease – hypokalemia, hypercalcemia, sickle cell anemia

PHYSICAL

- **GENERAL** – vitals
- **CVS** - orthostatic BP, HR (hypotension, tachycardia, ischemia)
- **ENDO** – signs of multisystem effects secondary to other hormone deficiencies (pituitary)
- **RENAL** – urine volume (d/t polyuria)
- **CNS** – neurologic assessment (visual disturbance, altered sensorium)

INVESTIGATIONS

- **Labs**
  - Urine and serum electrolytes and osmolalities
    - Urine osmolality < 300 mOsm/L and serum Na > 150 mEq/L
    - Hypo-osmolar urine in the face of rising serum Na and osmolality
    - Serum calcium (hypercalcemia)
- **Imaging**
  - ECG
  - CT / MRI head as required
- **Special**
  - Endocrinology consult

OPTIMIZATION

- Optimize volume status → NS fluid boluses to euvolemia
- Rule out additional hormone deficiencies
- Management of serum sodium
  - Diuretics to improve urinary Na excretion, hypotonic crystalloid
  - DDAVP (IV or intranasal)
- Management of inciting event → d/c drugs causing DI (lithium, mannitol etc.), treat reversible renal causes

- With known history of DI give usual dose of DDAVP prior to surgery
- In patients with some ADH activity, DDAVP only necessary if serum osmolality > 290 mOsm/L
    - Chlorpropamide (200–500 mg/d) stimulates ADH release & sensitizes renal tubules to the hormone (may cause hypoglycemia!)
    - Clofibrate
    - Hydrochlorothiazide (antidiuretic effect!)

ANESTHETIC OPTIONS

- Local, regional, neuraxial, general once stable
- Be aware of increased MAC requirements for IH agents in setting of hypernatremia

ANESTHETIC SETUP

- **Drugs**
    - Hypotonic crystalloids available
    - Diuretics
    - DDAVP (100 mU IV + infusion 100-200 mU/h)
    - Usual emergency drugs
- **Equipment**
    - Usual CAS monitors + Foley to urometer
    - Consider arterial line for blood sampling (serum Na, osmolality)
    - CVP, PAC, TEE as needed to monitor volume status

MANAGEMENT OF ANESTHESIA

- **Induction**
    - Patient may be hypovolemic with BP and HR fluctuation, electrolyte abnormalities and arrhythmias
- **Maintenance**
    - Hemodynamic instability
    - Fluoride toxicity from prolonged Enflurane or sevoflurane extremely rare as cause of renal tubular dysfunction (but bear in mind for maintenance IH)
    - Monitor urine output, serum Na
    - Fluid management (as per Miller):
        - Hourly maintenance fluid + 2/3 of the previous hour's urine output, usually D5-½NS
        - If hourly fluids > 350 – 400 mL, add DDAVP
    - DDAVP therapy titrated to allow breakthrough polyuria to avoid iatrogenic SIADH
- **Emergence**
    - Altered sensorium; unable to protect airway

DISPOSITION & MONITORING

- May require HDU / critical care

COMPLICATIONS

- Vasopressin therapy causes vasoconstriction, and acute treatment could precipitate myocardial ischemia in unstable patient with CAD
- Plasma osmolality affected by increases in BUN or glucose; urine osmolality below that of serum in severe cases of DI; urine osmolality will not increase following ADH therapy of nephrogenic origin

PATHOPHYSIOLOGY

- Secretion of vasopressin (or ADH) from the posterior pituitary is in response to increased serum osmolality or presence of hypotension (via stimulation of vagal receptors of the left atrium and carotid sinus)
- Lack of ADH results in DI → presents in extreme thirst, polyuria and polydipsia
- Etiology
    - Central / neurogenic
        - Abnormality of hypothalamus or pituitary
            - Head trauma / neurosurgery
            - Brain tumor
    - Nephrogenic
        - Effects of ADH on distal tubules of kidney impaired
            - Renal disease, lithium toxicity, hypercalcemia, hypokalemia
            - Ureteral obstruction, medullary cystic disease
        - Neurogenic vs. nephrogenic differentiated on basis of response to DDAVP → which concentrates urine in the presence of neurogenic DI, but not nephrogenic
    - Other problems to be considered:
        - Psychogenic polydipsia (increased water intake)
        - Osmotic diuresis
        - DM type 1
- 50% of patients with central DI have associated idiopathic disease
- ? autoimmune process → impairs the response of the hypothalamus to hypertonicity
- DI post-pituitary surgery usually occurs in 4-12 hours, rarely arises intraoperatively
- Hypernatremia increases the MAC for inhalational agents
    - ? enhanced Na conductance during depolarization of excitatory membranes

REFERENCES

- Miller 6th Ed. pp 1767-1768, p. 1053, p. 2159
- Stoelting Co-Existing Disease pp 437 - 438
- Roizen & Fleisher p. 114