Disorders of Sodium in the Neurocritical Care Patient: Case-Based Learning

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Objectives

- Discuss why sodium is so important in the neurocritical care patient.

- Discuss common disorders that affect sodium balance:
  - Syndrome of inappropriate diuretic hormone (SIADH)
  - Cerebral Salt Wasting (CSW)
  - Diabetes Insipidus (DI)
  - Iatrogenic

- Conduct review of relevant case study

- No disclosures or conflicts of interest
Why is sodium so important in the neurocritical care patient?

- Patients with neurological injury commonly acquire changes in their sodium levels.
  - 17% hyponatremia, 5-51% hypernatremia

- Effect on Cerebral Volume

- Quick changes in sodium level can lead to serious complications:
  - Neuronal damage, cerebral edema, hemorrhage, increased intracranial pressure, herniation, and even death.

(Mahanna et al., 2015)
Hyponatremia

- Syndrome of inappropriate antidiuretic hormone (SIADH)
- Cerebral Salt Wasting (CSW)
Syndrome of Inappropriate Antidiuretic Hormone

(SIADH)
What is SIADH?

- SIADH is a disorder of impaired water excretion caused by the inability to suppress the secretion of antidiuretic hormone (ADH).
- The most common cause of euvolemic hyponatremia.
What is ADH?

- ADH (antidiuretic hormone) also known as AVP (arginine vasopressin) is a neurohypophysial hormone found in most mammals.
- ADH is made in the hypothalamus and released by the posterior pituitary.
- Primary ADH functions:
  - Retain water in the body
  - Constrict blood vessels.
As your body level of water reduces, you become thirsty and your pituitary gland makes more ADH which works on the kidneys to preserve water.
SIADH Pathophysiology

- ADH regulation is impaired in SIADH
- Nonphysiological secretion of ADH results in enhanced water reabsorption, leading to dilutional hyponatremia.
SIADH Causes and Risk Factors

- CNS disorders
- Chronic lung diseases
- Malignancies
- Pharmacological agents
- Surgical procedures
Clinical Features & Presentation of SIADH

- Laboratory findings
- Symptoms vary based on rate of development & severity of hyponatremia.
- Most patients with SIADH and chronic moderate hyponatremia (serum Na+ 120-129meq/L) appears asymptomatic.

- **Mild to moderate symptoms:**
  - Chronic hyponatremia (serum Na+ <120meq/L that developed over >48hrs).
  - Dizziness, gait disturbances, forgetfulness, confusion, lethargy.

- **Severe symptoms:**
  - Acute hyponatremia (serum Na+ <120meq/L that developed in <48hrs)
  - Seizures, coma, permanent brain damage, respiratory arrest, brain stem herniation, death.
Diagnostic Criteria for SIADH

- Exclusion of renal, adrenal, or other endocrine disease.
- Hypo-osmolar hyponatremia
- Concentrated urine
- High urine sodium
- High ratio of urine to serum osmolality
- Decreased urinary output (400 to 500mL/24hrs)
- Possible generalized weight gain (>5% of body weight)

(Hickey, 2009, p. 201)
Laboratory Criteria for the Diagnosis of SIADH

- **Hyponatremia:**
  - Serum Na+ level <135 mEq/L

- **Hypoosmotic plasma:**
  - Serum osmolality <280 mOsm/kg

- **Hyperosmotic urine:**
  - Urine osmolality >100 mOsm/kg

- **Hypernatremic urine:**
  - Urinary sodium level >20 MEq/L
Figure 2. Decision tree for the evaluation and management of hyponatremia in critically ill neurologic patients. CSW, cerebral salt wasting; SIADH, syndrome of inappropriate secretion of antidiuretic hormone.

(Rabinstein & Wijdicks, 2003)
Use of Hypertonic saline for hyponatremia

**Indications:** Severe hyponatremia causing life-threatening symptoms e.g. seizures

**Rate of correction:** Not to exceed > 2.5 meq/l/hr or 15 meq/l/day

**Calculation:** meq NaCl needed = 0.6 X Wt in Kg X (Desired Na - Actual Na)
1 Lit 3% NaCl = 513 meq NaCl
3% NaCl in ml needed = (meq NaCl needed X 1000) / 513

**Rate of infusion:** Adjust to increase Na at 1-2.5 meq/L/hr

**Check:** Na Q 4 hrs

http://www.clevelandclinicmeded.com/medicalpubs/micu/hyponatremia.htm
How can you tell CSW from SIADH?

<table>
<thead>
<tr>
<th>Features</th>
<th>CSW</th>
<th>SIADH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume Status</td>
<td>Decreased</td>
<td>Normal/Increased</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Albumin</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Bun/Cr</td>
<td>Increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Potassium</td>
<td>Normal/Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Uric Acid</td>
<td>Normal/Decreased</td>
<td>Decreased</td>
</tr>
</tbody>
</table>
SIADH Management

- Clearly identify the problem SIADH vs. CSW
- Treat underlying disease if possible
- Fluid restriction
- Intravenous saline
- High solute intake
- Salt plus a loop diuretic
- Vasopressin receptor antagonists
  - conivaptan (Vaprisol)
  - tolvaptan (Samsca)
- Demeclocycline or lithium
Rate of Hyponatremia Correction

- The maximum rate of correction of chronic hyponatremia (>48hrs) should be less than 9 meq/L in any 24-hour period.

- An initial rate of correction of 4 to 6 meq/L in the first two to four hours may be beneficial in patients with severe symptoms (eg, seizures). Monitor serum Na+ every 2-3hrs until patient stabilizes.

(Sterns, 2013)
Key Points

- The hyponatremia in SIADH is a result of an excess of water and not a deficiency of Na+.
- Typically, half of all diagnosed cases of hyponatremia are due to SIADH.
- Mild hyponatremia has been associated with increased mortality.
- Mild hyponatremia may rapidly progress to severe hyponatremia.
- Acute severe hyponatremia is associated with morbidity and mortality.
Cerebral Salt Wasting
(CSW)
Cerebral Salt Wasting

- What is it?
- How do we distinguish it from similar disorders?
- How do we safely and effectively manage it?
CSW: A History lesson

- First described in the 1950s, followed by the discovery of SIADH
- For many years, SIADH was thought to be the culprit of hyponatremia in patients with neurological diseases
- Research in the 1980-early 90s found a greater understanding of hyponatremia post neurological injury, leading to acceptance of CSW as an important factor and changing the way patients were managed.

(Yee et al, 2010)
CSW: What is it?

- Defined by the development of extracellular volume depletion due to a renal sodium transport abnormality in patients with intracranial disease and normal adrenal and thyroid function.

- Occurs due to cerebral disease in the setting of normal kidney function.

  - Reports vary
    - 7-75% after aSAH
    - 5-10% of TBI patients

- Characterized by hyponatremia in the setting of hypovolemic shock.

(Yee, Burns & Wijdicks, 2010)
Two Major Theories

- The Sympathetic Nervous System Hypothesis
- Natriuretic Peptide Hypothesis
  - Both involve changes in the Renin Angiotensin-Aldosterone system
Loss/decrease of adrenergic tone to the nephron after neurologic injury leads to two major consequences:

- Decrease renin secretion causing lower levels of aldosterone and decreased sodium reabsorption at proximal convoluted tubule.

- Dilatation of afferent arteriole; leading to increased filtration of plasma and sodium.

Flaw in theory: in general CNS injury leads to a surge of sympathetic tone in the acute phase of the injury.
Natriuretic Peptide Theory

- **Natriuretic peptides**
  - 4 peptides assoc. w/CSW: ANP, BNP, CNP, DNP
    - Atrial natriuretic peptide (ANP)
    - Brain natriuretic peptide (BNP)
    - C-type natriuretic peptide (CNP)
    - Dendroaspis natriuretic peptide (DNP)

- **Functions**
  - normally antagonize the RAAS system
  - promotes vascular relaxation
  - inhibit generation of vasoconstrictor peptides
NPT, cont

- Theory suggests an increase in peptides after CNS injury.
  - peptides cause their effects on the nephron
  - causes increased filtration of water and sodium at glomerulus
  - natriuretic and diuretic effects on renal tubules
  - possible paracrine inhibitory effects on mineralocorticoid production.

(Ruiz-Juretschke et al, 2011)
How to distinguish CSW in the Clinical setting?

- Basic w/u includes: CBC, electrolytes, renal function, albumin, bicarbonate, serum and urine osmo and Na levels

<table>
<thead>
<tr>
<th>Table 1 – Differential diagnosis of cerebral salt wasting (CSW) and syndrome of inappropriate antidiuretic hormone secretion (SIADH)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Extracellular fluid</strong></td>
</tr>
<tr>
<td>Salt balance</td>
</tr>
<tr>
<td>Urine output</td>
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<tr>
<td>Central venous pressure</td>
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<tr>
<td>Serum</td>
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<tr>
<td>Sodium (mmol/L)</td>
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<tr>
<td>Osmolarity (mOsm/L)</td>
</tr>
<tr>
<td>Creatinine</td>
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<tr>
<td>Uric acid</td>
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<tr>
<td>Urine</td>
</tr>
<tr>
<td>Sodium</td>
</tr>
<tr>
<td>Osmolarity</td>
</tr>
</tbody>
</table>

(Ruiz-Juretschke et al, 2012)
Key Component?

- VOLUME STATUS
Volume status

- difficult to accurately determine
- requires vigilance
- requires a “whole picture” approach
  - lab values (i.e. elevated BUN/Creatinine, H/H, serum Bicarb)
  - Emesis, diaphoretic, polyuria? JVD, peripheral edema, pulm. Edema?
  - accurate I/O measurements, body weight
  - BP, CVP, Wedge pressures
CSW: Treatment

- **Goals:**
  - restore intravascular volume
  - correction of hyponatremia
  - avoid side effects of hypervolemia, hyponatremia and the potential of osmotic myelinolysis
CSW: Treatment options

- Re-establish euvolemia and correct hyponatremia with 0.9%NS bolus and maintenance fluid.
  - aim for even to slightly positive net fluid balance.

- NaCl tabs
  - 1-3grams TID w/meals
  - side effect of diarrhea, leading to net neg fluid balance

- Addition of mineralocorticoids
  - fludrocortisone 0.1 TID and titrate up to 0.3 TID.
  - temporary, requires close monitoring for side effects: heart failure, pulmonary edema, HTN, hypokalemia and potentially hyperglycemia
  - administer 5-7 days or until volume and Na levels normalized
CSW: Advanced Treatment Options

- necessary if requiring large volumes or Na levels running dangerously low (<125mEq/L)

- Hypertonic saline solutions
  - 2% or 3% sodium chloride
  - frequent checks: rec Q 6 hrs for 2% and Q 4 hours for 3%
  - rates between 50-150cc/hr
  - may require additional volume to maintain fluid balance
Hypernatremia

- Iatrogenic
- Diabetes Insipidus
Causes of Hypernatremia

- **Iatrogenic**
  - Hyperosmolar therapy: mannitol, hypertonic saline

- **Surgical**
  - Central DI, traumatic brain injury, pituitary surgery, intracranial aneurysm, brain tumor

- **Medical**
  - Central DI, nephrogenic DI, drug induced, acute kidney injury

*HYPERNATREMIA*

"THE MODEL"
(Causes of ↑ serum sodium)

- **M**: Medications, meals (too much sodium intake)
- **O**: Osmotic diuretics
- **D**: Diabetes insipidus
- **E**: Excessive H₂O loss
- **L**: Low H₂O intake
Iatrogenic Hypernatremia

Why? To reduce cerebral edema resulting from many pathophysiologic mechanisms such as:

- Ischemic stroke
- Traumatic brain injury
- Intracerebral hemorrhage

Normalization of sodium:

- no definitive guidelines exist. Providers must exercise their best judgement and decide when it is appropriate to begin treating hypernatremia and normalizing sodium.
Diabetes Insipidus (DI)
What is DI?

- Diabetes insipidus (DI) is a condition characterized by excessive thirst and excretion of large amounts of severely diluted urine. There are two major forms of DI:
  - Central (pituitary) DI
  - Nephrogenic (renal) DI
Nephrogenic DI

- Normal secretion of ADH but varying degrees of renal resistance to its water retaining effects.
- Can be acquired due to pyelonephritis, potassium depletion, sickle cell anemia, chronic hypercalcemia, medications (lithium, methicillin, etc.)
- Familial X-linked trait
Central DI

- Also called neurohypoposphenal or neurogenic DI
- Deficient secretion of ADH
- Can be induced by trauma, pituitary surgery, or hypoxic ischemic encephalopathy
- Most often idiopathic
DI Pathophysiology

- Central DI is caused by a decreased amount of ADH.
- Kidneys are unable to conserve water as they perform their function of filtering blood.
Central DI

Causes and Risk Factors

- Idopathic causes (autoimmune injury to the ADH-producing cells)
- Trauma
- Pituitary surgery
- Hypoxic or ischemic encephalopathy
Clinical Features & Presentation of DI

- Polydipsia (fluid intake 5-20 L/day)
- Polyuria (urine output 2-20 L/day) and nocturia
- Weight loss, Fatigue
- Dizziness, Changes in level of consciousness
- Elevated temperature
- Tachycardia, hypotension
- Poor skin turgor and dry mucous membranes
Diagnostic Criteria for DI

- Polyuria
- Markedly dilute urine
- Low urine specific gravity
- Hypernatremia
- Normal to high serum osmolality
- Hypovolemia
- Polydipsia

(Hickey, 2009, p. 202)
Laboratory Criteria for the Diagnosis of DI

- **Hypernatremia:**
  - Serum Na$^+$ >142 meq/L

- **Hyperosmotic plasma:**
  - Serum osmolality >290 mOsm/kg

- **Hypoosmotic urine:**
  - Urine osmolality <100 mOsm/kg

- **Urine specific gravity low <1.005**
Diagnostic Approach

Urine output
- Low
  - Urine Osmolality
    - High
      - Hypotonic fluid loss:
        - Insensible loss
        - GI loss
        - Prior renal loss e.g. diuretics
  - Low
    - Diabetes Insipidus
  - High
    - Osmotic Diuresis

Response to DDAVP?
- Yes
  - Central Diabetes Insipidus
- No
  - Nephrogenic Diabetes Insipidus

Correction of hypernatremia

Calculate TBW
- Calculate DBW
- BWD = DBW - TBW
- Replace BWD in addition to maintenance fluid

Choice of fluid:
- For oliguric / hypotensive patient: Normal saline
- For stable patients: D5W

Rate of Correction:
- Replace 50% of BWD over 24 hours
- Rest over next 24-48 hours

Follow-up:
1. Follow serum Na Q 6 hours
2. Rate of decrease of plasma osmolality not to exceed >1 mosm/liters

Common Causes of Hypernatremia in ICU:
- Diarrhea
- Lactulose therapy
- Diuretics
- Insensible loss
- Osmotic diuresis

Current Body Water (CBW) = 0.6 X Current body weight (in Kg)
[Use 0.4-0.5 for females and cachectic patients]
Desirable Body Water (DBW) = [Current Na+ / 140] X CBW
Body Water Deficit (BWD) = DBW - CBW

http://www.clevelandclinicmeded.com/medicalpubs/micu/hypernatremia.htm
DI Management

- Fluid replacement
  - PO:
    - Estimate free water deficit: FW
    - Deficit = 0.6 x weight (kg) x [(Current Na ÷ 140) -1]
  - IV Therapy (crystalloid fluids):
    - Correct hypovolemia with isotonic saline. Administer free water or hypotonic fluid to correct Na+.
    - If serum Na+ >150, give D5W IV to replace ½ volume deficit in 12-24hrs.
    - When Na+ <150, substitute 0.45% NaCl or 0.9% NS.

- Rapid lowering of serum Na+ indicated for acute symptomatic hypernatremia; chronic hypernatremia must be treated more slowly.

- DDAVP

(Barkley & Myers, 2008)
DDAVP

- **Dosing Forms & Strengths**
  - **Injectable solution:**
    - 4mcg/mL
  - **Tablet:**
    - 0.1mg
    - 0.2mg
  - **Nasal spray:**
    - 0.1mg/mL (5mL): Delivers 10mcg/spray
    - 1.5mg/mL (2.5mL): Delivers 150mcg/spray
DDAVP Dosing for Central DI

- **Intranasal:** 5-40mcg/day qDay or divided q8-12hr.
  - Adjust morning and evening doses separately for appropriate diurnal rhythm of water turnover.

- **PO:** Initial 0.05mg q12hr; effective range 0.1-1.2mg divided q8hr-q12hr; observe fluid restriction.
  - If switching to PO from intranasal, start PO at least 12hrs after last intranasal dose.

- **IV/SC:** 2-4mcg/day divided q12hr or one-tenth the maintenance of intranasal dose.
DIABETES INSIPIDUS

History of:
- Head Injury
- Pituitary Tumor
- Craniotomy

Rx:
Vasopressin
DDAVP

S&S:
- Up to 20L Urine/Day
- ↓ Specific Gravity
- ↓ Osmolarity
- Hypovolemia
- ↑ Thirst
- Tachycardia
- ↓ BP

Nursing Care:
- Monitor Fluids
- Replace Fluids
- Neuro Status
- Vital Signs
- Mucous Membranes
Key Points

- DI is primarily a defect in the secretion of ADH.
- Accurate measurement of I/O is essential.
- After assessing fluid status and serum Na+ level, treat both dehydration and hypernatremia.
- DDAVP will only be effective for patients with central DI.
Case Study

- 51 yo F with PMH of hyperlipidemia admitted with sellar/suprasellar mass
  - 8/20 resected by Neurosurgery
    - Complicated by small intracerebral hemorrhage in the surgical bed.
  - Admitted to PACU/SDU post operatively
  - Pathology confirms non-secratary pituitary adenoma
Case Study

- 8/21: Foley catheter inserted
  - UO 11L, Na 158
- Dx’d with frank post surgical DI
  - Tx’d w/ 2 doses vasopressin, each lasting 36-40hrs
  - Stable for next 48 hrs
- 8/23: Na trends 142, 147, 142
  - UO avg 6L per day
Case Study

8/25-26
- Na levels 133>129>125
- UO from 6L>2L per day
- What is happening?

SIADH (2\textsuperscript{nd} phase DI) Why?
- Classic pattern (gen. lasts 5-15 days)
- Uninhibited release of ADH from necrosing nerve endings on pituitary stalk
- Tx’d w/ HTS d/t concern of CNS sequelae from hyponatremia & fluid restriction of 1 Liter.
Case Study

8/27

- Na level 140s on HTS
  - Stopped and transferred to NICU d/t change in level of consciousness
  - Na dropped to 129, restarted on HTS
  - No major improvement in MS

- Was HTS treatment necessary?
  - Typically the hyponatremia is self limiting and rarely becomes severe
  - Fluid restriction still maintain of tx for SIADH
Case Study

- **8/27 (night shift)**
  - Na 125>145 in 6hrs
  - UO 100>700>1L x 2 hrs

- **What is happening now?**
  - DI, again!

- **Treatment: DDAVP x 2 and started on vasopressin infusion**
  - Improvement in UO to 300-400/hr range
  - Na 140-142

- **Stabilized DI with intermittent DDAVP dosing w/endocrinology closely following.**
  - Treatment pearl of wisdom: Treat not on increases in UO, instead monitor for associated shifts in Na level. Otherwise may cause iatrogenic SIADH
Case Study

**8/30-9/10**

- developed cerebral vasospasm
  - Treated with IA verapamil
- Developed acute onset of hypovolemic, hyponatremia.
  - Did not respond to DDVAP
  - What Next????
Case Study

- Cerebral Salt Wasting complicating DI
  - Why??
- Two thoughts
  - ICH lead to increased BNP > CSW
  - Possible low renin and aldosterone levels
- Treated w/fludrocortisone and usual DI management w/ DDAVP based on Na and urine output
Case Study

- In summary; one patient, three physiologically distinct endocrinopathies, with different treatment regimens within a 3 week time-span all stemming from the same initial illness.
- Highlights the complication of neuro-endocrine connection.
- Critical thinking, accurate recording and collaboration of treatment teams.
Hypoosmolality

Disease states:
- SIAD
- CSWS

Cerebral edema

iatrogenic:
- Hypotonic fluids

Non-osmotic stimuli that trigger AVP release:
- Decreased arterial blood pressure
- Decreased arterial blood volume
- Nausea, emesis
- Pain, stress, anxiety, fear
- Postoperative state

Autoregulatory mechanisms

Sodium, potassium, chloride, water egress
- Rapid phase

Organic osmolyte loss, water egress
- Later phase

Hypotonicity detected by the hypothalamus suppresses AVP release, urine is more dilute

Normal Osmolality

Normal brain

iatrogenic/therapeutic:
- Hypotonic saline
- Mannitol
- Salt tablets
- Diuretics
- Fluid restriction

Hypothalamus

Supraoptic nucleus

Paraventricular nucleus

Posterior Anterior

Pituitary

AVP secreted

AVP stored

Hypertonicity induces thirst and AVP release from the posterior pituitary, causing water reabsorption via aquaporin channels in the distal convoluted tubule and the collecting duct, concentrating urine

Autoregulatory mechanisms

Rapid phase

Na+, K+, Cl- transport into cells, water follows

Regulatory volume increase

Later phase

Organic osmolytes return to cells, water follows

Hyperosmolality

Cellular dehydration

Treatment:
- Hypotonic saline
- Free water
- Vasopressin
- Desmopressin acetate

Renal collecting duct

Aquaporin channels

AVP binds

Urine

Rapid overcorrection

Cell volume normalized, but hypertonic state persists

Treatment:
- Fluid restriction
- Salt tablets
- Hypertonic saline
- AVP receptor antagonists

Kidney

Rapid overcorrection

Cell volume normalized, but hypotonic state persists

Osmotic demyelination syndrome

(Wright, 2012)
<table>
<thead>
<tr>
<th>Feature</th>
<th>Central neurogenic diabetes insipidus</th>
<th>Syndrome of inappropriate secretion of antidiuretic hormone (ADH)</th>
<th>Cerebral salt-wasting syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definition</td>
<td>Fluid imbalance due to decreased secretion of ADH in the posterior lobe of the pituitary gland or to renal unresponsiveness to the release of ADH</td>
<td>Persistent production or overproduction of ADH resulting in water intoxication and a volume-expanded state</td>
<td>Renal loss of sodium leading to true hyponatremia and a volume-contracted state in which the kidneys do not reabsorb sodium</td>
</tr>
<tr>
<td>Cause</td>
<td>Hypotension, stress, pain, anxiety, and an upright position Trauma, surgery, or damage of the hypothalamus</td>
<td>Head trauma, brain tumor, abscess, subarachnoid hemorrhage, hydrocephalus, meningitis, encephalitis, Guillain-Barré syndrome</td>
<td>Cause unclear but often occurs in patients with intracranial abnormalities (head trauma, stroke, subarachnoid hemorrhage, brain tumors) Loss of both intravascular fluid and sodium</td>
</tr>
<tr>
<td>Serum level of sodium, mEq/L</td>
<td>Hyponatremia (&gt;145 (high))</td>
<td>Hyponatremia (&lt;135 (low))</td>
<td>Hyponatremia (&lt;135 (low))</td>
</tr>
<tr>
<td>Serum osmolality, mOsm/kg</td>
<td>&gt;295 (high)</td>
<td>&lt;275 (low)</td>
<td>&lt;275 (low)</td>
</tr>
<tr>
<td>Urinary osmolality, mOsm/kg</td>
<td>Decreased (&lt;200)</td>
<td>Elevated (&gt;100)</td>
<td>Elevated (&gt;100)</td>
</tr>
<tr>
<td>Urinary level of sodium, mEq/L</td>
<td>Within normal reference range or decreased</td>
<td>Within normal reference range or elevated (&gt;25)</td>
<td>Elevated (&gt;25)</td>
</tr>
<tr>
<td>Urine output</td>
<td>Increased (&gt;250 mL/h)</td>
<td>Decreased (400-500 mL/24 h)</td>
<td>Decreased</td>
</tr>
<tr>
<td>Urinary specific gravity</td>
<td>&lt;1.005 (very dilute)</td>
<td>&gt;1.010 (concentrated, dark)</td>
<td>&gt;1.010 (concentrated, dark)</td>
</tr>
<tr>
<td>Extracellular fluid volume</td>
<td>Decreased</td>
<td>Increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Serum urea nitrogen</td>
<td>Elevated</td>
<td>Normal or low (dilutional)</td>
<td>Elevated</td>
</tr>
<tr>
<td>Mental status</td>
<td>Normal to impaired</td>
<td>Confusion Lethargy</td>
<td>Decreased level of consciousness, agitation, coma</td>
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<tr>
<td>Body weight</td>
<td>Decreased</td>
<td>Normal or increased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Tachycardia</td>
<td>Slow or normal</td>
<td>Resting or postural tachycardia</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Normal to mildly hypertensive progressing to hypotension</td>
<td>Hypertensive</td>
<td>Postural hypotension</td>
</tr>
<tr>
<td>Treatment</td>
<td>Fluid replacement (0.45% saline intravenously replaced milliliter for milliliter, or greater) ADH replacement with desmopressin acetate intranasally or orally, lyspressin intranasally, or aqueous vasopressin intravenously</td>
<td>Fluid restriction (800-1000 mL/24 h) Slow sodium replacement with normal saline or hypertonic (3%-5%) saline intravenously</td>
<td>Replacement of fluid volume and sodium No restriction of fluids Slow sodium replacement with hypertonic (3%) saline intravenously</td>
</tr>
</tbody>
</table>

Table: Comparison of central neurogenic diabetes insipidus, syndrome of inappropriate secretion of antidiuretic hormone, and cerebral salt-wasting syndrome.

(John & Day, 2012, pg. e4)
References


