Central Diabetes Insipidus (Arginine Vasopressin Deficiency) Pathway

Pathway Purpose: Provide high quality, safe and transparent care to hospitalized patients with central diabetes insipidus (arginine vasopressin deficiency).

Oral, Inhaled, or Subcutaneous DDAVP (Desmopressin) Therapy

Inclusion Criteria:
- *Central* Diabetes Insipidus on DDAVP (oral, inhaled or subcutaneous)
- Hospital admission (acute care or ICU)

Exclusion Criteria:
- Age < 12 months
- Hyperhydration for chemo
- IV Vasopressin (see pg 2)

→ Consult Endocrinology

Upon Admission:
- Consult endocrinology to discuss Diabetes Insipidus (DI) plan (DDAVP dose/freq, fluid goals, sodium goals)
- Consult hospitalist service if surgical team primary
- Place Diabetes Insipidus (DI) Orderset
- Primary MD/APP, bedside RN & charge RN to huddle on DI management plan

Monitoring:
- q2h **strict** intake & output **** If patient voids, urine output (or diaper) must be checked again in one hour***
- Daily weights

LOW Risk Diabetes Insipidus:
Patient with appropriate sense of thirst, medically cleared and able to drink to thirst, not on any IV fluids or medications which affect sodium or fluid balance, no significant comorbidities

Fluid & Sodium Management:
- Allow patient to drink ad lib (will regulate fluid intake to thirst)
- Fluids to drink easily accessible at bedside
- q12h serum Na until stable, then q24h
- **Call endocrinology if making patient NPO**

HIGH Risk Diabetes Insipidus:
NPO status, abnormal sense of thirst, enteral tube feeds, comorbidities (cerebral edema, altered mental status, renal dysfunction, heart disease, sepsis, etc.)

Fluid & Sodium Management:
- Discuss fluid I/O goals DAILY with Endocrinology
- Replace significant free water deficits per pg 2
- q4h serum Na until stable, then q8-12h
- **Call endocrinology if making patient NPO**

Desmopressin (DDAVP) Therapy – *RN to give a scheduled OR PRN dose of DDAVP ONLY when:*
(1) Patient has **met criteria** for high dilute urine output
Patients less than 75 kg: Urine output over 4mL/kg/h between two consecutive voids
Patients greater than 75 kg: Urine output over 300mL/h between two consecutive voids
(2) MD/APP notified and in agreement with giving scheduled or PRN DDAVP dose.

RN to Notify Primary Team MD/APP:
- IF serum sodium < 135 or > 145, **OR** shift > 5mEq/L since prior check
- IF new evidence of fluid overload (edema, weight gain) or dehydration (tachycardia, weight loss)
- IF UOP does not decrease significantly within 3 hours of patient receiving a DDAVP dose
- IF patient meets criteria for DDAVP therapy (as above), but no PRN dose is available
- IF any scheduled DDAVP doses are delayed or held (for low UOP or any other reason)

If worsening fluid/sodium status, consider transfer to PICU for vasopressin IV & closer monitoring (see pg 2)

Owner: Sejal Shah
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Associated Order Set: Diabetes Insipidus
Associated Policies: none

Feedback?

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Vasopressin (IV) Therapy - (ICU only)

Inclusion Criteria:
- Diagnosis of *Central* Diabetes Insipidus
- ICU admission

Exclusion Criteria:
- None

Upon Admission:
- Consult endocrinology
- Place Diabetes Insipidus Orderset
- Primary MD/APP, bedside RN, charge RN to huddle on fluid & Na goals, I &O monitoring and DDAVP/vasopressin plan.

Monitoring:
- q1h strict Intake & output (place Foley catheter only if needed)
- q2h serum Na until stable, then q4h thereafter
- daily weights

Free Water Deficits (Hypernatremia) Management:
- Calculate **free water deficit (mL)** = 4 mL × weight (kg) × [current serum Na - desired serum Na (mEq/L)][5]
  Note: recommend choosing a desired serum Na between 145 and 150 to prevent too rapid correction of hypernatremia.
- Replace significant/chronic free water deficits slowly to avoid cerebral edema. Correction should not exceed a fall of serum sodium greater than 0.5 mEq/L per hour (ie, 10 to 12 mEq/L per day), initial strategy could be to replace first 50% of calculated deficit over 8 hours, last 50% over subsequent 16 hours, but may need to individualize based on patients rate of serum sodium correction.[5]

Fluid Management:
- IV + PO at 2/3 maintenance for ongoing losses/maintenance (in addition to free water deficit correction), using isotonic fluids (normal saline or lactated ringers)
- ***NEVER*** order fluids for ongoing urine output replacement after urine output controlled on vasopressin drip

Vasopressin Therapy:
Start vasopressin at 0.5 milliunits/kg/h and titrate rate frequently (q5-10 minutes) by 0.5 milliunits/kg/h, up to 10 milliunits/kg/h, aiming to achieve a urine output of less than 4mL/kg/h (goal 1-2mL/kg/h, or the urine output goal specified for the patient)

RN to Notify PICU MD/APP:
- Serum Na < 140, > 150 **OR** shift > 5mEq/L
- Excessive urine output (UOP)
  - *Patients <75 kg: UOP > 4mL/kg/h*
  - *Patients >75 kg: UOP > 300mL/h*
- Clinical evidence of fluid overload (edema, weight gain) or dehydration (tachycardia, weight loss)
- Urine output outside goal range 1-2mL/kg/h, or the urine output goal specified for the patient

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Definitions:

Anti-diuretic hormone (ADH), also called **vasopressin,** is a hormone produced by the brain which acts on the kidney to retain water and increase blood pressure.

Desmopressin (DDAVP) is a synthetic form of vasopressin which comes in oral, inhaled and subcutaneous injectable forms, and is used to treat patients with central diabetes insipidus. The IV infusion formulation is called **vasopressin.**

Central diabetes insipidus (Arginine Vasopressin Deficiency) is the inability to concentrate urine secondary to the lack of ADH production by the brain – typically due to a tumor or congenital malformation in the region of the pituitary gland or a brain injury (meningitis, trauma, hypoxia, hemorrhage), but may also be hereditary[1,3]. In children, central DI is much more common than *nephrogenic* diabetes insipidus, which is the inability to concentrate urine secondary to an inability of the kidneys to respond to ADH.

Diagnostic Criteria:

- Polyuria

*<75 kg: UOP > **4mL/kg/h***
*>75 kg: UOP > **300mL/h***

- Serum Na high normal
- Serum osmolality high normal
- Urine osmolality under 300 mOsm/kg
- Other etiologies ruled out (see below)

Diagnosis confirmed by endocrinology with DDAVP trial; water restriction testing rarely used[2].

Pathophysiology:

In healthy people, ADH is released into the blood by the pituitary in response to high blood osmolarity or low blood volume / blood pressure. ADH then acts on the kidney to retain water. The amount of ADH released is perfectly regulated to plasma sodium and total body water, second to second, to maintain homeostasis[1].

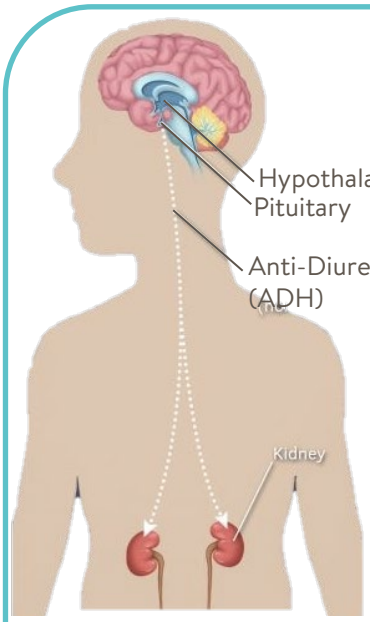
In central DI patients, a dose of DDAVP nearly "turns off" urine production for several hours - the bigger the dose, the longer the effect. If drinking or receiving IV/enteral tube fluids over the several hours following a dose, serum sodium drifts downward and total body water drifts upward. When the DDAVP wears off, the kidneys begin producing urine again (typically a large, rapid diuresis) raising serum sodium and decreasing total body water, (can happen quickly)[1-3].

Giving DDAVP too early: It is important that the patient void at least 4mL/kg/per hour between 2 consecutive voids (if < 75kg) or 300mL/hr between 2 consecutive voids (if > 75kg). This occurs in a short period of time (under 2 hours) before taking their next DDAVP dose – otherwise the patient can become **hypo**natremic and fluid overloaded (stacked DDAVP doses).

Giving DDAVP too late: If the patient has a prolonged period of diuresis, they may become dehydrated and **hyper**natremic – particularly if NPO, or if patient with abnormal sense of thirst.

Polyuria DDX:

- Central diabetes insipidus
- Diabetes Mellitus
- Hypercalcemia
- Nephrogenic diabetes insipidus
- Osmotic diuresis (urea, mannitol, contrast, NaCl glucose, etc.)
- Post obstructive diuresis
- Primary polydipsia



Central Diabetes Insipidus affects the production of ADH[4]

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