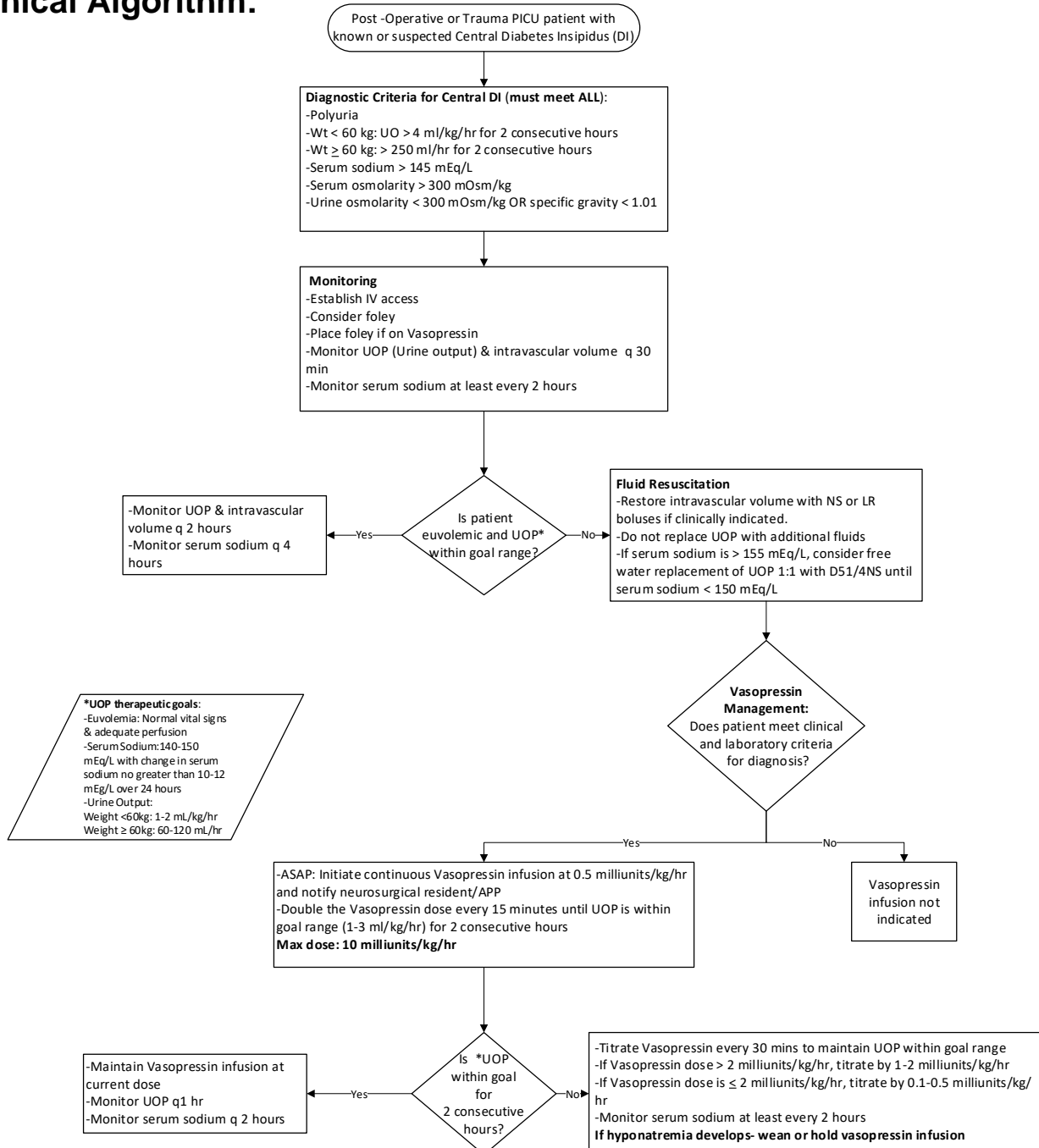


Diabetes Insipidus Management in the Post-Operative or Trauma Patient in the Pediatric Intensive Care Unit, Inpatient, Pathway

Updated: April 28, 2023

Clinical Algorithm:



Clinical Pathway Summary

CLINICAL PATHWAY NAME: Diabetes Insipidus Management in the Post-Operative or Trauma Patient in the Pediatric Intensive Care Unit

PATIENT POPULATION AND DIAGNOSIS: Pediatric post-operative or trauma patients in the Pediatric Intensive Care Unit (PICU) with confirmed or suspected Diabetes Insipidus

APPLICABLE TO: HDVCH

BRIEF DESCRIPTION: This guideline is intended for use in pediatric post-operative or trauma patients in the Pediatric Intensive Care Unit (PICU) with confirmed or suspected Diabetes Insipidus. Use in other patients such as those with brain injury, meningitis, etc., should only be used with PICU attending approval.

OPTIMIZED EPIC ELEMENTS (if applicable): N/A

IMPLEMENTATION DATE: March 2023

LAST REVISED: April 28, 2023

Clinical Pathways Clinical Approach

TREATMENT AND MANAGEMENT:

A. Background

- I. Diabetes insipidus (DI) results from deficiency of arginine vasopressin
- II. Patients at risk for DI include those following sellar or suprasellar tumor resection (75% of patients with transcranial resection of pituitary tumor; 10 to 44% after transsphenoidal pituitary surgery), or pre-existing diagnosis of central DI
- III. Symptoms can develop intraoperatively or immediately post-operatively (first 12 hours is typical)
- IV. Management of DI in these children can be complicated by over or under hydration and electrolyte imbalances, which can cause or worsen cerebral edema, and/or result in seizures

B. Purpose

- I. To facilitate safe and effective diagnosis and management of children with perioperative DI with the goal to minimize fluctuations in serum sodium concentrations, fluid balance, and subsequent complications.

C. Diagnostic Criteria for Central DI

- I. Patients must meet ALL criteria
 - a. Polyuria
 - i. Weight < 60 kg: urine output > 4 ml/kg/hour for 2 consecutive hours
 - ii. Weight ≥ 60 kg: > 250 ml/hour for 2 consecutive hours
 - b. Serum sodium > 145 mEq/L
 - c. Serum osmolality > 300 mOsm/kg

d. Urine osmolality < 300 mOsm/kg OR specific gravity < 1.01

- II. Exclude other causes of polyuria, such as hyperglycemia, diuretic administration (e.g. intra-operative Mannitol), acute or chronic kidney injury

D. Therapeutic Goals

- I. Euvolemia: Normal vital signs and adequate perfusion
- II. Serum sodium: Between 140-150 mEq/L, with change in serum sodium no greater than 10-12 mEq/L over 24 hours
- III. Urine output
 - a. Weight < 60 kg: 1-3 mL/kg/hr
 - b. Weight ≥ 60 kg: 60-120 ml/hr

E. Monitoring

- I. Establish IV access
- II. Consider foley catheter placement
- III. Place foley catheter for patients on vasopressin
- IV. Monitor UOP and intravascular volume every 30 minutes, serum sodium at least every 2 hours until patient is euvolemic and UOP is within goal range. Thereafter, monitor UOP, intravascular volume status every hour, with sodium monitoring every 4 hours

F. Fluid Resuscitation

- I. Restore intravascular volume only for patients with clear intravascular depletion with normal saline or Lactated Ringer's boluses
- II. If patient is NPO or has impaired thirst mechanism, place on IVFs with D5NS or D5LR at maintenance rate; consider D5 ½ NS in patients whose free water deficit or hyponatremia remains persistent despite treatment
 - a. An established DI patient who is NPO, discuss with Endocrinology for IVF recommendation
- III. If patient can take PO with intact thirst, consider allowing patient to drink to thirst while on vasopressin infusion and discontinue IV fluids
- IV. Do not replace UOP with additional fluids unless serum sodium is > 155 mEq/L, then consider free water replacement of UOP 1:1 with D5 1/4NS until serum sodium < 150 mEq/L

G. Vasopressin Management

- I. Continuous infusion of vasopressin should be initiated as soon as possible once clinical and laboratory criteria for diagnosis are met
 - a. Initiate vasopressin infusion at 0.5 milliunits/kg/hr
 - b. Double the dose every 15 minutes until UOP is within goal range (1-3 ml/kg/hr) for 2 consecutive hours
 - c. Max dose: 10 milliunits/kg/hr
- II. Once urine output is within goal range, titrate vasopressin every 30 minutes to maintain UOP within that goal
 - a. If vasopressin dose is > 2 milliunits/kg/hr, titrate by 1-2 milliunits/kg/hr
 - b. If vasopressin dose is ≤ 2 milliunits/kg/hr, titrate by 0.1-0.5 milliunits/kg/hr
 - c. Monitor serum sodium at least every 2 hrs
- III. If hyponatremia develops:
 - a. Wean or hold vasopressin infusion
 - b. Be aware of potential "triple phase" response following pituitary surgery, where SIADH may develop after polyuric phase

- c. Consider other causes of hyponatremia (cerebral salt wasting, CSF losses, etc)
- IV. Neurosurgical resident or APP should be notified at the time the vasopressin drip has been ordered and notify neurosurgical attending if indicated
- V. If the patient is out of the PICU for a prolonged period of time (i.e post-operative MRI), hourly UOP needs to be called to the PICU fellow caring for the patient. Labs need to be sent on schedule and results reported to the PICU fellow
- VI. On day 2 post-op, consult endocrinology service regarding initiation of home DI regimen
- VII. Ensure DDAVP present at bedside at least 1 hour prior to anticipated administration and BEFORE stopping vasopressin infusion

Pathway Information

OWNER(S): Elora Hussain, MD, Pediatric Neurocritical Care

CONTRIBUTOR(S): Christel Keefe, MD, Pediatric Endocrinology; Michael Bercu, MD, Pediatric Neurosurgery; Julie Miller, CPNP-AC, Pediatric Neurosurgery; James DeCou, MD, Pediatric Trauma and Surgery, Lindsey Jelsma DNP

EXPERT IMPROVEMENT TEAM (EIT): Pediatric Trauma Performance Committee 3/16/2023

CLINICAL PRACTICE COUNCIL (CPC): Children's Health

CPC APPROVAL DATE: March 28, 2023

OTHER TEAM(S) IMPACTED : Neurosurgery, Trauma, PICU, Endocrinology

References

Alharfi, Ibrahim M. MD^{1,2}; Stewart, Tanya Charyk MSc^{3,4}; Foster, Jennifer MD^{1,6}; Morrison, Gavin C. MRCP, DCH¹; Fraser, Douglas D. MD, PhD^{1,5-9}. Central Diabetes Insipidus in Pediatric Severe Traumatic Brain Injury. *Pediatric Critical Care Medicine* 14(2):p 203-209, February 2013. | DOI: 10.1097/PCC.0b013e31827127b5

Pratheesh, R., Swallow, D. M., Rajaratnam, S., Jacob, K. S., Chacko, G., Joseph, M., & Chacko, A. G. (2013). Incidence, predictors and early post-operative course of diabetes insipidus in paediatric craniopharyngioma: a comparison with adults. *Child's nervous system : ChNS : official journal of the International Society for Pediatric Neurosurgery*, 29(6), 941–949. <https://doi.org/10.1007/s00381-013-2041-8>

Seckl, J. R., Dunger, D. B., & Lightman, S. L. (1987). Neurohypophyseal peptide function during early postoperative diabetes insipidus. *Brain : a journal of neurology*, 110 (Pt 3), 737–746. <https://doi.org/10.1093/brain/110.3.737>

Wise-Faberowski, L., Soriano, S. G., Ferrari, L., McManus, M. L., Wolfsdorf, J. I., Majzoub, J., Scott, R. M., Truog, R., & Rockoff, M. A. (2004). Perioperative management of diabetes insipidus in children. *Journal of neurosurgical anesthesiology*, 16(3), 220–225. <https://doi.org/10.1097/00008506-200407000-00006>