Insidious Insipidus: An Atypical Postoperative Presentation of Severe Hypernatremia Secondary to Idiopathic Central Diabetes Insipidus

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Learning Objectives

1. Describe the signs and symptoms of central diabetes insipidus.
2. Differentiate central diabetes insipidus from nephrogenic diabetes insipidus.
3. Justify the role of pharmacological therapy in treating central diabetes insipidus.

Introduction

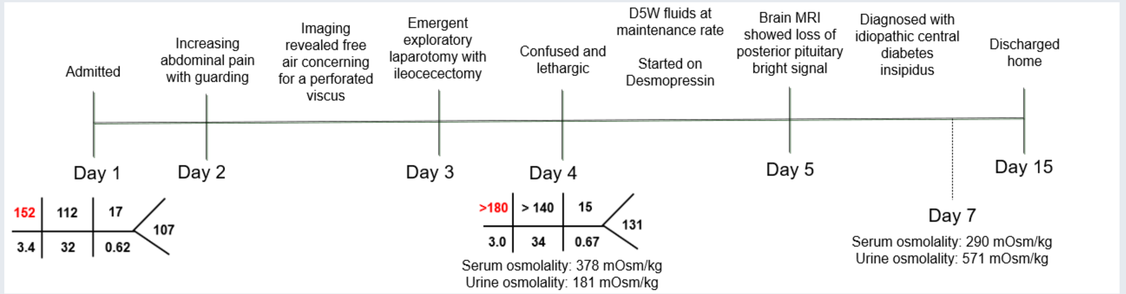
Central diabetes insipidus (CDI) is caused by a decreased arginine vasopressin level (ADH), and is characterized by hypotonic polyuria and polydipsia. CDI may result from any pathology that impairs synthesis, transport, or release of ADH, which acts on V2 receptors in the kidney to promote reabsorption of free water. If central DI is suspected, testing of pituitary hormones and MRI imaging of the pituitary gland will help to elucidate potential etiologies, including idiopathic DI, intracranial tumors, histiocytosis, and autoimmune causes.

Case Presentation

37-year-old woman with a history of Crohn's disease and small bowel obstructions:

- Presented to the ED with lower abdominal pain, nausea, vomiting, and oral intake intolerance. Physical examination: soft and nondistended abdomen with right lower quadrant tenderness.
- Abdominal x-ray: dilated loops of small bowel consistent with a moderate grade partial obstruction.
- The following morning, she developed severe abdominal pain with diffuse exquisite tenderness and voluntary guarding. An abdominal x-ray demonstrated subdiaphragmatic free air concerning for a perforated viscus.
- Underwent emergent exploratory laparotomy with ileocecectomy and creation of an end ileostomy.
- Found to be confused and lethargic the following morning. A basic metabolic panel revealed severe hypernatremia, hyperchloremia, and hypokalemia. Serum osmolality was elevated, and urine osmolality was low suggesting diabetes insipidus.

Hospital Course



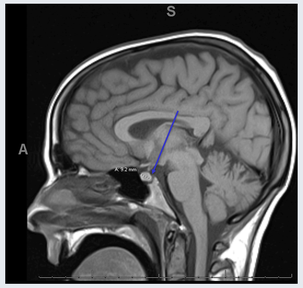


Figure 1

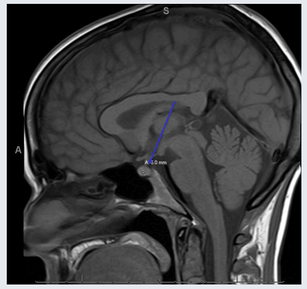
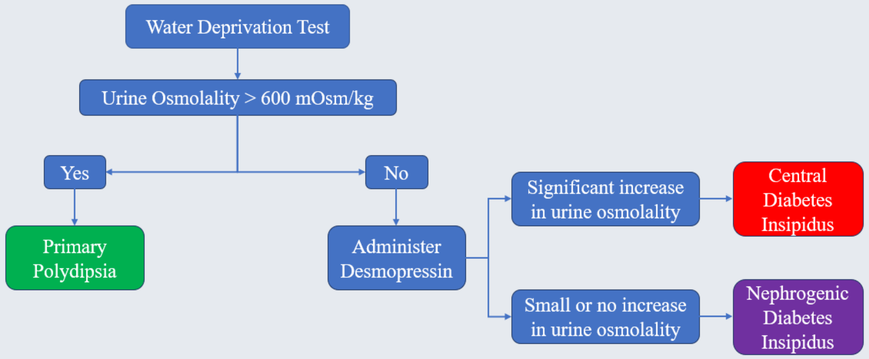


Figure 2

Figure 1 demonstrates a normal bright signal in the posterior pituitary.

Figure 2 demonstrates loss of the bright signal in the posterior pituitary, suggesting central diabetes insipidus.

Water Deprivation Test



Take Home Points

- Free water restriction will distinguish primary polydipsia from diabetes insipidus.
- In a healthy individual, fluid restriction will transiently increase serum osmolality, leading to ADH release and increased water resorption in the distal tubule, concentrating the urine.
- In both central and nephrogenic DI, the impaired effect of ADH leads to increased plasma osmolality and dilute urine.
- The desmopressin stimulation test can distinguish central DI, which involves an absolute ADH deficiency, from nephrogenic DI, in which the collecting tubules are non-responsive to ADH stimulation.
- Pharmacologic therapy with desmopressin, a vasopressin analogue, can be administered to patients with CDI to correct the deficit and resolve plasma and urine osmolality.

Approach to Polyuria

	Serum sodium	Urine osmolality following water deprivation	Urine osmolality following vasopressin administration
Normal	Normal	Increased	No additional increase
Central diabetes insipidus	High	No change or mildly increased	Significant increase
Nephrogenic diabetes insipidus	High	No change or mildly increased	Mild increase
Primary polydipsia	Low	Increased	No additional increase

References

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