



Case Report

Hypernatremia and central Diabetes Insipidus following Neurosurgical procedure of Trauma

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INTRODUCTION

The greater risk of hypernatremia in patients over 65 years old are associated with impaired mental status and physical disability which may result in impaired sensation to thirst, impaired ability to express thirst, and/or decrease access to water [1,2]. Normally, anti-diuretic hormone (ADH, also known as arginine-vasopressin, AVP) is secreted in response to 1-2% increase in osmolality which stimulate thirst, as do hypovolemia and hypotension. Broadly, speaking hypernatremia is due to hypotonic fluid loss or hypertonic sodium gain. Hypotonic fluid loss is mainly caused by diabetes insipidus. There are many causes of central diabetes insipidus (CDI). Pituitary and hypothalamus injuries caused by trauma, neurosurgical procedures, hemorrhage, ischemia, autoimmune diseases e.g. hypophysitis, IgG4 related disease or tumors involving hypothalamic-pituitary axis (HPA) are but few causes of CDI. Central diabetes insipidus is also reported with traumatic brain injuries causing ischemia to the HPA [3] and presented with exertion of large volumes of diluted urine (polyuria). Polyuria is defined by a urine volume in excess of 2 L/m²/24 h or approximately 40-50 ml/kg/24 h in older children or adult.

This is a case of permanent central diabetes insipidus following traumatic brain injury with decompression surgery presented with polyuria, high plasma osmolality, and hypernatremia which responded nicely to desmopressin (dDAVP) treatment. Attempts to decrease the dDAVP dose to assess whether the CDI is transient or permanent confirmed the permanent nature of central diabetes insipidus. In patients with CDI, a normal sense of thirst with free access to fluid intake will allow normal plasma electrolytes and osmolality. Increase plasma osmolality (>300 mosm/L) with decreased urine osmolality and a urine/plasma osmolality ratio <1 clenches the diagnosis of DI. The response to dDAVP will differentiate between CDI and nephrogenic diabetes insipidus (NDI).

CASE HISTORY

The case is of 68 years male patient with history of ventilation dependent respiratory failure, septic shock secondary to aspiration pneumonia, severe protein-calorie malnutrition, multiple pressure ulcers, toxic metabolic encephalopathy, and dysphagia on tube feeding. He sustained traumatic brain injury and underwent bilateral decompression craniotomy for subdural hematomas. At the time of patient's encounter, physical examination revealed that he had intellectual disability, his maximum temperature was 98F, heart rate was 96 beats per minute, blood pressure was 104/68 mmHg, with respiratory rate of 14 per minute. Examination of the heart and abdomen were normal. Respiratory examination revealed scattered rhonchi, with



poor respiratory effort. He was on T-Piece ventilation with FIO2 28% and oxygen saturation of 94%. Two days following the decompression craniotomies, his laboratory work showed that his plasma sodium was 155 mEq/L (Table 1).

DISCUSSION

The incidence of diabetes insipidus in the neurosurgical patients ranges from 6.7% in post-craniotomy, 4% after aneurysmal surgery, and 2% after traumatic brain injury. The diagnosis of diabetes insipidus can be clenched by urine osmolality <200 mosm/L, urine specific gravity (USG) <1.005, UOP>2.5 ml/kg/h, normal/high serum osmolality, and urine/plasma osmolality ratio <1. It is important to rule out polyuria due to fluid diuresis, or osmotic diuresis secondary to glycosuria or mannitol administration. Low urine specific gravity with low urine osmolality rule out osmotic diuresis.

Idiopathic diabetes insipidus accounts for the majority of cases of central DI [4-6]. However, primary and secondary tumors with involvement of the pituitary or hypothalamic area, infiltrative disease [7], (e.g. histiocytosis or sarcoidosis), neurosurgery, and trauma account for the rest causes of CDI.

In children the most common secondary causes of CDI were craniopharyngeom (25.2%) [8], infiltrative disease in 38% and idiopathic in 52% of cases [6]. Diabetes insipidus can become apparent for the first time in pregnancy as a results of catabolism of the ADH by the enzyme vasopressinases secreted by the placenta [9,10]. This type of DI is usually transient and resolved soon after delivery.

Neurosurgical procedures and trauma to the hypothalamus and posterior pituitary may result in CDI (10-20% to 60-80%) depending on the underlying type of the neurosurgical procedure [11-15]. Lower rate of CDI has been reported with minimally invasive endoscopic neurosurgical pituitary procedures (2.7-13.6%), [16]. High serum sodium (>145 mEq/l) in the first five days postoperatively had a high predictive value for permanent DI.

The course of postoperative CDI can be transient, permanent, or triphasic. The triphasic pattern occurs in 3.4% of patients who undergo trans-sphenoidal surgery, and only the first 2 phases occur in 1.1% of patients [17]. In the triphasic pattern, the patient goes into CDI, followed by a second oliguric phase of SIADH, and then the third phase of permanent CDI [18,19].

The case presented here developed permanent CDI. Attempts to decrease the dose of dDAVP resulted in polyuria with low USG and urine osmolality and high serum sodium. In a study of 1571 patients with pituitary adenomas who underwent trans-sphenoidal surgery only 0.9% were still receiving ADH or had polyuria [17]. Degeneration of hypothalamic ADH secreting neuronal cell bodies is the underlying cause of the third phase of permanent CDI which occurs within 2 weeks of the surgery [20].

Table 1: The rest of the work up in the following few days.

SNa	SK	Chloride	HCO3	BUN	Creatinine	I/O	USG	Uosm/Posm
155	3.8	117	26	35	0.8	5030/3775	1.005	175/330
145	3.3	112	28	36	0.8		1.007	259/310
157*	4.8	112	28	31	0.75		1.004	169/330
147	3.9	107	29	39	0.78			/313
146	3.4	106	28	40	0.77	3020/2050		/314
146	3.9	104	28	34	0.7			/310
142	4.2	102	30	31	0.63		1.011	385/295
141	4.0	101	27	28	0.63	2920/1950	1.012	480/295
140	4.2	102	26	26	0.56	2640/1850	1.014	490/294

SNa – serum sodium, SK – serum potassium, USG- urine specific gravity, Uosm- urine osmolality, Posm- plasma osmolality.* dDAVP intruded

Adipsic DI is another entity which impaired thirst mechanism and aggravate hypernatremia. It can result from clipping of the anterior communicating aneurysm with dramatic rise in plasma ADH levels occurring during non-osmotic stimuli like hypotension and apomorphine [21]. It indicates that the osmoreceptors in the anterior pituitary are affected. Disruption of blood supply provided by the anterior communicating artery result in destruction of these nuclei and therefore, ADH secretion in response to thirst or hyperosmolarity is impaired but the response to hypotension and other stimuli are intact [21].

It is interesting in this case that serum chloride was high following the serum sodium levels. Hyperchloremia is due to loss of electrolyte-free water or administration of sodium chloride-containing fluids. Loss of electrolyte-free fluid occurs due to increased insensible losses as a result of increased sweating (e.g. fever), and in hypermetabolic states as accompanied thyrotoxicosis as well as in inadequate water replacement secondary to loss of thirst perception as seen in elderly, infants or individuals with altered mental status. Loss of electrolyte-free water also occurs in central or nephrogenic diabetes insipidus as seen in the patient under discussion. High chloride with metabolic acidosis usually without increase in serum sodium occurs in proximal and distal renal tubular acidosis due to loss of bicarbonates. In these cases the serum chloride concentration is above 110 mEq/L but the serum sodium is normal.

Hypernatremia as defined by serum sodium >145 mEq/L is less common than hyponatremia. It indicates the severity of the underlying disease process. Mortality in head injuries with associated hypernatremia is higher as compared to those with normal serum sodium [22]. It occurs when access to water or thirst is impaired as seen in patients with altered mental status, decreased level of consciousness, and those on the ventilations, or frailty. The other contributing causes for dehydration are fever, dehydration and osmotic diuresis [23]. A urine specific gravity of <1.005 in the presence of high serum sodium is a useful sign which points to CDI [24]. Treatment of CDI is accomplished by replacement of fluid loss and dDAVP supplementation with careful monitoring of serum sodium especially if the thirst mechanism is impaired or interfered with due to any cause.

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