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Case Report

Symptomatic Hyponatremia after Continuous Infusion of Vasopressin: A Case Report

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Abstract

Arginine Vasopressin (AVP), also known as antidiuretic hormone, is a endogenously secreted peptide by the posterior pituitary in response to hyperosmolar plasma or systemic hypoperfusion states. Patients in refractory shock associated with severe sepsis, cardiogenic or vasodilatory shock, or cardiopulmonary bypass have inappropriately low plasma levels of AVP ('relative vasopressin deficiency') and supersensitivity to exogenously-administered vasopressin. Low doses of vasopressin can restore vasomotor tone in conditions that are resistant to catecholamines, with preservation of renal blood flow and urine output. This agent exerts its vasoconstriction effects through smooth muscle V1 receptors and also has antidiuretic activity via renal V2 receptors. This interaction with the renal V2 receptors results in the integration of aquaporin 2 channels in the apical membrane of the renal collecting duct leading to free water reabsorption. Thus, water intoxication with subsequent hyponatremia, although rare, is a potentially serious side effect of exogenous vasopressin administration. We present 1 patient who developed hyponatremia with initiation of vasopressin infusion. The patient required the use of hypertonic saline for more rapid normalization of serum sodium due to her seizure.

Keywords: Antidiuresis; Hyponatremia; Vasopressin; Vasodilatory shock; Vasopressors

Introduction

Arginine Vasopressin (AVP), also known as Antidiuretic Hormone (ADH), is produced in the hypothalamus and secreted into the circulation through the posterior pituitary gland. Although AVP is secreted in response to stress or shock states, its circulating levels are inappropriately low in patients with refractory hypotension associated with hypovolemia, severe sepsis, cardiogenic shock, or cardiac arrest ('relative vasopressin deficiency') [1,2], which is thought to contribute to the hypotension of shock. In refractory shock, endovascular AVP is depleted [1,3] and exogenous vasopressin exerts profound vasopressor effects even at doses that would not affect arterial blood pressure in normal subjects [4]. Fluid resuscitation and catecholamine support are the standard therapeutic strategy for hemorrhagic shock. However, when the shock is prolonged, the response to both fluid and catecholamine vasopressors can be poor because of acidosis, desensitized receptors, persistent vasodilation and/or NO release [3]. AVP enhanced hemodynamic performance and improved survival, neurologic outcome in animal models in which severe uncontrolled blood loss was induced [5,6]. As with any pharmaceutical agent, the use of vasopressin can result in both beneficial and detrimental effects. In vasodilatory shock, the desirable vasopressor action is counterbalanced by the potential side effects, including water intoxication with subsequent hyponatremia. Here we present 1 patient in which the use of vasopressin resulted in severe hyponatremia.

Case Report

Moriom, 7 years old girl, weighing 15 kg, was a diagnosed case of obscure GI bleeding, got admitted into BSMMU due to hematemesis and melena with hypovolemic shock. On admission, she was severely pale, anicteric, feeble pulse with tachycardia, CRT>3 seconds, BP was 50/30 mm of Hg. She was managed with bolus dose of Normal saline infusion 20 ml/kg followed by 10 ml/kg for 2 times, subsequently with whole blood transfusion. She was treated with exogenous Vasopressin infusion 0.3 unit/kg bolus followed by maintainence dose with 0.2 unit/1.73 m^2/min with 5% D/A saline. Her investigation revealed Hemoglobin 5.4 gm/dl, Total WBC count-7500/cmm, Platelet count- 2500000/cmm, S.ALT-28 U/L, PT- 13 seconds, INR-1.1, S. Albumin-35g/L, S.Creatinine-0.5, S. Sodium-135mmol/l, S. Potassium -3.2 mmmol/l, S.Chloride - 98 mmol/l.On 2nd day of treatment, patient had decreased urine output and became drowsy. Her GCS was 13/15. Subsequent electrolyte reports showed S.Sodium-122 mmol/L, S. Potassium-2.9 mmol/L, S.Osmolarity-280mosm/L, RBS-5.4mmol/L. Patient was managed with 5% Dextrose in Normal saline infusion. On 3rd day of admission, she developed generalized tonic clonic seizure whish was single episode, persisted for 3 minutes. Her electrolyte reports now showed severe hyponatremia (S. Sodium-110 mmol/l), S.Potassium- 3.3 mmol/l, S.osmolarity-265 mosm/l. Patient was managed with 3% Sodium Chloride infusion. But her subsequent electrolyte report after calculated correction showed still persisting hyponatremia (S.Sodium-115mmol/L). So, we thought that this persisting hyponatremia might be due to the exogenous vasopressin infusion and stopped vasopressin infusion. 8 hours after stoppage of vasopressin infusion her electrolyte reports showed dramatic improvement of S.Sodium which was 125 mmol/L and after 24 hours it was 135 mmol/l. Therefore, it is concluded that Vasopressin use in

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this patient lead to rapid changes in serum sodium levels concerning for cerebral edema development and it is imperative to monitor serum sodium when a patient is on Vasopressin. After stabilization of patient we did other relevant examination to find out the source of bleeding. Endoscopy of upper and lower GIT revealed normal anatomy, Meckel's scan was negative for ectopic gastric tissue, CT angiography reports were also normal.

Discussion

Release of endogenous vasopressin from the neurohypophysis can be triggered by several mechanisms including osmotic, nonosmotic (hypotension and decreased effective blood volume), and hormonal stimuli [7-9]. Vasopressin is a nonspecific peptide hormone which exerts its actions through interaction with at least 4 well-known receptors [10,11]. Through interaction with V1 receptors vasopressin exerts the vasoconstrictive effects in vascular smooth muscle. By V1-mediated vasoconstriction of efferent arterioles in the kidneys vasopressin can lead to increased urine output [12,13]. On the other hand, vasopressin can also exert antidiuretic effects through interaction with V2 receptors in the renal collecting duct cells. Vasopressin mediated activation of V2 receptors results in a cyclic Adenosine Monophosphate (cAMP) signaling cascade leading to phosphorylation, translocation, and incorporation of Aquaporin 2 Channels (AQP-2) into the apical plasma membrane of the renal collecting duct allowing passive free water reabsorption across an osmotic gradient [10-13]. Water intoxication with subsequent hyponatremia is the potential consequence of V2 receptor activation by exogenous vasopressin administration. However, in clinical trials of vasodilatory shock the incidence of vasopressin-induced hyponatremia has been rare [14-16]. For instance, in the randomized, double-blind, Vasopressin and Septic Shock (VASST) trial comparing vasopressin (0.01-0.03 units/min) versus norepinephrine infusion in 778 adult patients with septic shock (396 randomized to vasopressin), only 1 patient of the 102 developed hyponatremia [17]. Also, there was no observed hyponatremia in the treatment or control groups, in a randomized controlled trial, in pediatric patients with vasodilatory shock given low-dose vasopressin (0.0005-0.002 units/kg/ min) in combination with other vasopressors [16]. A search of the literature did not yield any peer-reviewed publications reporting significant rates of hyponatremia when vasopressin is used for vasodilatory shock. Although, several high rates of hyponatremia have been reported in studies where vasopressin is used for indications other than vasodilatory shock [18-20]. For example, in a pilot safety study, 24 critically ill but hemodynamically stable children were administered either low-dose vasopressin (0.005 units/kg/min) or normal saline placebo [18]. In all, 8 (66%) children in the vasopressin group developed hyponatremia versus 1 (8%) in the control group despite a trend toward higher sodium intake in the vasopressin group and no differences in fluid intake between the groups [18]. Increased diuresis and subsequent rise in serum sodium ensued within hours of discontinuing vasopressin. Significant hyponatremia was also observed following high dose (0.4 units/min) of vasopressin administration in a small study comparing somatostatin with vasopressin for thetreatment of acute variceal hemorrhage [19]. Similarly, a case report described the development of severe hyponatremia (serum sodium 116 mEq/ mL from baseline of 141 mEq/mL) in a 35-year-old woman after3 days of vasopressin infusion for variceal hemorrhage at 0.4 units/min [20]. Prompt diuresis and subsequent normalization of serum sodium occurred in this patient within 24 hours of discontinuing vasopressin. Our patient also improved clinically with diuresis and normalization of her seum sodium after discontinuation of exogenous vasopressin.

Conclusion

Vasopressin-induced hyponatremia, a rare event, can occur when vasopressin is used in patients with vasodilatory shock. Although rare, the case report presented demonstrate that hyponatremia can still manifest. However, more research is needed, if hyponatremia does ensue, additional factors must be assessed for their negative impacton plasma sodium concentrations such as vasopressin dose, patient body weight, adrenal insufficiency, and other medications.

Financial Support

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Conflict of Interest

None.

Contribution by Authors

All authors contributed in the management of case.

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