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Diabetes Insipidus after Vasopressin Withdrawal: A Case Report and Review of The Literature

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1. Abstract

Diabetes insipidus (DI) is a relatively rare disorder presenting with the excretion of highly hypotonic urine, resulting in polydispia and high plasma osmolarity. Vasopressin, which is also referred as antidiuretic hormone (ADH), is a peptidic hormone, produced by the hypothalamus and released from the posterior lobe of the pituitary gland; its primary role in the human body is to regulate the blood pressure and the water balance.

This article presents an interesting case of a transient episode of diabetes insipidus in a 48-year-old male patient with chronic psychiatric illness hospitalized intubated with Acute Respiratory Distress Syndrome, after discontinuation of vasopressin, which was administered for the treatment of septic shock. The condition was managed with the prescription of desmopressin (0.05mg), given once. As the use of vasopressin is routine practice for the treatment of septic shock, physicians should acknowledge this rare adverse effect of diabetes insipidus after the withdrawal of vasopressin and treat accordingly.

2. Case Report

A 48 year-old male patient, suffering from chronic psychiatric disease was admitted intubated in the Intensive Care Unit (ICU) from the Emergency Department due to decreased level of consciousness and respiratory failure. The relatives reported that he was afebrile the day before. (ή γράψε από πότε αναφέρουν οι συγγενείς ότι δεν είναι καλά). On arrival in the ICU, the patient presented with severe hemodynamic instability with acidosis (pH: 7.12, pCO2: 50.3 mmHg and Lactate: 5.3) and while fluids were administered for resuscitation, vasopressors were also initiated (noradrenaline 0.23 μg/kg/min, vasopressin 2.3 ml/h). Echocardiography ruled out cardiogenic shock (ejection fraction 40-45%, VTI 20 cm, absence of pericardial effusion and satisfactory right ventricular function), troponin was negative (0.012 ng/dl) and $ScvO_2$ was 81%. The electrocardiogram, revealed sinus tachycardia with 120 bpm. Regarding the respiratory system, he was set on volume control ventilation (tidal volume 500ml, PEEP 10 cmH_2O, Pplateau 12cmH_2O) the PaO_2/FiO_2 was 100 mmHg. Chest x-ray showed bilateral lung infiltrations and the CT revealed bilateral consolidation in the lower lobes and ground glass opacities in the right middle lobe. Purulent secretions were present, while Pneumococal and Legionella urine antigens and rapid PCR for respiratory viruses and bacteria (film array) were negative. Brain CT was negative, while chest. A lumbar puncture was performed to rule out Central Nervous System infection. On the fifth day his oxygenation significantly improved (PaO2/FiO2 436mmHg) and shock had improved, thus it was decided to wean the patient from sedation. After sedation withdrawal, the patient was normotensive and vasoactive drugs were discontinued. Two days after vasopressin discontinuation, the patient presented increased urine output (250 ml/h, specific gravity 1023, serumsodium 149mmol/l (το οποίο ήταν αυξανόμενο?)). No extra fluids had been administered during the preceding hours. Among the conditions which were included in the differential diagnosis, wa acute renal failure, Cushing's syndrome, hypercalcemia, diabetes insipidus and pharmaceutical polyuria. Thus, it was suspected that the patient might present an episode of diabetes insipidus on the same day, probably

due to discontinuation of vasopressin, and 0.05 mg of desmopressin was administered. The patient responded promptly; his urine output was reduced (80ml/h), and serum sodium was normalized (143mmol/l) during the next 3 hours without the administration of any fluid. The following day he was successfully extubated and was discharged from the ICU. No other episode of diabetes insipidus was recorded thereafter.

3. Discussion

Diabetes insipidus (DI) is a relatively rare disorder and the main characteristic feature is the excretion of highly hypotonic urine, resulting in high plasma osmolarity [1]. DI can be classified into three main types:

1) Neurogenic (or central), which is due to disordered metabolism (synthesis, storage and / or excretion) of vasopressin from hypothalamus and hypophysis,

2) Nefrogenic, in which the pathophysiological disorder is localized in the kidneys, which have poor response to vasopressin and finally,

3) Gestational, during pregnancy, due to the production of very large amounts of vasopressinase by the placenta [2].

Vasopressin, which is also referred as antidiuretic hormone (ADH), is a peptidic hormone, produced by the hypothalamus and released from the posterior lobe of the pituitary gland; its primary role in the human body is to regulate the blood pressure and the water balance [3]. Over the last years, vasopressin has gained considerable attention, due to its remarkable vasoconstrictive properties; recent research focusing on vasopressin as a first-line vasoconstrictor has gained particular interest [4-6].

We present an interesting case of a transient diabetes insipidus episode in a 48 year-old male patient with chronic psychiatric illness, who was hospitalised intubated due to ARDS, after withdrawal of vasopressin administered for the management of septic shock. The condition has been sporadically described in the literature; Ferenchick et al., (2019) after reviewing 1896 patients who received vasopressin for the treatment of septic shock, found out that the incidence of the occurrence of DI after the withdrawal of vasopressin was 1.53% (29 patients) [8]. Persico et al., (2022), in a scoping literature review, described the occurrence of acute DI after withdrawal of vasopressin, in 51, in total, patients some of which resulted in severe complications, such as hypernatremia and hypovolemia. The incidence of the complication was 1.53% [7]. The main reason ICU admission was septic shock (23.5%), followed by cardiovascular (20.4%), respiratory (20.4%), surgical (17.6%) and neurological (16.3%) complications.

The diagnosis is mainly based on clinical findings. Bohl et al. (2017) published a case series of six neurosurgical patients who developed severe transient DI after withdrawal of vasopressin, having as the main laboratory findings decreased urine specific gravity, increased urine output and elevated serum sodium. The mean time for DI occurrence after vasopressin's withdrawal, was 5 hours-while in one, DI occurred one hour after vasopressin's discontinuation [10]. The same clinical findings served diagnosis in the patients included in the review by Persico et al. [7]. In our patient although the urine sg was 1023, he presented signs of hypovolemia (serum sodium 149 mmol/l) and DI did not present again after its first presentation and successful treatment with desmopressin. One of the most useful biomarkers for the diagnosis of the disease is copeptin, a peptide derived from vasopressin, which is particularly stable and easy to identify and measure. Elevated baseline copeptin concentrations in the plasma accurately identify DI [11]. Unfortunately, copeptin levels are not measured in our institution.

The exact pathogenetic mechanism of the syndrome has not yet been fully elucidated. Ferenchick et al. [8]. Hypothesized, that it could be due to the transient down-regulation of V2 receptors, caused by the increased levels of vasopressin that had been administered to the patient for the treatment of septic shock [8]. As this complication is not common, we hypothesize that an idiosyncratic reaction may be also implicated in these patients.

The management of DI caused by the withdrawal of vasopressin involves three treatment modalities: re-administration of vasopressin, restoration of fluid status and electrolyte balance and thirdly the administration of desmopressin (D-arginine-vasopressin, DDAVP), which avoids rendering the patient hypertensive, circumventing potential complications resulting from increased blood pressure such as hemorrhagic complications. Moreover, desmopressin presents a better antidiuretic effect in comparison to vasopressin [12, 13]. In Persico's review, the treatment included use of desmopressin (D-arginine-vasopressin, DDAVP) (31.4% of the patients), fluid management (31.4%) and restoring the administration of vasopressin (16.3%). Two children were included among the cases, one of which was treated with fluids and electrolytes, whereas the second with re-administration of vasopressin [7]. The cases presented in Bohl's study were treated with resumption of vasopressin – nevertheless DI re-appeared during the attempt to taper the dose of vasopressin in all of six of them, the authors advice for the routine use administration of desmopressin in order to control the levels of sodium, until transient DI is resolved [10]. After administration of desmopressin, the patient's electrolyte status and fluid balance should be monitored every 3 hours, giving special attention to the possibility of developing the syndrome of inappropriate ADH secretion [9].

Reviewing the cases that have been published in the literature until now, administration of desmopressin was the modality used in the majority of them (31%), followed by re-administration of vasopressin (16%). Regarding the group of patients treated with desmopressin, 56% had complete response, while the remaining 44%

had partial resolution of their symptoms [7]. It seems, that in some cases, resuming the administration of vasopressin, in consecutive sessions, may be necessary, until the administration of intermittent doses of desmopressin alone can prevent the recurrence of DI [8]. Administration of desmopressin in our patient, not only remitted DI but may have also contributed in the complete resolution of this side effect, avoiding the vicious circle of potential re-manifestation of DI, if vasopressin was re-administered.

4. Conclusion

As the use of vasopressin and its analogues has become increasingly popular in the recent years in the treatment of severe septic shock, it is considered very important for the treating physicians to be able to promptly recognize potential diabetes insipidus occurrence after the withdrawal of vasopressin.

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