Differentiating Electrolyte and Fluid Disorders

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2020 ANNA Poster Presentation

Vasopressin or anti-diuretic hormone (ADH) is a nonapeptide that is synthesized in the hypothalamus and plays an important role in the control of the body's osmotic balance, blood pressure regulation and kidney function. ADH induces expression of water transport proteins in the late distal tubule and collecting duct to increase water re-absorption. ADH is produced by neurons within the supraoptic nuclei of the hypothalamus, stored in secretary vesicles and transported through the hypothalamic-hypophyseal tract and released in the posterior pituitary. The secreted hormones then enter nearby fenestrated capillaries where they enter the body's systemic circulation. These neurons express osmo receptors that are responsive to blood osmolarity and respond to changes as little as 2 mOsm/L.

Therefore slight elevations in osmolarity result in secretion of ADH. ADH acts in the kidneys to increase water re-absorption, thus returning the osmolarity to baseline. ADH is also secreted in times of hypovolaemia. Decreased arterial blood volume is sensed by bar receptors in the left atrium, carotid artery and aortic arch and the signal transported to the vagus nerve which directly stimulates the release of ADH. ADH then promotes water re-absorption in the kidneys to increase effective arterial blood volume and increase blood pressure to maintain tissue perfusion. Osmolarity and volume status are the two greatest factors that affect ADH secretion. There are three pathologic states related to ADH.

Hyponatraemia

Hyponatraemia has multiple etiologies with the treatment potentially different depending on the cause. Hyponatraemia is defined as a sodium concentration less than 135 mmol/L, it is a common water balance disorder that often poses a diagnostic or therapeutic challenge (Hoorn & Zietse, 2017). Hyponatramia causes neurologic symptoms by reducing the plasma osmolality to less than 285 mOsm/kg. Low osmolality produces a shift in the osmotic gradient between the extracellular and intracellular compartments, causing water to flow into the intracellular compartment and resulting in intracellular oedema and rupture of cell membranes. Hyponatraemia has a predilection for causing neurologic symptoms because the blood brain barrier is impermeable to sodium. In very acute hyponatraemia the cells are especially vulnerable to injury. It is important that sodium levels are not corrected too rapidly. Levels should not increase more than 8 mmol/l in 24 hours, as rapid correction of severe hyponatramia can result in serious neurologic complications including the risk of Central Pontine Myolosis (George & Zafar, 2018).

Hypernatraemia

Hypernatraemia is defined as a serum sodium concentration greater than 145mmol/L. Hypernatraemia occurs because of either increased sodium (hypervolemic/ hypernatraemia) with iatrogenic administration of hypertonic fluids the most common etiology or free water loss (hypovolemic hypernatraemia) including diarrhoea and sweating. Renal water loss can be due to osmotic diuresis from hyperglycaemia, mannitol administration and central diabetes insipidus. In osmotic diuresis, the urine osmolarity is high, whereas in diabetes insipidus it is low.

Diabetes Insipidus

Diabetes Insipidus (DI) and the syndrome if Innappropriate Antidiuretic Hormone lie at opposite ends of the spectrum of disordered renal handling of water (Harrois & Anstey, 2019).Central DI is a deficiency of ADH caused by destruction or degeneration of the neurons that originate in the nuclei of

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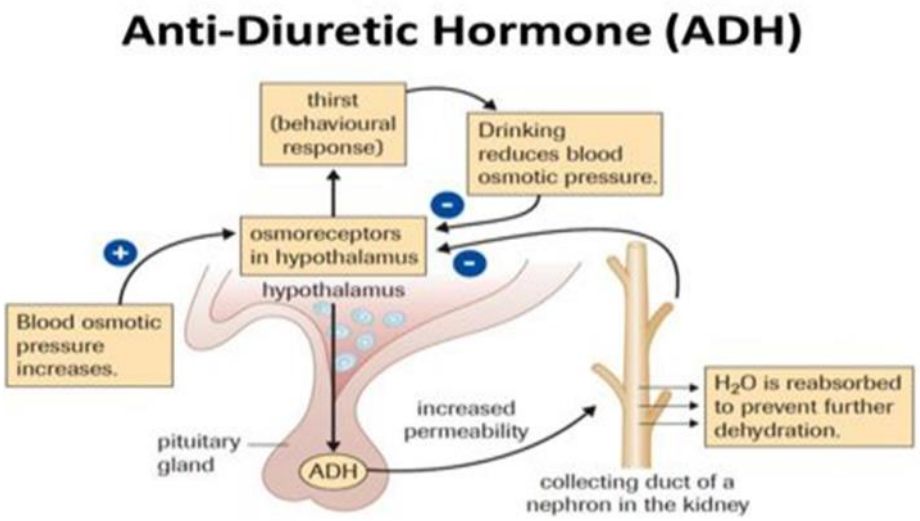
DOI: 10.21307/ajon-2021-006



the hypothalamus which may be caused by hypothalamic tumours (craniopharyngioma and germinoma) or other infiltatrative processes such as leukemia, lymphoma or sarcoidosis. DI may also be caused by the destructive lesion or post operative for removal of hypothalamic/pituitary tumours. Treatment of DI in mild cases is typically to increase water intake to even out fluid balance. When DI is caused by an abnormality in the pituitary gland or hypothalamus, such as a tumour, medication is required as treatment. This medication, desmopressin (DDAVP) replaces the missing anti-diutretic hormone and decreases urination.

It is advised to monitor the administration of DDAVP closely as too much can cause a severe drop in sodium levels.

Cerebral Salt Wasting

Cerebral salt wasting (CSW) is another potential cause of hyponatremia in those with central nervous system disease, particularly patients with subarachnoid hemorrhage. In CSW, excretion of sodium and chloride in the kidney is increased causing a reduction in extracellular volume. The low extracellular volume stimulates ADH secretion which occurs despite the progressively worsening huponatraemia. It is often seen as a response to trauma, cerebral bleeds or cerebral tumours. CSW is believed to be the most common cause of hyponatraemia in patients with subarachnoid haemorrhage. Symptoms include polyuria, polydipsia and salt cravings. Advanced symptoms include muscle cramps, vertigo and hypotension. Treatment is fluid resuscitation. As the hypovolaemia improves, the lower osmolality will inhibit ADH secretion and plasma sodium will normalize.

Syndrome of Inapropriate Antidiuretic Hormone

The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is a condition in which the body makes too much ADH. The hormone helps the kidneys control the amount of water your body excretes through the urine. The syndrome causes the body to retain water and certain levels of electrolytes in the blood to fall (such as sodium). The differentiation of SIADH from CSW is extremely important because of divergent therapeutic goals of appropriately water restricting those with SIADH and increasing salt and water with CSW to avoid iatrogenic increases in morbidity and mortality (Maesaka, Imbriano & Mattana, 2014).

SIADH generally occurs when there is insult to the central nervous system with diseases that directly stimulate the hypothalamus, the site of control of ADH secretion. Each person may experience symptoms differently. Symptoms, in more severe cases of SIADH, may include: nausea or vomiting; cramps or tremors; irritability; personality changes, such as combativeness, confusion, hallucinations; seizures or coma.

The most common form of treatment for SIADH is fluid and water restriction. If the condition is chronic, fluid restriction may need to be long term. The diagnosis and management of hyponatraemia continue to evolve. Diagnostic accuracy is improved by assessing serum and urine osmolality as well as urine sodium. Avoiding over correction of hyponatremia is crucial to avoid osmotic demyelination syndrome, although even careful correction can cause osmotic demyelination syndrome in patients who have other risk factors (Jacoby, 2020).

	CSW	**SIADH**	**Diabetes Insipidus**
Volume Status	Hypovolemia	Normovolemia or hypervolaemia	Hypovolemia
Serum Sodium Concentration	Decreased	Decreased	Increased
Urine Sodium Concentration	Increased	Increased	Decreased
Urine Output	Increased	Normal	Increased
Mechanism	Excessive secretion of sodium and water	Water retention due to elevated ADH (vasopressin)	Free water loss due to decreased ADH (vasopressin)

References

George J. Zafar W. Bucaloiu I. Chang A. Risk factors and outcomes of rapid correction of severe hyponatraemia. Journal American Society of Nephrology. 2018; 13 (7): 984-992.

Hoorn E, Zietse R. Diagnosis and treatment of hyponatraemia: compilation of the guidelines. Journal American Society of Nephrology. 2017; 28(5): 1340-1349.

Harrois A, Anstey J. Diabetes insipidus and syndrome of inappropriate antidiuretic hormone in critically ill patients. Critical Care Clinical. 2019; 35(2): 187-200.

Jacoby N. Electrolyte Disorders and the Nervous System. Neurology of Systemic Disease. 2020; 26(3): 632-658.

Maesaka J. Imbriano L. Mattana J. Differentiating SIADH from cerebral salt wasting: failure of the volume approach and need for a new approach to hyponatraemia. Journal Clinical Medicine. 2014; 3(4): 1373-1385. Verbalis J. Disorders of water metabolism: diabetes insipidus and the syndrome of inappropriate antidiuretic hormone secretion. Handbook Clinical Neurology. 2014: 124; 37 -52