Commentary

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Deficiency of Diabetes Insipidus by Vasopressin

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Description

Diabetes Insipidus (DI) is a condition characterized by large amounts of dilute urine and increased thirst. The amount of urine produced can be almost 20 liters per day. The fluid reduction has little effect on the urine concentration. Complications can be dehydration. There are four types of DI, each with different causes: The Central Diabetes Insipidus (CDI) is caused by a deficiency in the hormone vasopressin (antidiuretic hormone). This could be due to an injury to the hypothalamus or pituitary gland, or genetics. NDI) occurs when the kidneys do not respond adequately to vasopressin. Dipsogenic Diabetes Insipidus (DDI) is the result of excessive fluid intake due to damage to the hypothalamic thirst mechanism. It is more common in people with certain psychiatric disorders or who are taking certain medications. Gestation Ti occurs only during pregnancy. The term "diabetes" is derived from the Greek word "siphon". The subject of excessive and extreme thirst and increased fluid intake (especially with cold water and sometimes ice water) is typical, but the risk remains persistent. Electrolyte and volume homeostasis is a complex mechanism that balances the body's need for blood pressure and the key electrolytes, sodium and potassium. The volume is greatly reduced, the body stores water at the expense of changes in the level of electrolytes. The regulation of urine production occurs in the hypothalamus, which produces ADH in the supraoptic and paraventricular nuclei. The hormone is transported in neuro-secretory granules through the axon of the hypothalamic neuron to the posterior lobe of the pituitary gland, where it is stored for later release. alamus regulates the feeling of thirst in the ventromedial core by recognizing an increase in osmolarity. Serum and transmit this information to the central cortex/DI neurogens resulting from the ADH attack; Occasionally, it can occur with decreased thirst as the regulation of thirst and the production of ADH in the hypothalamus are very close. It is found as a result of hypoxic encephalopathy, neurosurgery, autoimmunity, or cancer, or sometimes without an underlying cause (idiopathic). The main effector organ of fluid homeostasis is the kidney. ADH works by increasing water permeability in the collecting ducts and distal convoluted tubules; In particular, it acts on proteins called aquaporins, and more specifically aquaporin 2 in the following cascade: When released, ADH binds to V2 G protein-coupled receptors in distal, tortuous tubules, increasing that associated with protein kinase A. cyclic AMP Protein kinase A pairs and stimulates the translocation of aquaporin-2 channels stored in the cytoplasm of the distal convoluted tubules and collecting channels towards the apical membrane. These transcribed channels allow water to enter multiple cells. The permeability allows water to be reabsorbed into the bloodstream, which concentrates the urine. Due to a lack of aquaporin channels in the distal collecting tube (reduced superficial expression and transcription). It is observed with lithium toxicity, hypercalcemia, hypokalemia, or release of ureteral obstruction. Therefore, the lack of ADH prevents reabsorption of water and increases the osmolarity of the blood; with increasing osmolarity, osmoreceptors in the hypothalamus recognize this change and stimulate it. This test measures changes in body weight, urine output, and composition when fluids are retained to cause dehydration. The body's normal response to dehydration is to conserve water by concentrating the urine. People with DI continue to urinate in large amounts of dilute urine despite the lack of water. In primary polydipsia, urinosmolality should increase and stabilize with fluid restriction above 280 mOsm / kg, while stabilization at a lower level indicates diabetes insipidus. In this test, stabilization means more precisely when the increase in urinosmolality is less than 30 osm/kg per hour for at least three hours. Sometimes it is also necessary to measure the level of ADH in the blood towards the end of this test, but it is more time-consuming. Desmopressin stimulation is also used to differentiate between the main forms; Desmopressin can be given as an injection, nasal spray, or tablet. While taking desmopressin, a person should only drink fluids if they are thirsty and not at other times, as this can lead to a sudden build-up of fluid in the central nervous system. Desmopressin reduces urine output and increases urinary osmolarity, hypothalamic ADH production is poor, and the kidney responds normally to exogenous vasopressin (desmopressin). If DI is due to kidney disease, desmopressin does not change urine output or osmolarity (since endogenous vasopressin levels are already high).