PSYCHOEMOTIONAL CHANGES IN DIABETES INSIPIDUS .

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https://doi.org/10.5281/zenodo.7588040

Annotation: This article describes diabetes insipidus, the mechanism of its development, and its relevance. Today's modern methods of diagnosis and treatment of diabetes mellitus, determining the genetic factor of the disease and measures to prevent its spread among the population.

Key words: antidiuretic hormone, gestational DI, fluid intake,vasopressin, urine tests, blood tests, central DI, nephrogenic DI, craniopharyngioma.

Introduction.Diabetes insipidus (DI), recently renamed to Arginine Vasopressin Deficiency (AVP-D) and Arginine Vasopressin Resistance (AVP-R), is a condition characterized by large amounts of dilute urine and increased thirst. The amount of urine produced can be nearly 20 liters per day. Reduction of fluid has little effect on the concentration of the urine. Complications may include dehydration or seizures.There are four types of DI, each with a different set of causes. Central DI (CDI) is due to a lack of vasopressin (antidiuretic hormone) production.This can be due to injury to the hypothalamus or pituitary gland or genetics. Nephrogenic DI (NDI) occurs when the kidneys do not respond properly to vasopressin. Dipsogenic DI is a result of excessive fluid intake due to damage to the hypothalamic thirst mechanism. It occurs more often in those with certain psychiatric disorders or on certain medications. Gestational DI occurs only during pregnancy. Diagnosis is often based on urine tests, blood tests and the fluid deprivation test. Diabetes insipidus is unrelated to diabetes mellitus and the conditions have a distinct mechanism, though both can result in the production of large amounts of urine.Diabetes insipidus is caused by a problem with vasopressin production in the pituitary gland (central) or the action of vasopressin in the kidneys (nephrogenic). Based on the site of its pathology, it can be divided into central diabetes insipidus (CDI) and nephrogenic diabetes insipidus (NDI). Central Diabetes Insipidus (CDI)Based on a literature review, the most common cause of Central diabetes insipidus (CDI) is idiopathic diabetes insipidus (DI). In a report of 79 participants, CDI was idiopathic in 52 percent of cases. Other cases were from a tumor or infiltrative disease in 38 percent of cases. Idiopathic CDI.Approximately, 30 to 50 percent of cases of CDI are idiopathic. They are suggested to be associated with an autoimmune etiology in most patients. The autoimmune process is characterized by lymphocytic inflammation

of the pituitary gland, specifically the pituitary stalk and the posterior pituitary gland. Early in its course, imaging of the gland (MRI pituitary gland sequence) reveals thickening or enlargement of these structures. A longitudinal study demonstrated the presence of cytoplasmic antibodies directed against vasopressin cells (Ab-positive) in patients with endocrine abnormalities. Another study of 150 patients with CDI evaluated their association with other autoimmune diseases and their correlation with imaging findings. The study reported etiology of CDI was idiopathic in 43 percent, familial in 4 percent, granulomatous in 8 percent, and an acquired cause like cranial trauma, tumor, or surgery in 45 percent of cases.[8 Antibodies to vasopressin cells were found in approximately one-third of the patients with idiopathic disease and about one-quarter with non-idiopathic disease.Antibody positivity was independently associated with age less than 30 years at disease onset in those with the idiopathic illness, a history of autoimmune disease, or pituitary stalk thickening. Autoimmune CDI was highly probable in young patients with autoimmune disease and pituitary stalk thickening history.The autoantigens involved in idiopathic CDI are not entirely elucidated. In patients with lymphocytic infundibuloneurohypophysitis (LINH), autoantibodies to rabphilin-3A, a regulator of secretory vesicle trafficking, are found in a large majority of patients. Other autoimmune conditions associated with CDI include Immunoglobulin (Ig) G4-related systemic syndrome, granulomatosis with polyangiitis (PGA), and autoimmune polyglandular syndrome type I.

Familial and congenital disease .Many familial and congenital diseases have been associated with CDI. These include familial CDI, Wolfram syndrome, proprotein convertase subtilisin/kexin type 1 (PCSK1) gene deficiency, and congenital diseases such as congenital hypopituitarism and septo-optic dysplasia.

The prognosis for most patients with DI is excellent as long as the underlying primary cause can be treated. Lithium discontinuation can restore normal kidney function, but the nephrogenic DI may be permanent in some patients.

Without medical treatment, the potential diabetes insipidus complications include:

Chronic dehydration,Tachycardia,Decreased temperature,Hypotension,Weight loss,Fatigue,Headaches,Kidney damage,Brain damage .As long as the individual has access to water, mortality can be avoided. However, the condition can lead to cardiovascular collapse, fever, and hypernatremia in children and older patients.

Diagnosis. Used to diagnose diabetes insipidus include:

Water deprivation test. While being monitored by a doctor and health care team, you'll be asked to stop drinking fluids for several hours. To prevent dehydration

while fluids are restricted, ADH allows your kidneys to decrease the amount of fluid lost in the urine.While fluids are being withheld, your doctor will measure changes in your body weight, urine output, and the concentration of your urine and blood. Your doctor may also measure blood levels of ADH or give you synthetic ADH during this test. This will determine if your body is producing enough ADH and if your kidneys can respond as expected to ADH.

Magnetic resonance imaging (MRI). An MRI can look for abnormalities in or near the pituitary gland. This test is noninvasive. It uses a powerful magnetic field and radio waves to construct detailed pictures of brain tissues.

Genetic screening. If others in your family have had problems with excess urination, your doctor may suggest genetic screening.

Treatment options depend on the type of diabetes insipidus you have.Central diabetes insipidus. If you have mild diabetes insipidus, you may need only to increase your water intake. If the condition is caused by an abnormality in the pituitary gland or hypothalamus (such as a tumor), your doctor will first treat the abnormality.

Typically, this form is treated with a synthetic hormone called desmopressin (DDAVP, Nocdurna). This medication replaces the missing anti-diuretic hormone (ADH) and decreases urination. You can take desmopressin in a tablet, as a nasal spray or by injection.Most people still make some ADH, though the amount can vary day to day. So, the amount of desmopressin you need also may vary. Taking more desmopressin than you need can cause water retention and potentially serious low-sodium levels in the blood.Other medications might also be prescribed, such as chlorpropamide. This can make ADH more available in the body.

Nephrogenic diabetes insipidus. Since the kidneys don't properly respond to ADH in this form of diabetes insipidus, desmopressin won't help. Instead, your doctor may prescribe a low-salt diet to reduce the amount of urine your kidneys make. You'll also need to drink enough water to avoid dehydration.

Treatment with the drug hydrochlorothiazide (Microzide) may improve your symptoms. Although hydrochlorothiazide is a type of drug that usually increases urine output (diuretic), it can reduce urine output for some people with nephrogenic diabetes insipidus.If your symptoms are due to medications you're taking, stopping these medicines may help. However, don't stop taking any medication without first talking to your doctor.

Gestational diabetes insipidus. Treatment for most people with gestational diabetes insipidus is with the synthetic hormone desmopressin.

Primary polydipsia. There is no specific treatment for this form of diabetes insipidus, other than decreasing fluid intake. If the condition is related to a mental illness, treating the mental illness may relieve the diabetes insipidus symptoms.
DDAVP, an ADH analog, can be administered orally, intranasally, subcutaneously, or intravenously. In adults, the dose is ten mcg by nasal insufflation or 4mcg subcutaneously or intravenously. In newborns or young infants, the dose is one mcg subcutaneously or intravenously over 20 minutes with a maximum dose of 0.4 mcg/kg.It is essential to replete fluid losses in diabetes insipidus, as some patients may have thirst impairment and will not respond adequately to water intake.Thirst is essential so that the excess urinary water losses can be replaced. Patients without an intact thirst mechanism can develop severe hypernatremia.
Conclusion.
An endocrinology consult is often warranted to identify the etiology of hypernatremia secondary to central or nephrogenic causes. A neurosurgeon is involved in cases of pituitary tumors or craniopharyngioma. Patients with significant electrolyte abnormalities should be closely monitored and admitted to the hospital.There are many causes of DI, and the disorder is best managed by an interprofessional team that includes the primary care provider, nurse practitioner/physician assistant, internist, and pharmacist. Patient education is crucial. The key is to hydrate, replace the electrolytes, and then manage the primary condition causing DI. The pharmacist should keep track of all medications that can cause DI and make the appropriate recommendations to the clinician. The nurse should educate the patient on traveling to hot destinations because dehydration can exacerbate the symptoms. If possible, travel should be avoided until the condition is treated. In post-operative patients, the specific gravity of the urine and osmolality should be monitored before administering desmopressin. Also, regular monitoring of electrolytes should be considered.The outlook for patients with DI depends on the cause. The prognosis for benign causes is good, but the prognosis is guarded if the cause is a malignancy.

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