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DISORDERS OF THE NEUROHYPOPHYSIS, DIABETES MELLITUS CAUSED BY ARGININE VASOPRESSIN DEFICIENCY AND ITS DIFFERENTIAL DIAGNOSIS

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Abstract
The neurohypophysis, or posterior pituitary, is formed by axons that originate in large cell bodies in the supra optic and paraventricular nuclei of the hypothalamus. It produces two hormones: (1) arginine vasopressin (AVP), also known as antidiuretic hormone, and (2) oxytocin. AVP acts on the renal tubules to reduce water loss by concentrating the urine. Oxytocin stimulates postpartum milk letdown in response to suckling. AVP deficiency causes diabetes insipidus (DI), which is characterized by the production of large amounts of dilute urine. Excessive or inappropriate AVP production predisposes to hyponatremia if water intake is not reduced in parallel with urine output.

Keywords: arginine vasopressin (AVP), osmoregulatory, osmoreceptors, antidiuretic effect, blood pressure, Diabetes insipidus.

Introduction

AVP is a nonapeptide composed of a six-member disulfide ring and a tripeptide tail. It is synthesized via a polypeptide precursor that includes AVP, neurophysin, and copeptin, all encoded by a single gene on chromosome 20. After preliminary processing and folding, the precursor is packaged in neurosecretory vesicles, where it is transported down the axon, further processed to AVP, and stored in neurosecretory vesicles until the hormone and other components are released by exocytosis into peripheral blood. AVP secretion is regulated primarily by the “effec tive” osmotic pressure of body fluids. This control is mediated by specialized hypothalamic cells known as osmoreceptors , which are extremely sensitive to small changes in the plasma concentration of sodium and certain other solutes but normally are insensitive to other solutes such as urea and glucose. The osmoreceptors appear to include inhibitory as well as stimulatory components that function in concert to form a threshold, or set point, control system. Below this threshold, plasma AVP is suppressed to levels that permit the development of a maximum water diuresis. Above it, plasma AVP rises steeply in direct proportion to plasma osmolarity, quickly reaching levels sufficient to effect a maximum antidiuresis. The absolute levels of plasma osmolarity/sodium at which minimally and maximally effective levels of plasma AVP occur, vary appreciably from person to person, apparently owing to genetic influences on the set and sensitivity of the system. However, the average threshold, or set point, for AVP release corresponds to a plasma osmolarity

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or sodium of about 280 mosmol/L or 135 meq/L, respectively; levels only 2–4% higher normally result in maximum antidiuresis.

Though it is relatively stable in a healthy adult, the set point of the osmoregulatory system can be lowered by pregnancy, the menstrual cycle, estrogen, and relatively large, acute reductions in blood pressure or volume. Those reductions are mediated largely by neuronal afferents that originate in transmural pres sure receptors of the heart and large arteries and project via the vagus and glossopharyngeal nerves to the brainstem, from which postsynaptic projections ascend to the hypothalamus. These pathways maintain a tonic inhibitory tone that decreases when blood volume or pressure falls by >10–20%. This baroregulatory system is probably of minor importance in the physiology of AVP secretion because the hemodynamic changes required to effect it usually do not occur during nor mal activities. However, the baroregulatory system undoubtedly plays an important role in AVP secretion in patients with large, acute disturbances of hemodynamic function. AVP secretion also can be stimulated by nausea, acute hypoglycemia, glucocorticoid deficiency, smoking, and, possibly, hyperangiotensinemia. The emetic stimuli are extremely potent since they typically elicit immediate, 50- to 100-fold increases in plasma AVP even when the nausea is transient and is not associated with vomiting or other symptoms. They appear to act via the emetic center in the medulla and can be blocked completely by treatment with antiemetics such as fluphenazine. There is no evidence that pain or other noxious stresses have any effect on AVP unless they elicit a vasovagal reaction with its associated nausea and hypotension.

The most important, if not the only, physiologic action of AVP is to reduce water excretion by promoting con centration of urine. This antidiuretic effect is achieved by increasing the hydroosmotic permeability of cells that line the distal tubule and medullary collecting ducts of the kidney (Fig. 3-2). In the absence of AVP, these cells are impermeable to water and reabsorb little, if any, of the relatively large volume of dilute filtrate that enters from the proximal nephron. This results in the excretion of very large volumes (as much as 0.2 mL/ kg per min) of maximally dilute urine (specific gravity and osmolarity ~1.000 and 50 mosmol/L, respectively), a condition known as water diuresis. In the presence of AVP, these cells become selectively permeable to water, allowing the water to diffuse back down the osmotic gradient created by the hypertonic renal medulla. As a result, the dilute fluid passing through the tubules is concentrated and the rate of urine flow decreases. The magnitude of this effect varies in direct proportion to the plasma AVP concentration and, at maximum levels, approximates a urine flow rate as low as 0.35 mL/ min and a urine osmolarity as high as 1200 mosmol/L. This action is mediated via binding to G protein–coupled V2 receptors on the serosal surface of the cell, activation of adenyl cyclase, and insertion into the luminal surface of water channels composed of a protein known as aquaporin 2 (AQP2). The V2 receptors and aquaporin 2 are encoded by genes on chromosomes Xq28 and 12q13, respectively. At high concentrations, AVP also causes contraction of smooth muscle in blood vessels and in the gastrointestinal tract, induces glycogenolysis in the liver, and potentiates adrenocorticotropic hormone (ACTH) release by corticotropin-releasing factor. These effects are mediated by V1a or V1b receptors that are coupled to phospholipase C. Their role, if any, in human physiology/pathophysiology is uncertain.

AVP distributes rapidly into a space roughly equal to the extracellular fluid volume. It is cleared irreversibly with a t1/2 of 10–30 minutes. Most AVP clearance is due to degradation in the liver

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and kidneys. During pregnancy, the metabolic clearance of AVP is increased three- to fourfold due to placental production of an N-terminal peptidase.

Because AVP cannot reduce water loss below a certain minimum level obligated by urinary solute load and evaporation from skin and lungs, a mechanism for ensuring adequate intake is essential for preventing dehydration. This vital function is performed by the thirst mechanism. Like AVP, thirst is regulated primarily by an osmostat that is situated in the anteromedial hypothalamus and is able to detect very small changes in the plasma concentration of sodium and certain other effective solutes. The thirst osmostat appears to be "set" about 5% higher than the AVP osmostat. This arrangement ensures that thirst, polydipsia, and dilution of body fluids do not occur until plasma osmolarity/sodium start to exceed the defensive capacity of the antidiuretic mechanism.

Defeciencies of vasopressin secretion and action. Diabetes insipidus. Clinical characteristics: decreased secretion or action of AVP usually mani fests as diabetes insipidus, a syndrome characterized by the production of abnormally large volumes of dilute urine. The 24-hour urine volume is >50 mL/kg body weight, and the osmolarity is Deficient secretion of AVP can be primary or secondary. The primary form usually results from agenesis or irreversible destruction of the neurohypophysis and is referred to variously as neurohypophyseal DI, pituitary DI, or central DI. It can be caused by a variety of congenital, acquired, or genetic disorders, but in about one-half of all adult patients it is idiopathic. The surgically induced forms of pituitary DI usually appear within 24 hours and then go through a 2- to 3-week interim period of inappropriate antidiuresis, after which they may or may not recur. The genetic form usually is transmitted in an autosomal dominant mode and is caused by diverse mutations in the coding region of the AVP–neurophysin II (or AVP-NPII) gene. All the mutations alter one or more amino acids known to be critical for correct folding of the prohormone, thus interfering with its processing and trafficking through the endoplasmic reticulum. The AVP deficiency and DI develop gradually several months to years after birth, progressing from partial to severe and permanent DI. They appear to result from accumulation of misfolded mutant precursor followed by selective degeneration of AVP-producing magnocellular neurons. An autosomal recessive form due to an inactivating mutation in the AVP portion of the gene, an X-linked recessive form due to an unidentified gene on Xq28, and an autosomal recessive form due to mutations of the WFS 1 gene responsible for Wolfram's syndrome [diabetes insipidus, diabetes mellitus, optic atrophy, and neural deafness (DIDMOAD)] have also been described. A primary deficiency of plasma AVP also can result from increased metabolism by an N-terminal aminopeptidase produced by the placenta. It is referred to as gestational DI since the signs and symptoms manifest during pregnancy and usually remit several weeks after delivery. Secondary deficiencies of AVP result from inhibition of secretion by excessive intake of fluids. They are referred to as primary polydipsia and can be divided into three subcategories. One of them, dipsogenic DI, is characterized by inappropriate thirst caused by a reduction in the set of the osmoregulatory mechanism. It some times occurs in association with multifocal diseases of the brain such as neurosarcoid, tuberculous meningitis, and multiple sclerosis but is often idiopathic. The second subtype, psychogenic polydipsia, is not associated with thirst, and the polydipsia seems to be a feature of psychosis or obsessive compulsive disorder. The third SECTION I Pituitary, Thyroid, and Adrenal Disorders subtype, iatrogenic polydipsia, results from recommendations to increase fluid intake for its presumed health benefits. Primary

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deficiencies in the antidiuretic action of AVP result in nephrogenic DI. They can be genetic, acquired, or drug induced. The genetic form usually is transmitted in a semirecessive X-linked manner and is caused by mutations in the coding region of the V2 receptor gene that impair trafficking and/ or ligand binding of the mutant receptor. Autosomal recessive or dominant forms are caused by AQP2 gene mutations that result in complete or partial defects in trafficking and function of the water channels in distal and collecting tubules of the kidney. Secondary deficiencies in the antidiuretic response to AVP result from polyuria per se. They are caused by washout of the medullary concentration gradient and/ or suppression of aquaporin function. They usually resolve 24–48 hours after the polyuria is corrected but can complicate interpretation of some acute tests used for differential diagnosis.

Differential diagnosis: When symptoms of urinary frequency, enuresis, nocturia, and/or persistent thirst are present, the possibility of DI should be evaluated after excluding glucosuria by collecting a 24-hour urine on ad libitum fluid intake. If the volume exceeds 50 mL/kg per day (3500 mL in a 70-kg male) and the osmolarity is < 300 mosmol/L, DI is confirmed and the patient should be evaluated further to determine the type.

In differentiating among the various types of DI, the history alone may be sufficient if it reveals a likely ante cedent such as pituitary surgery. Usually, however, that type of indicator is absent, ambiguous, or misleading and other approaches are needed. Except in the rare patient with hypertonic dehydration under basal conditions, differentiation should begin with a fluid deprivation test. It can be performed on an outpatient basis if the necessary staff and facilities are available. To minimize patient dis comfort, avoid excessive dehydration, and maximize the information obtained, the test should be started in the morning and continued with hourly monitoring of body weight, plasma osmolarity and/or sodium concentration, urine volume, and urine osmolarity until either of two endpoints is reached. If fluid deprivation does not result in urine concentration (osmolarity > >300 mosmol/L, specific gravity >1.010) before body weight decreases by 5% or plasma osmolarity/sodium rise above the upper limit of normal, the patient has severe pituitary or severe nephrogenic DI. These disorders usually can be distinguished by administering desmopressin (0.03 µg/kg SC or IV) and repeating the measurement of urine osmolar ity 1–2 hours later. An increase of >50% indicates severe pituitary DI, whereas a smaller or absent response is strongly suggestive of nephrogenic DI.

An alternative method of differential diagnosis is MRI of the pituitary and hypothalamus. In most healthy adults and children, the posterior pituitary emits a hyperintense signal in T1-weighted midsagittal images. This “bright spot” is almost always present in patients with primary polydipsia but is invariably absent or abnormally small in patients with pituitary DI. It is usually also small or absent in nephrogenic DI presumably because of high secretion and turnover of AVP. Thus, a normal bright spot virtually excludes pituitary DI, argues against nephrogenic DI, and strongly suggests primary polydipsia. Lack of the bright spot is less help ful, however, because it is absent not only in pituitary and nephrogenic DI but also in some healthy adults and patients with empty sella who do not have DI or AVP deficiency.

In conclusion, the signs and symptoms of uncomplicated pituitary DI can be eliminated completely by treatment with desmopressin (DDAVP), a synthetic analogue of AVP. DDAVP acts selectively at V2 receptors to increase urine concentration and decrease urine flow in a dose-dependent

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manner. It is also more resistant to degradation than is AVP and has a three- to fourfold longer duration of action. Desmopressin can be given by IV or SC injection, nasal inhalation, or oral tablet. The doses required to control pituitary DI completely vary widely, depending on the patient and the route of administration.

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