Diabetes Insipidus: An Overview

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The term "diabetes" refers to prolific urine flow. Diabetes mellitus, a chronic endocrine disease, is characterized by insulin deficiency or resistance, whereas diabetes insipidus—a far more rare condition—results from antidiuretic hormone (ADH or vasopressin) deficiency. Insufficient ADH leads to polyuria (passage of a large volume of urine in a given time frame) and polydipsia (chronic, excessive thirst).

A recent increase in the incidence of diabetes insipidus has been correlated with the use of hypophysectomy—removal of the pituitary gland—to treat breast cancer. However, in 50% of diabetes insipidus cases, causation can be traced to familial or idiopathic factors. Secondary diabetes insipidus can result from intracranial neoplasms or metastatic lesions, hypophysectomy or other intracranial surgery, or head trauma that includes damage to the neurohypophyseal structures. Infection, granulomatous disease, and vascular lesions can also cause diabetes insipidus. Prognosis for complete recovery is good when diabetes insipidus is not complicated by an underlying disorder.

FORMS, CAUSES, AND PATHOPHYSIOLOGY
Diabetes insipidus takes one of three forms: neurogenic, nephrogenic, or psychogenic. Neurogenic diabetes insipidus often results when a lesion involving the hypothalamus, infundibular stem, or posterior pituitary interferes with the synthesis, transport, or release of ADH. Since it can also occur following hypophysectomy, the neurogenic form would be of more interest to the surgical technologist than the two other forms of the disease. Nephrogenic diabetes insipidus results from inadequate renal tubule response to ADH, which is usually caused by disorders and/or drugs that damage the renal tubules. The psychogenic form of diabetes insipidus is driven by psychological factors that lead to an abnormally high intake of fluids, which results in decreased ADH levels.

The basic pathophysiology of diabetes insipidus—the inability to concentrate urine—results from chronic production of large amounts of urine causing a "washout" phenomenon in the renal medullary concentration gradient. ADH controls final urine product concentration by increasing water permeability in the last segment of the distal tubule and in the collecting ducts that pass through the inner and outer zones of the medulla. When ADH is present, water resorption increases because of the high osmotic gradient in the medullary interstitium. Water then diffuses into the ascending limb of the vasa recta and is returned to the systemic circulation.

Insufficient ADH causes immediate increased production of dilute urine, increased plasma osmolality, and triggers the thirst mechanism. In about half the cases, urine output varies from 4 L to 8 L per day. Output increases to as much as 12 L per day in a quarter of the cases. Specific gravity of urine remains low and serum electrolytes are usually unaffected; however, dehydration will develop quickly without adequate fluid replacement.
SYMPTOMS
Diabetes insipidus produces the following symptoms, which usually begin and end abruptly:
- Extreme polyuria (4 L to 16 L—or more—of dilute urine per day)
- Polydipsia (often combined with a preference for cold fluids)
- Slight nocturia
- Fatigue (in extreme cases) from inadequate rest caused by excessive voiding
- Symptoms of dehydration
  The related symptoms of dehydration include the following:
  - Poor skin turgor
  - Dry mucous membranes
  - Constipation
  - Muscle weakness
  - Dizziness
  - Hypotension

DIAGNOSIS AND TREATMENT
Diagnostic procedures involve establishing baseline vital signs, weight, urine osmolality, and plasma osmolality. Fluid intake is then eliminated, and hourly measurements are taken of urine output, body weight, urine osmolality, and plasma osmolality. Testing continues until the patient loses 3% of body weight—indicating severe dehydration—or until severe postural hypotension occurs. However, testing may be terminated if urine osmolality does not rise after taking three consecutive samples or if plasma osmolality is greater than normal. Once diabetes insipidus has been diagnosed, a subcutaneous injection of vasopressin (5 units) is necessary to distinguish between the neurogenic and nephrogenic forms. Patients with nephrogenic diabetes insipidus will respond to vasopressin with decreased urine output, while those with neurogenic diabetes insipidus will have no change in urine output.

In an effort to correct the condition, one or combinations of several forms of vasopressin may be administered to control fluid balance and prevent dehydration. Fluids must also be replaced, and intake and output must be monitored to prevent dehydration and hypovolemic shock. Constipation may require both preventive and treatment efforts. Patients with coronary artery disease should be given special attention since vasopressin is an arterial constrictor.

SUMMARY
Diabetes insipidus is a potentially dangerous condition resulting from one of three causes: low levels of ADH, inadequate renal tubule response to ADH, or psychological factors. Neurogenic diabetes insipidus can appear following hypophysectomy (see the continuing education article entitled "Transsphenoidal Approach to Pituitary Tumors" in this issue of the journal). While the patient may experience several difficult days before resolution of diabetes insipidus, in the absence of underlying disorders, the prognosis is good.

REFERENCES

The Surgical Wound
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