Case Reports

Psychogenic Diabetes Insipidus

A Case Report with Description of Certain Differential Diagnostic Procedures

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Diabetes insipidus is a clinical syndrome characterized by the passage of large volumes of dilute urine and the presence of an inordinate thirst. Polydipsia and polyuria may occur in organic disturbances of the mechanism of thirst,7 lesions of the supraoptical-neurohypophyseal system,8,10,12 familial and acquired renal tubular dysfunction,11,14,15,17 potassium deficiency,15 hypercalcemia,18 acute and chronic renal disease,13 diabetes mellitus, administration of diuretics, and miscellaneous clinical disorders.

Designation of polydipsia and polyuria as indicative of factitious or psychogenic diabetes insipidus implies that the patient has no fundamental disturbance of the mechanism regulating the balance of body water, but continues to drink large amounts of water as a manifestation or a consequence of a personality disorder.4 This communication is a report of an individual who ingested and excreted over 30 l. of fluids per day and who was admitted to the neurosurgical service with the presumptive diagnosis of true diabetes insipidus and brain tumor. The difficult differential diagnosis is reviewed.

Case Report

A 34-year-old white man was admitted to the Neurosurgical Service of the University of Florida Teaching Hospital complaining of insatiable thirst that compelled him to drink over 7 gallons of water per day. Although chronically anxious, the patient had enjoyed good health until 5 years previously, when manifestations of agitated depression developed and he began drinking excessive amounts of water. The frequent ingestion of water and urination caused him no concern; there was no associated desire for cold water. He was employed at a local water works plant. Subsequent electric-shock therapy improved his mood, but the marked degree of polydipsia and polyuria continued. There was no history of headache, seizures, visual disturbances, motor or sensory deficit, diabetes mellitus, renal disease, anorexia, or loss of weight. He was referred to the University Hospital with a tentative diagnosis of true diabetes insipidus secondary to a suspected brain tumor.

Examination. No focal neurological deficit could be demonstrated. The patient was oriented, intelligent, and very anxious. He was placed in the intensive care unit where careful observations of oral intake of fluid and urinary output were recorded. These averaged 23 to 30 l. per 24 hours; the specific gravity of urine was never greater than 1.005. Complete blood count, urinalysis, urine culture, serologic test for syphilis, blood chemistries including blood sugar, creatinine, electrolytes, calcium, phosphorus, and protein-bound iodine were within normal limits. The serum osmolality was 285 mOsm./l. and the urine osmolality was 170 mOsm./l. X-rays of the chest and skull, mercury brain scan, spinal fluid, and charted visual fields were normal. The neurosurgical staff in consultation with the Section of Endocrinology performed a series of diagnostic tests to confirm the initial clinical impression of psychogenic polydipsia (factitious diabetes insipidus). A modified Hickey-Hare procedure was carried out in which the patient received an intravenous infusion of 2.5 per cent saline administered at a rate of 0.2 cc./kg./min. over a period of 45 minutes; urine was collected at 15-minute intervals. The volume of urine decreased from 16.5 to 9 ml./min. and the specific gravity rose to 1.015. The administration of 0.1 unit of aqueous Pitressin intravenously produced no significant change.

A careful study during deprivation of water was then carried out over a 48-hour period in which fluids were restricted to 2 l. per day. The patient was under constant observation. The specific gravity of urine rose from 1.003 to 1.014. At the end of a 5-day period on this regime a specific gravity of urine of 1.045 was obtained. It was therefore concluded that the hypothalamic-hypophyseal-renal systems were intact.

Consulting psychiatrists agreed that the patient had a marked schizoid personality disorder and that the factitious polydipsia was a source of oral gratification. The patient was discharged from the hospital, advised to restrict intake of water to comfortable levels, and placed on appropriate dosage of chlorpromazine. He is being followed in the psychiatric and neurological-neurosurgical clinics.

Comment

The diagnosis of true diabetes insipidus is usually established on the basis of history, laboratory findings, and the patient’s inability to form a concentrated urine except after the administration of antidiuretic hormone (ADH). The differential diagnosis, in a patient with polyuria and polydipsia may be difficult despite our knowledge of the pathogenesis of this syndrome and the variety of tests which permit a more precise evaluation (Table 1).

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Polydipsia is usually the result of a partial or complete absence or suppression of ADH. The action of this hormone is to increase the rate of reabsorption of water in the distal convoluted and collecting tubules in response to alterations in the osmolarity of the blood. Since healthy renal tubules reabsorb as much as 100 l. of water daily, interference with only a small percentage of this reabsorption will lead to an enormous increase in the volume of urine.\(^2\)

Characteristically, the patient with true diabetes insipidus will note the abrupt onset of polyuria and intense thirst. The urinary output varies between 5–15 l. per day, but has been reported as high as 48 l. per day. He prefers to drink iced fluids. Deprivation of water is often intolerable. The nephrogenic form of diabetes insipidus becomes manifest in early childhood and, with rare exceptions, is familial. Patients with factitious polydipsia often give a history of an insidious onset of polyuria and polydipsia. A psychoneurotic or psychotic disturbance is invariably present. A preference for iced fluids is most unusual.

Although these basic aspects are helpful in differentiating diabetes insipidus and compulsive polydipsia, objective criteria are desirable which prove the integrity of the hypothalamic-hypophyseal system as well as the tubular response to ADH. Biologic methods for the direct estimation of ADH in the serum and urine have met with mixed success and remain controversial.\(^4\) Indirect tests designed to demonstrate that the patient can initiate antidiuresis under standardized conditions constitute the accepted methods of indicating ADH activity. These tests are:

1. Effect of vasopressin (ADH) on polyuria and the specific gravity of the urine.
2. Deprivation of water.
3. Infusion of hypertonic saline.
4. Stimulation of hypothalamic nuclei with nicotine.

The prompt and complete relief from polyuria, elevation of the specific gravity to 1.015, and the rehydration of the patient following the administration of a therapeutic dose of vasopressin are significant indications of diabetes insipidus. The recurrence of the manifestations of uncontrolled diabetes insipidus after the effect of the injection of vasopressin has passed is final positive evidence.

The water deprivation test\(^3\) is helpful in differentiating between the polyuria of diabetes

### TABLE 1

**The clinical types and differential diagnosis of diabetes insipidus**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Urine Specific Gravity (SG)</th>
<th>Duration of Polyuria</th>
<th>Evidence of Renal Disease</th>
<th>Glycosuria</th>
<th>Effect of H2O Restriction</th>
<th>Effect of Salt Loading</th>
<th>Effect of Decrease in Solute Load</th>
<th>Response to Pitressin</th>
<th>Response to Chlorothiazide</th>
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</thead>
<tbody>
<tr>
<td>Supraspinal-hypophyseal diabetes insipidus</td>
<td>1.001±</td>
<td>Continuous</td>
<td>None</td>
<td>None</td>
<td>No Effect</td>
<td>No Effect</td>
<td></td>
<td>No Effect</td>
<td>Urine: S.G.↓ Vol.↓</td>
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<td>- Constitutional</td>
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<tr>
<td>Chronic renal disease</td>
<td>1.010±</td>
<td>Continuous</td>
<td>Yes</td>
<td>None</td>
<td>No Effect</td>
<td>No Effect</td>
<td>Minimum Effect</td>
<td></td>
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<tr>
<td>Diuresis</td>
<td>1.06±</td>
<td>Transient</td>
<td>None</td>
<td>None</td>
<td>No Effect</td>
<td>No Effect</td>
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<td></td>
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<tr>
<td>Diabetes mellitus</td>
<td>1.02±</td>
<td>Variable</td>
<td>Maybe</td>
<td>Glycosuria with marked hyperglycemia</td>
<td>No Effect</td>
<td>Urine: S.G.↓ Vol.↓ (if glyco)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(\uparrow\) = Increase; \(\downarrow\) = Decrease; \(-\) = No Change
insipidus and that of either psychogenic or nephrogenic origin. A true deficiency or absence of ADH will be indicated by slight, if any, change in the volume or specific gravity of the urine during the 6 hours of dehydration, and appreciable reduction in body weight, and a significant increase in the hematocrit, as well as a marked increase in serum osmolarity. Polyuria of psychogenic origin is indicated if during the test, a prompt increase in the specific gravity of the urine occurs, the volume of urine decreases, and there is little or no change in body weight or hematocrit.

The hypertonic saline infusion test, by increasing the plasma osmolarity, provides a stimulus for antidiuresis if ADH is present. As described in detail by Carter and Robbins;4,5 2.5 per cent hypertonic saline is given intravenously at a rate of 0.25 cc./kg./min. for 45 minutes. In patients with diabetes insipidus, in contrast to those with psychogenic polydipsia and normal persons, there will be no appreciable reduction in the flow of urine in the first 30 minutes. If the flow of urine does not decrease in this half-hour interval, the effect of vasopressin, 0.1 unit given intravenously, on the flow of urine is observed. The failure to reduce the flow of urine by attempting to stimulate production of ADH by the hyperosmolarity induced by hypertonic saline and the abrupt reduction in the flow of urine after the administration of Pitressin (vasopressin) are characteristics of true diabetes insipidus (Fig. 1).

Stimulation of the hypothalamic nuclei with nicotine6 may be of value in differentiating obliteration of the function of the hypothalamic nuclei and osmoreceptor function from instances of selective blocking of osmoreceptor function with the hypothalamic nuclei remaining intact. Essentially, 1 to 3 mg. of nicotine base is administered at a time when the polyuria is pronounced. The response in normal and polydipsic subjects is indicated by reduction of the urinary output by approximately 80 per cent. Patients with diabetes insipidus show little or no response to this test.

A very simple test involves determining the serum osmolarity in untreated patients with polyuria and polydipsia.7 The patient with true diabetes insipidus usually will have a low serum osmolarity (i.e. less than 170 mOsm/L), whereas it is normal or low-normal in the compulsive drinking patient.

A most helpful and less well known observation in the differential diagnosis has been a therapeutic test contrasting the effect of a placebo with that of a small dose of pitressin tannate. An injection of sesame or peanut oil is used as a placebo. In the patient with psychogenic polydipsia there is often a marked reduction in thirst and volume of urine, whereas in the patient with true diabetes insipidus there is no significant response. The intramuscular administration of 0.2 ml. Pitressin tannate is followed by a prompt reduction in thirst and urinary volume in the patient with true diabetes insipidus. The patient with psychogenic polydipsia exhibits the same response as to the placebo or continues to drink large amounts of water despite reduction in urinary volume.8

In general, most observers would consider the causes of diabetes insipidus in man in 8 categories. In a series of 65 patients seen at the Johns Hopkins Hospital during a 20-year period, one third had diabetes insipidus attributed to an intracranial tumor, either primary or metastatic involving the hypothalamus or neurohypophysis. In another third of the patients no specific etiology could be determined. In the remaining third, the diabetes insipidus was secondary to a wide variety of diseases. That a search for a specific etiology should be continued in patients with "idiopathic" diabetes insipidus was demonstrated by 2 patients who within 5 years after initial evaluation were found to have intracranial tumors.9

Summary

Diabetes insipidus is a clinical syndrome of varied etiology characterized by the passage of

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**Fig. 1.** The results of a hypertonic saline infusion test in a normal subject, a patient with true diabetes insipidus, and a patient with psychogenic polydipsia are shown. Note the lack of antidiuretic response to an increase in plasma osmolarity contrasted with the response in the normal subject and the patient with factitious polydipsia.
large volumes of dilute urine and the presence of inordinate thirst. In general, one third of such patients are found to have an intracranial tumor involving the hypothalamus or neurohypophysis; in another third it is secondary to a wide variety of disease; and in the remaining one third no specific etiology can be determined. The differential diagnosis in patients with suspected diabetes insipidus frequently taxes the ingenuity of the physician because historical and laboratory findings may not be classical. Indeed, the true cause may not be discovered until a later date.

Psychogenic aberration or habit may cause a patient to drink enormous amounts of fluids. Plasma osmolarity is subsequently decreased which, in turn, suppresses ADH secretion. Polyuria factitia ensues. The presence of a psychoneurotic disorder, the absence of a preference for iced fluids, and prompt antidiuresis in response to the administration of hypertonic saline, nicotine, or water deprivation will usually establish the diagnosis.

References