Arginine vasopressin deficiency

Terminology: Central Diabetes Insipidus vs Arginine Vasopressin Deficiency

Both terms are used, but the terminology is evolving. Here's a comparison to guide usage:

Central Diabetes Insipidus

- **Definition**: Classic term for reduced vasopressin secretion from the hypothalamus/posterior pituitary.
- **Abbreviation**: CDI
- **Context**: Widely used in clinical practice, textbooks, and ICD coding.
- **Limitations**:
  - Can be confused with diabetes mellitus.
  - Describes symptoms more than cause.

Arginine Vasopressin Deficiency

- **Definition**: Precise, pathophysiology-based term indicating a deficiency of AVP (also called ADH).
- **Abbreviation**: AVP-D
- **Context**: Increasingly used in modern research and guidelines.
- **Advocated by**: European Society of Endocrinology and patient advocacy groups.
- **Advantages**:
  - Reduces confusion with diabetes mellitus.
  - Reflects the hormonal nature of the disease.

When writing for research or academic audiences, prefer **Arginine Vasopressin Deficiency (AVP-D)**. In clinical documentation, **Central Diabetes Insipidus** may still be appropriate.

Arginine vasopressin (AVP) deficiency, or central diabetes insipidus, is a common complication after pituitary surgery.

Early detection is critical and can be done by identifying hypotonic urine, defined as urine with specific gravity < 1.005 g/mL.

 Objective To assess whether patients can accurately use urine test strips to screen for AVP deficiency, compared to measurements done by nurses using a refractometer.

 Methods Setting: Neurosurgical inpatient ward.

Participants: 110 patients provided 609 urine samples.

Tools:

Patients used Combur-10 urine test strips.

Nurses used an ATAGO MASTER-SUR/Nα refractometer.

Agreement analysis:

Weighted Kappa

Intraclass Correlation Coefficient (ICC)

 Results Patient vs. nurse (refractometer):

Moderate agreement (Kappa = 0.47, ICC = 0.69)

Patient vs. nurse (both using test strips):

Substantial to good agreement (Kappa = 0.82, ICC = 0.89)

A cut-off of 1.015 g/mL on test strips ensured no cases of hypotonic urine were missed, and could reduce nurse workload by 50%.

Satisfaction scores:

Patients: 7.8 / 10

Nurses: 6.4 / 10

 Conclusions Patients can reliably self-screen for hypotonic urine using urine test strips.

Though less precise than nurses, using a higher threshold (1.015 g/mL) makes the method safe and effective.

This approach promotes patient involvement and reduces nurse workload in postoperative monitoring of AVP deficiency.

Diabetes insipidus is either a problem with the production of antidiuretic hormone (central diabetes insipidus) or kidney's response to antidiuretic hormone (nephrogenic diabetes insipidus).

The most common type in humans is the neurological form, called (central diabetes insipidus) (CDI), which involves a deficiency of arginine vasopressin (AVP), also known as antidiuretic hormone (ADH).

Also called neurogenic diabetes insipidus. It is also known as neurohypophyseal diabetes insipidus referring to the posterior pituitary (neurohypophysis), which is supplied by the hypothalamus in the brain. This condition has only polyuria in common with diabetes and although not mutually exclusive, with most typical cases, the name diabetes insipidus is a misleading misnomer.

A better name might be “hypothalamic-neurohypophyseal ADH deficiency”.

Epidemiology

Central diabetes insipidus epidemiology.

Etiology

Central diabetes insipidus etiology.

Treatment

In patients with central DI, desmopressin is the drug of choice.

A synthetic analogue of antidiuretic hormone (ADH), desmopressin is available in subcutaneous, IV, intranasal, and oral preparations.

Generally, it can be administered 2-3 times per day. Patients may require hospitalization to establish fluid needs. Frequent electrolyte monitoring is recommended during the initial phase of treatment.

Alternatives to desmopressin as pharmacologic therapy for DI include synthetic vasopressin and the nonhormonal agents chlorpropamide, carbamazepine, clofibrate (no longer on the US market), thiazides, and nonsteroidal anti-inflammatory drugs (NSAIDs). Because of side effects, carbamazepine is rarely used, being employed only when all other measures prove unsatisfactory. NSAIDs (eg, indomethacin) may be used in nephrogenic DI, but only when no better options exist. In central DI, the primary problem is a hormone deficiency; therefore, physiologic replacement with desmopressin is usually effective. Use a nonhormonal drug for central DI if response is incomplete or desmopressin is too expensive.

Outcome

Combined central diabetes insipidus and cerebral salt wasting syndrome is a rare clinical finding. However, when this happens, mortality is high due to delayed diagnosis and/or inadequate treatment.

Case report

A 42-year-old white man was referred to neurosurgery due to a nonfunctioning pituitary macroadenoma. He underwent a partial resection of the tumor on July 2, 2015. On the day following surgery he presented polyuria with sodium 149 mEq/L, plasma osmolality 301 mOsm/kg, and urine osmolality 293 mOsm/kg. He started nasal desmopressin 0.05 mg/day with good response. He was already on dexamethasone 4 mg and levothyroxine 75 mcg due to hypopituitarism after surgery. On July 9 he became confused. Cerebral computed tomography was performed with no significant

changes. His natremia dropped to 128 mEq/L with development of polyuria despite maintenance of desmopressin dose. His hemoglobin and hematocrit rose from 9.1 g/L to 11.6 g/L and 27.5 to 32.5, respectively. His thyroid function was normal and he was on hydrocortisone 30 mg/day. At 12 p.m. 150 mg/hydrocortisone infusion was initiated, but sodium did not increase. Plasma and urine osmolality were 264 mOsm/kg and 679 mOsm/kg, respectively. At 4 p.m. hydrocortisone was increased and hypertonic saline replacement started. Two hours later he was dehydrated with polyuria and vomiting, and natremia of 124 mEq/L. Hyponatremia was very resistant to treatment despite hypertonic saline replacement, hence desmopressin was suspended. The following day, urine spot analysis showed that natriuresis was 63 mEq/L with serum sodium 132 mEq/L. This was interpreted as a cerebral salt wasting syndrome and control was achieved with aggressive hypertonic saline replacements and fludrocortisone 0.1 mg/three times a day.

Costa et al., from the Centro Hospitalar de São João, Porto, Portugal, present a rare case of a patient with diabetes insipidus and cerebral salt wasting syndrome, who was successfully treated. Hyponatremia in a patient with diabetes insipidus may erroneously be interpreted as inadequate diabetes insipidus control or as syndrome of inappropriate antidiuretic hormone secretion, leading to therapeutic errors. Thus, all clinical and analytical data should be evaluated together for early and correct diagnosis [1)].

Key concepts

● due to low levels of ADH (or, rarely, renal insensitivity to ADH)

● high output of dilute urine (< 200 mOsmol/kg or SG < 1.003) with normal or high plasma osmolality and high serum sodium.

● often accompanied by a craving for water, especially ice-water

● danger of severe dehydration if not managed carefully

Definition

Although they have a common name, diabetes mellitus and diabetes insipidus are two entirely separate conditions with unrelated mechanisms. Both cause large amounts of urine to be produced (polyuria), and the term “diabetes” is derived from the Greek word meaning siphon. However, diabetes insipidus is either a problem with the production of antidiuretic hormone (central diabetes insipidus) or kidney's response to antidiuretic hormone (nephrogenic diabetes insipidus), whereas diabetes mellitus causes polyuria via a process called osmotic diuresis, due to the high blood sugar leaking into the urine and taking excess water along with it.

Diabetes insipidus (DI) is a condition characterized by excessive thirst and excretion of large amounts of severely diluted urine, with a reduction of fluid intake having no effect on the concentration of the urine.

Classification

Diabetes insipidus classification.

Epidemiology

The incidence of diabetes insipidus in the general population is 3 in 100,000.

Etiology

Central diabetes insipidus etiology.

Nephrogenic diabetes insipidus etiology.

Diagnosis

Arginine vasopressin deficiency diagnosis

Treatment

The scarcity of studies comparing different treatment and monitoring strategies for these disorders and the lack of prior clinical guidelines makes it difficult to provide recommendations following a methodology based on grades of evidence [2)].

Most patients with diabetes insipidus (DI) can drink enough fluid to replace their urine losses. When oral intake is inadequate and hypernatremia is present, replace losses with dextrose and water or an intravenous (IV) fluid that is hypo-osmolar with respect to the patient's serum. Do not administer sterile water without dextrose intravenously, as it can cause hemolysis.

To avoid hyperglycemia, volume overload, and overly rapid correction of hypernatremia, fluid replacement should be provided at a rate no greater than 500-750 mL/h. A good rule of thumb is to reduce serum sodium by 0.5 mmol/L (0.5 mEq/L) every hour. The water deficit may be calculated on the basis of the assumption that body water is approximately 60% of body weight.

Case series

Diabetes insipidus case series.

Case reports

A 42-Year-Old Male with Diabetes Insipidus [3].

Books

MRI of the Pituitary Gland By Jean-François Bonneville, Fabrice Bonneville, Françoise Cattin, Sonia Nagi

This clinically oriented book will familiarize the reader with all aspects of the diagnosis of tumors and other disorders of the pituitary gland by means of magnetic resonance imaging (MRI). The coverage includes acromegaly, Cushing's disease, Rathke cleft cysts, prolactinomas, incidentalomas, Clinically Non-Functioning Pituitary Neuroendocrine Tumors, other lesions of the sellar region, hypophysitis, and central diabetes insipidus. Normal radiologic anatomy and the numerous normal variants are described, and guidance is also provided on difficulties, artifacts, and other pitfalls. The book combines concise text and high-quality images with a question and answer format geared toward the needs of the practitioner. MRI is today considered the cornerstone in the diagnosis of diseases of the hypophyseal-hypothalamic region but the relatively small size of the pituitary gland, its deep location, the many normal anatomic variants, and the often tiny size of lesions can hinder precise evaluation of the anatomic structures and particularly the pituitary gland itself. Radiologists and endocrinologists will find MRI of the Pituitary Gland to be full of helpful information on this essential examination, and the book will also be of interest to internists and neurosurgeons.

1)
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2)
Lamas C, del Pozo C, Villabona C; Neuroendocrinology Group of the SEEN.. Clinical guidelines for management of diabetes insipidus and syndrome of inappropriate antidiuretic hormone secretion after pituitary surgery. Endocrinol Nutr. 2014 Apr;61(4):e15-24. doi: 10.1016/j.endonu.2014.01.005. English, Spanish. PubMed PMID: 24588923.
3)
Bremer Pais I, Bernreuther C, Minnemann T, Saeger W, Hagel C, Iking-Konert C, Aberle J, Flitsch J. A 42-Year-Old Male with Diabetes Insipidus. Brain Pathol. 2017 Sep;27(5):695-696. doi: 10.1111/bpa.12541. PubMed PMID: 28805007.