



## Central Diabetes Insipidus is caused by the Neuroendocrine Disorders contains Antidiuretic Hormones

Morze Karolina\*

Department of Endocrinology, University of Antwerp, Belgium

### INTRODUCTION

Once Central Diabetes Insipidus (CDI) is diagnosed, every effort should be made to determine the underlying cause. The autoimmune etiology of CDI was first suggested in 1983 in adults and children with this disease by the detection of autoantibodies against hypothalamic vasopressin-producing cells (AVPcAb) using an indirect immunofluorescence assay. Rabfilin-3A, a protein of secretory vesicles of the neurohypophyseal system, has now been identified as the major autoantigen in autoimmune CDI. Autoimmune CDI is primarily detected in patients diagnosed with autoimmune endocrine disorders. A radiological and morphological correlation with autoimmune DI is lymphocytic infundibulum neurohypophysitis (LINH), substantiated by magnetic resonance imaging and biopsy, and associated with lymphocytes and some plasma cells, shows massive infiltration of the posterior pituitary and infundibulum and fibrosis in late stages of the disease.

### DESCRIPTION

However, in most cases it is temporary. It is defined as excretion of abnormally large amounts of dilute urine associated with increased serum osmolality. The incidence of CDI after pituitary surgery has been reported to be 0%-90%. Large tumor size, total resection, and perioperative cerebrospinal fluid leakage usually increase the risk of CDI, as seen in craniopharyngioma and Rathke's fissure cysts. CDI can lead to high morbidity and mortality if not recognized and treated in time. It is also important to rule out other causes of postoperative polyuria to avoid unnecessary medications and iatrogenic hyponatremia. Once a diagnosis of CDI has been made, close monitoring is required to assess response to treatment and determine whether CDI is transient or permanent. This review article describes the evaluation and management of his CDI patient after surgery for pituitary and suprasellar tumors to help establish the diagno-

sis, discuss differential diagnoses, initiate therapeutic interventions, and guide surveillance and long-term treatment.

Insufficient production of the antidiuretic hormone arginine vasopressin (AVP) by magnocellular neurons that make up the posterior pituitary gland (hypothalamic DI); renal dysfunction of AVP (nephrogenic DI); decreased AVP secretion due to excessive water intake or degradation of AVP by placental vasopressinase (gestational DI). All types of DI can be caused or exacerbated by other medical conditions. Hypothalamic and renal DI can also be caused by mutations in genes encoding AVP prohormones, its AVP-2 receptors in the kidney, or the aquaporin-2 water channel that mediates antidiuresis. Familial hypothalamic DI is usually inherited in an autosomal dominant manner, but there are also autosomal recessive or X-linked recessive forms. Familial nephrogenic DI is usually inherited in an X-linked recessive manner, but can be inherited autosomal recessively or dominantly. Therefore, the mode of inheritance does not always specify the type of DI.

### CONCLUSION

Central Diabetes Insipidus (CDI) is a complex disorder in which large volumes of dilute urine are excreted due to arginine-vasopressin deficiency and is caused by various disorders affecting the hypothalamic-posterior pituitary network. Differential diagnosis is difficult and requires a detailed history, physical examination, biochemical approach, imaging studies, and possibly histologic confirmation.

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### CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

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**Corresponding author** Morze Karolina, Department of Endocrinology, University of Antwerp, Belgium, E-mail: karolina@lak-takeuta.pl

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