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Vasudev G. Magaji MD, MS

Lehigh Valley Health Network, vasudev_g.magaji@lvhn.org

Gretchen Perilli MD

Lehigh Valley Health Network, gretchen_a.perilli@lvhn.org

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Diabetes Insipidus in Patient With Bodydysmorphic and Eating Disorder

Vasudev Magaji MD, and Gretchen Perilli

Division of Endocrinology, Department of Medicine, Lehigh Valley Health Network, Allentown, Pennsylvania

OBJECTIVE

Importance of diet and lifestyle assessment in diabetes insipidus.

CASE PRESENTATION

A 26 year old female had 6 months of polyuria and polydipsia. She had positive water deprivation test results for central diabetes insipidus during which she had 4lbs weight loss, serum sodium increased above 145meq, undetectable ADH and serum osmolality >300mosm/kg. DDAVP administration improved urine osm from 190 to 563 and serum sodium improved from 148 to 145. Since the urine osmolality increased by 100% she was diagnosed with central diabetes insipidus (table-1). She had a normal pituitary MRI without any abnormal enhancement, pituitary stalk displacement and sellar or para sellar lesion. Her menstrual cycles were normal and her serum pregnancy test was negative ruling out diabetes insipidus in pregnancy. Her BMI was 22.5 but complained of weight gain from lack of physical activity. Desmopressin 0.05mcg oral bid resolved her polyuria and polydipsia. Subsequently she acknowledged diagnosis of body dysmorphic disorder and reported a daily dietary intake of 1200 kcal and running at least 3 miles a day. She had poor body image perception, along with diet pattern of binge eating and fasting. Clinically she did not have deficiency of pituitary hormones given her normal menstrual cycles. Also it was less likely that she had either adrenal insufficiency or hypothyroidism since these conditions could impair renal free water clearance masking diabetes insipidus. Consistent with her clinical picture, her labs were normal (Table-1). The patient continues to seek psychiatric counseling and is being treated with desmopressin for the management of diabetes insipidus.

TABLE 1.

Lab	Value
Luteinizing Hormone	11.2 mIU/mL(2.4-12.6)
Follicle Stimulating Hormone	16.6 mIU/mL(4.7-21.5)
Cortisol	10.9 mcg/dL (2.3-19.4)
TSH	2.090 uIU/mL (0.45-4.50)
Prolactin	10.7ng/mL(4.8-23.3)
Estradiol	43.1 pg/mL (12.5-166.0)
Thyroxine	7.2mcg/dL(4.5-12.0)
T3 uptake	29% (24-39)
Free Thyroxine Index	2.1 (1.2-4.9)

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DISCUSSION

Hypothalamus is essential for integration and coordination through neuronal and hormonal pathways needed for homeostatic regulation and autonomic function. Like other hormones such as oxytocin that influences maternal behavior and corticotroph-releasing hormone that modulates stress-behavior, antidiuretic hormone influences behaviour through its vasopressin receptor. Eating disorders like anorexia nervosa are associated with multiple endocrine changes¹. Anorexia patients most commonly have decreased gonadotropins causing hypogonadotropic hypogonadism resulting in amenorrhea and also have low bone density. These patients have low insulin-like growth factor 1 with high growth hormone levels, low triiodothyronine and high cortisol levels that could be reflective of normal adaptation by suboptimal hormone response to under nutrition²⁻³. While most of the hormonal evaluation is focused on abnormalities anterior pituitary, it is important to assess for symptoms of diabetes insipidus in patients with an eating disorder. Water metabolism abnormalities could result from abnormal anti diuretic hormone levels and suboptimal renal response to anti diuretic hormone (ADH)⁴⁻⁶. Latent DI could exist even before development of anorexia nervosa⁷. Though our patient had normal body mass index and normal menstruation, it appears DI might be an initial endocrine manifestation of her eating disorder. Her clinical course needs close monitoring from an endocrine standpoint. In case her eating disorder was to resolve then reversibility of water metabolism abnormality needs to be assessed.

CONCLUSION

During evaluation of patients with eating disorders, the focus is mostly on hypothalamic and pituitary dysfunctions attributable to hypothalamic anterior pituitary axis. This case underscores that derangement of hypothalamic-posterior pituitary axis can be a manifestation of eating disorders. Hence it is important to assess for eating disorders for patients with DI whose work up is unrevealing.

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