Diabetes Insipidus in Patient With Body Dysmorphic and Eating Disorder

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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 145 meq, 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 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 diabetes insipidus.

During evaluation of patients with eating disorders, the focus is mostly on hypothalamic and pituitary dysfunctions attributable to hypothalamic anterior pituitary axis. This abnormality needs to be assessed.

In case her eating disorder was to resolve then reversibility of water metabolism abnormalities could result from abnormal anti diuretic hormone levels and 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 nutrition. 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). Latent DI could exist even before development of anorexia nervosa. 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.

TABLE 1.

<table>
<thead>
<tr>
<th>Lab</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Luteinizing Hormone</td>
<td>11.2</td>
</tr>
<tr>
<td>Follicle Stimulating Hormone</td>
<td>16.6</td>
</tr>
<tr>
<td>Cortisol</td>
<td>10.9</td>
</tr>
<tr>
<td>TSH</td>
<td>2.090</td>
</tr>
<tr>
<td>Prolactin</td>
<td>10.7</td>
</tr>
<tr>
<td>Thyroxine</td>
<td>43.1</td>
</tr>
<tr>
<td>T3 uptake</td>
<td>7.2mcg</td>
</tr>
<tr>
<td>Free Thyroxine Index</td>
<td>2.1</td>
</tr>
</tbody>
</table>


REFERENCES:

DISCUSSION
Hypothalamic 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, anti diuretic hormone influences behaviour through its vasopressin receptor. Eating disorders like anorexia nervosa are associated with multiple endocrine changes. 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 nutrition. 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). Latent DI could exist even before development of anorexia nervosa. 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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