Case Report

Acute urinary retention and hyponatremia from central hypothyroidism

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Abstract

A mass arising from the pituitary gland commonly damages cells of the anterior pituitary gland and affects the secretion of gonadotropins and growth hormone. However, central hypothyroidism and secondary adrenal insufficiency from such damage is a rare phenomenon. Acute urinary retention as the main symptom of central hypothyroidism is also an unusual initial presentation. We report a male patient who comes with frequent urinary retention and hyponatremia at our hospital.

Introduction

Central hypothyroidism (CH) refers to thyroid hormone deficiency due to a disorder of the pituitary, hypothalamus, or hypothalamic-pituitary portal circulation. It is a rare cause of hypothyroidism, estimated to occur in 1:20,000 to 1:80,000 in the general population [1]. The causes of central hypothyroidism include pituitary or hypothalamic pathologies. The most frequent cause of central hypothyroidism is Pituitary macroadenomas [2]. The diagnosis of CH is suggested by the findings of low thyroid hormone concentrations, associated with inappropriately low/normal TSH levels. The clinical manifestations of Central Hypothyroidism are similar to those of primary hypothyroidism. Here we present a man who seeks medical attention for recurrent acute urinary retention and hyponatremia as a result of central hypothyroidism.

Case presentation

A 64- year old male hypertensive patient on nifedipine comes to the emergency room complaining of fatigue, persistent vomiting, loss of appetite, and epigastric discomfort accompanied by a failure to pass urine for a few days. He reported no headache, fever, seizure, or any form of abnormal body movement or body weakness. He had undergone supra-pubic prostatectomy years ago as he had bladder outlet obstruction secondary to benign prostatic hyperplasia.

At presentation, his vital signs were stable; his physical examinations were unremarkable except for lassitude. The blood workup showed a normal complete blood count and organ function tests. The serum electrolyte study was notable for hyponatremia.

The abdominal ultrasound revealed absent prostate otherwise normal findings. The hyponatremia was corrected, and the gastrointestinal symptoms were managed by antiemetic and proton pump inhibitor. Since the patient had marked improvement symptomatically, he was discharged from the hospital.

After three weeks of discharge from the hospital, he comes again with a complaint of acute urine retention and difficulty walking. He also complained of easy fatigability, constipation, and change in mentation for few days. Upon physical examination; His vital signs were stable. The neurologic evaluation showed a confused man who was not oriented to place and time. His muscle strength and tone of all his extremities were decreased. The other findings were unremarkable.

A blood sample for complete blood count, renal function test, and blood glucose level were taken and reported normal. The serum electrolyte, however, revealed hyponatremia. This time thyroid function and other serum hormone study done, and the results are shown in tables 1 and 2.

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He was admitted to the general medical ward for further workup; with a working diagnosis of quadriparesis, hyponatremia, and acute urinary retention. Later the Magnetic Resonance Imaging (MRI) revealed a Pituitary macroadenoma (Figure 1). The findings from Electromyogram (EMG) Test & Nerve Conduction Study (NCS) were suggestive of mild to moderate sensory-motor polyneuropathy.

After the serum hormone studies, we started the patient on thyroxin 50 μg per day and prednisolone 7.5 mg per day. Within a few days of commencing the treatment, serum sodium level corrected, the confusion, fatigability, and constipation abated. After weeks of initiation of therapy the patient regained his full motor abilities and bladder function.

**Discussion**

Hyponatremia is a common finding in a state of low thyroid hormones. However, acute bladder retention from peripheral neuropathies due to central hypothyroidism is an unusual initial clinical presentation. There are only a few case reports where bladder retention was a clinical presentation because of hypothyroidism. Urinary retention was found incidentally in a middle-aged woman who has all signs of hypothyroidism including myxedema, malaise a change in tone of voice [3].

There was also a report where a female patient had uremia from urinary retention and was found to have hypothyroidism [4]. In our case, we attribute the urinary retention of the patient to hypothyroidism related peripheral neuropathies as the patient responded well to thyroid hormonal replacement.

The most important mechanism of development of hyponatremia in a state of low thyroid hormone is the decreased capacity of free water excretion due to elevated antidiuretic hormone (ADH) levels [5,6].

In patients with myxedema, the accumulation of interstitial mucopolysaccharides results in fluid retention and decreased effective arterial blood volume leading to ADH-mediated water retention and reduced sodium levels [7,8].

Few hypothyroid patients have increased urine sodium concentration as a result of syndrome of Inappropriate ADH secretion and not to reduced cardiac output [9].

Secondary adrenal insufficiency is also related to hyponatremia through increased ADH secretion. In this particular patient, the low ACTH was suggestive of the presence of secondary adrenal insufficiency; and we believed some of the symptoms have abated by the introduction of prednisolone.

**Conclusion**

Urinary retention could be the presenting symptom of hypothyroidism, and frequent finding of hyponatremia from an unknown cause needs a warrant for thyroid hormone evaluation.

**Ethical issues**

Permission to report the case was given by the patient after discussing and consent was made.

**References**

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