

Hypothyroidism Induced Myxedema Madness and Hyponatremia: A Case Report

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ARTICLE INFO

Article history:

Received on: 09/01/2017

Accepted on: 26/02/2017

Available online: 30/04/2017

Key words:

Antipsychotics,
hypothyroidism,
hyponatremia, levothyroxine,
myxedema madness.

ABSTRACT

Hypothyroidism is a frequently encountered medical condition in general population. Several disorders can arise when the thyroid hormone production is either too high or not enough. We report here about a hypothyroid patient who was presented to a tertiary hospital with psychotic manifestations. A 55 year old male with family history of psychosis in first degree relative had for the past 2 months reduced sleep at night, reduced appetite and less interaction with family members and gradually developed trouble in speaking. Registration of memory was impaired, features of delusions of persecution and reference and third person auditory hallucinations were present. Patient was started on antipsychotics for one week but without any improvement. Laboratory investigations demonstrated hyponatraemia and an abnormal thyroid profile: thyroid stimulating hormone: 10.89 uIU/ml, free triiodo threonine (FT3): 1.96 pg/ml and a normal thyroid on ultrasound. Tab levothyroxine 125 mcg was added along with the antipsychotics. The patient showed marked improvement in symptoms and the antipsychotics were withdrawn over a week. Thereafter there were no signs or symptoms of delusions or hallucinations and the patient was discharged on tab levothyroxine alone. Thyroxine supplementation also resolved the hyponatremia in this patient.

INTRODUCTION

Hypothyroidism is a common yet frequently overlooked medical condition with an estimated prevalence of approximately 10% among adults (Heinrich and Graham, 2003). Deficiencies in thyroid hormone can lead to global developmental abnormalities and acute metabolic derangement. The clinical presentations of thyroid hormone deficiency are diverse and complicated with many physical and neuropsychiatric manifestations (Bauer *et al.*, 2008). Hypothyroidism is usually represented by a cluster of signs and symptoms which include cold intolerance, dry skin, fatigue, hair loss, menstrual irregularities, hoarse voice, facial puffiness, slow speech, bradycardia etc. In addition, psychiatric disorders including anxiety, hallucinations, depression and

delusions are also accompanied by hypothyroidism (Heinrich and Graham, 2003). Once hypothyroidism is identified, appropriate thyroid hormone supplementation can be initiated and the patient will respond with the resolution of symptoms. Myxedema psychosis, more popularly known as myxedema madness, was first described (Asher, 1949) by "Asher" in 1949. Since then many case reports have been published strengthening the relationship between myxedema and psychosis (Westphal, 1997; McGaffee *et al.*, 1981, Tachman and Guthrie, 1984). Myxedema madness ensues as a rare consequence of hypothyroidism or due to non compliance with thyroxine replacement after thyroidectomy. The neuropsychiatric symptoms, including cognitive dysfunction, mood alterations and psychotic symptoms are referred to as myxedema psychosis. However, the mechanism of psychiatric disturbance caused by altered thyroid hormone levels is not fully understood. In the brain, triiodothyronine, the active form of thyroxine, binds to nuclear receptors altering gene transcription and thyroid receptors in the limbic system are responsible for the psychiatric symptoms in hypothyroid patients (Heinrich and Graham, 2003).

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The myxedema patients may report psychiatric manifestations only as presenting complaints. The psychiatric symptoms vary and the patient is first diagnosed with primary psychiatry disturbance rather than hypothyroidism (Pomeranze and King, 1966). Although the temporal association between hypothyroidism and hyponatremia are well documented and many investigations on the potential mechanisms have been carried out, the cause of this association is not well understood (Kimura, 2000). Patients with hypothyroidism have a diminished ability to excrete free water (Schrier, 2006; Derubertis *et al.*, 1971, Hanna and Scanlon, 1997)

CASE REPORT

A 55 year old unmarried male, living alone, with family history of psychosis in first degree relative and past history of alcohol dependence (abstinent for 15 years) was brought to the psychiatry department of a tertiary care hospital by his brother as the patient was observed to be confining himself to his house complaining that his neighbours were plotting to harm him since 2 months. There were also associated complaints of decreased sleep. Patient had been started on tab procyclidine 2.5mg and tab clozapine 25mg from a local hospital but did not achieve symptom control and issues with neighbours had worsened with patient showing physically and verbally abusive behavior towards neighbours. Patient believed that his thought was known to his neighbours through the surveillance methods they used. On admission to the psychiatry ward, his vital signs were: temperature: 98.6° F, pulse: 62 beats/min and blood pressure: 138/84mm of Hg. On physical examination, slight puffiness of face was noted with dry skin. Systemic examination findings were unremarkable, and he had a normal-size thyroid gland. On mental status examination, he was conscious and oriented to time, place and person with poor eye contact and attitude was guarded. Attention, language and orientation were intact. Registration of memory was impaired.

Thought broadcasting, delusion of persecution and reference and third person auditory hallucinations were present. In view of the presence of first rank symptoms and family history of psychotic disorder a provisional diagnosis of paranoid schizophrenia was put and patient was started on antipsychotics.

The course in the hospital included tab trifluoperazine 5mg TID and tab pimozide 2mg HS for his psychotic symptoms. Tab trihexyphenidyl 2mg was added for prevention of extra pyramidal side effects and tab olanzapine 2.5mg TID for presumed depressive disorder.

Investigations

The investigations revealed a remarkable increase in thyroid stimulating hormone level of 10.89 IU/ml (reference range: 0.50-5.00 IU/ml) and thyroxine (T4) level of less than 1.0 mcg/dl (reference range: 4.5-10.9 mcg/dl). Other investigations like hematogram, lipid panel, and renal and liver function tests were unremarkable but serum sodium concentrations were remarkably low. Details of serum sodium concentrations are shown in Fig.1.

Treatment

In view of profound hyponatremia (Table 1), treatment was commenced with 3% saline as intravenous infusion at 100ml/hour (Fig. 1). On the 4th day of admission, tab levothyroxine 100mcg was started but not continued due to oversight. The patient's clinical course was remarkable for fluctuating levels of consciousness, development of slight memory loss and puffy face with irregular speech, characteristics of myxedema coma. Endocrine consultation was sought for his high TSH value (10.89 IU/ml) and tab levothyroxine 125mcg was initiated and intravenous sodium chloride was continued for hyponatremia (Fig. 1). Fluid restriction was advised.

Soon after the initiation of treatment the patient experienced hypothermia, alteration of consciousness and was shifted to the intensive care unit. Psychiatric medications were withheld to avoid drowsiness, but levothyroxine was continued. Patient developed cough and was investigated and empirical antibiotics (inj. clindamycin 600mg TID and inj. cefoperazone 1gm BD for 9 days) were started. By the 14th day of hospitalization, the patient's clinical condition improved and became stable with corrected sodium and thyroid levels. Endocrine consultation was done and dose of levothyroxine was reduced to 100mcg OD and advised daily dietary intake of 8 grams of sodium chloride orally with fluid restriction to 1.5 litres/day.

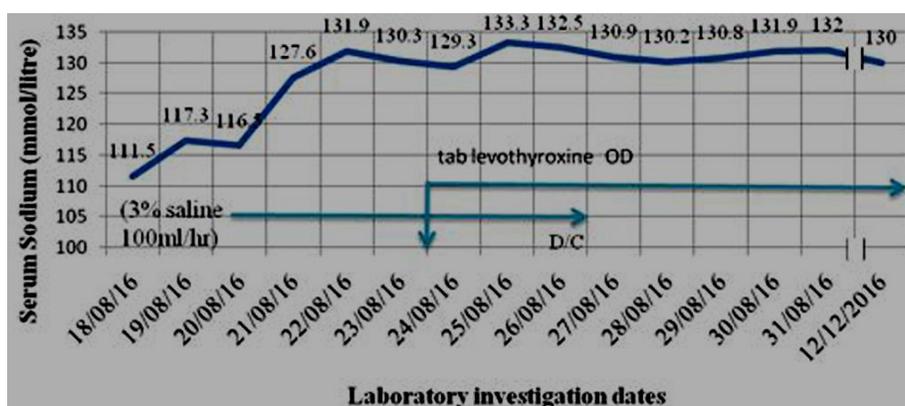


Fig. 1: Serum sodium levels at base line and at various time points after initiation of 3% saline and levothyroxine

Table 1: Thyroid panel and serum electrolyte levels of the patient.

Tests	18/8/16	20/8/16	23/8/16	25/8/16	27/8/16	29/8/16	30/8/16	12/12/16
Potassium (mmol/l)	4.4	-	3.9	3.8	3.9	3.9	4.1	4.3
Sodium (mmol/l)	111.7	116.6	130.3	133.3	130.9	130.8	132.9	130
TSH (μ IU/ml)	10.89	-	-	-	4.98	-	5.84	1.14
T4 (ng/dl)	0.55	-	-	-	0.75	-	0.92	10.4

Outcome and follow up

A repeat psychiatric evaluation was done to reconsider initiation of antipsychotics but patients psychotic symptoms had resolved without antipsychotics which revealed that the psychotic symptoms were due to altered thyroid panel. Patient was discharged with a final diagnosis of hypothyroidism and was directed for follow up in the endocrinology clinic. Two weeks later, on second follow up, patient was devoid of hallucinations and delusions and the sodium concentrations were also within the normal limits.

DISCUSSION

It is obvious to remember that many patients presenting with psychiatric symptoms may have alterations in endocrine function as thyroid dysfunction frequently ensues as a sequel to neuropsychiatric symptoms. Thyroid disease may be misdiagnosed as psychiatric disorders including depression and bipolar (Bauer *et al.*, 2008). So every patient presenting with psychotic signs or symptoms suggestive of hypothyroidism should have a complete thyroid work-up to avoid long term inadvertent administration of antipsychotics. This case illustrates the importance of routine thyroid panels and the need to rule out causes of psychiatric symptoms in patients presenting with psychiatric features. Additionally, psychosis due to hypothyroidism is reversible and can be easily treated (Hanna and Scanlon, 1997). Patients with neuropsychiatric symptoms and presenting with hyponatremia should have careful assessment of their thyroid status as it is assumed that over secretion of ADH and renal hypofunction in hypothyroidism may lead to hyponatremia (Kimura, 2000).

Financial support and sponsorship: NIL.

Conflict of Interests: There are no conflicts of interest.

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How to cite this article:

Gopinathan MV, James E. Hypothyroidism Induced Myxedema Madness and Hyponatremia: A Case Report. *J App Pharm Sci,* 2017; 7 (04): 206-208.