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Case Report

Hyperthyroidism and Psychosis

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Abstract

Thyroid disease presents with broad spectrum of symptoms. Here is case of thirty eight year old male presented to emergency room with affective psychosis developed over a week, after a thorough investigation he turned out to be a case of hyperthyroidism with psychosis. He was treated with anti thyroid medications, antipsychotic and mood stabilizer.

Introduction

Thyroid hormones are essential for mood regulation and cognition and there is a wide range of neuropsychiatric symptoms related with disease of thyroid [1]. Though hypothyroidism presents mainly with psychiatric symptoms psychosis has also been seen in 1% of cases of hyperthyroidism [2]. The underlying pathogenesis of neuropsychiatric symptoms in hyperthyroidism has been tried to be explained by a number of theories.

Density of β-receptor as well as their sensitivity to catecholamines in the brain is modulated by thyroid hormone and in hyperthyroidism β-receptor-mediated adrenergic activity is accentuated which is thought to be responsible for neuropsychiatric symptoms [3,4].

The first line of treatment for psychiatric symptoms in thyroid storm would be standard anti-thyroid therapy and β-blockers. If psychosis persists then antipsychotics are considered as an adjunctive. However, it has been reported in some studies that psychiatric symptoms resolve fully after treatment of hyperthyroidism [5,6].

Case Presentation

AB is a 38 years old Caucasian male who presented with acute onset mania including persecutory delusions of one week duration. AB's relatives reported talking for longer time than usual, screaming and being irritable. AB began to beat passersby as well as his family members saying that they belong to group who were conspiring against him. Since last 4 days prior to admission he had sense of increased energy. He would say that energy was given by goddess to fight back with the people who were trying to interrupt his mission. On asking further he would not elaborate more but seemed happy when he was expressing it to family member. Since 4 days he had decrease in need of sleep. Initially he used to sleep from 10 pm to 6 am; later though he slept at 10 pm, he would awake at 2 am in morning and gradually his sleep duration decreased to only 1 h to 2 h. During the sleepless period he would continue talking about people and injustice done to him. He would move around room. There was no burning sensation of eyes, fatigability, and daytime sleepiness the next day. He would not bother to eat. He would eat only when provided. Thought he took 2 major and 1 minor meal as he used to take previously but in a much reduced amount. Since last 4 days, he did not take care about changing clothes and bathing.

One week later he was brought to hospital. In the emergency room he was agitated with ongoing persecutory delusions. Initial assessment noted persecutory delusion, ideas, racing thoughts, rapid and pressured speech, and distractibility. At our centre temperature was documented to 1000 F, random blood sugar was 9.9 mmol/l, total and direct bilirubin was 35 u/l and 10u/l respectively; elevated SGPT (63U/L) and SGOT (84U/L) and sinus tachycardia on ECG. X-ray left foot was done for pain and swelling around fifth toe. X-ray showed undisplaced fracture of left phalynx (4^{th} and 5^{th}) and below knee posterior slab was applied.

Patient was admitted in ICU and shifted to nephromedicine ward after stabilization of vitals. Patient was prescribed risperidone 2 mg which was gradually built to 4 mg and sodium valproate 600 mg was added on fifth day after sustained irritability and clonazepam 0.5 mg bd which was gradually tapered after sustained improvement in sleep. Simultaneously Carbimazole 30 mg and Propanolol 40 mg was added from endocrine medicine for hyperthyroidism (fT3=29.9 pmol/L; fT4=70.6 pmol/L; TSH=<0.015 microIU/L). During his ward stay there was no complain regarding abnormal behavior. Patient gradually improved and become nearly premorbid. Patient was discharged on 21^{st} day and asked to follow up after 6 weeks with complete blood count, thyroid function and liver function test in endocrine medicine, psychiatry and in orthopedic OPD for slab removal.

AB denied previous diagnosis of hypothyroidism or ever having thyroid hormone levels checked. Physical symptoms of hyperthyroidism were not apparent and he denied physical symptoms in the past. There was no history of substance use.

Discussion

Hyperthyroidism is characterized by raised serum concentrations

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of Thyroxine (T4), Triiodothyronine (T3) and low serum Thyroid-Stimulating Hormone (TSH) concentrations.

Thyroid function in some persons plays important role in progression of affective disorders. In hyperthyroidism crisis of psychosis is uncommon but psychiatric manifestations are commonly seen in hypothyroidism either as "myxedema madness" or associated with abrupt correction of high levels of thyroid hormones (fT4) [7].

In differential diagnosis of psychosis crisis, trauma, autoimmune diseases, drug abuse, iatrogenic causes, strokes, tumors, congenital disorders (velocardiofacial syndrome), metabolic disturbances, sepsis, neurological infections, Addison disease, hyperparathyroidism, temporal lobe epilepsy, NMDA autoantibodies-associated encephalopathy, and schizophrenia are considered [8,9]. So a proper evaluation by a psychiatrist and a neurologist with specific treatment for each disease is needed.

The underlying relationship between the psychotic disorder of this patient and hyperthyroidism is taken into consideration as there is absence of previous psychiatric history, family history, precipitating psychological factors. Thus, the longitudinal courses of the illness will clarify it.

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