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MYXEDEMA MADNESS : A CASE REPORT ON HASHIMOTO THYROIDITIS

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ABSTRACT A 54 year old female patient who presented with general weakness, excessive perception of cold , insomnia , difficulty in word findings and periods of episodic confusions which was diagnosed as Hashimoto thyroiditis based upon biochemical reports (T3 76.0 ng/dl , T4 2.48 ug/dl, TSH 89.6 uIU/ml) and ultrasonographic findings. Additionally on USG neck there is multiple small hypoechoic foci associated with multiple thin echogenic strips are seen in involving both lobes of thyroid and the features suggestive of multiple adenomatous nodules with features of chronic thyroiditis. This study highlights the clinical presentation along with focus on psychiatric manifestation of Hashimoto thyroiditis as well as importance of thyroid evaluation in any psychiatric case to rule out any underlying hormonal deficiency

KEYWORDS : Hashimoto Thyroiditis , Hypothyroidism , Autoimmune Thyroiditis , Thyroid function tests , Psychiatric manifestations

INTRODUCTION :

The term "myxedema madness" traditionally refers to severe psychiatric symptoms associated with advanced hypothyroidism, or myxedema. This condition is characterized by an underactive thyroid gland, which leads to a deficiency in thyroid hormones. In its most severe form, hypothyroidism can cause a range of mental and neurological symptoms, Autoimmune thyroid inflammation and iodine deficiency are the most common causes of the disease. Hashimoto thyroiditis (HT), is now the most common autoimmune disease, that was described over a century ago . It is also known as lymphoid goiter affecting predominantly women population . In addition to the classic form there are several other clinico-pathologic entities are now included under the term HT: fibrous variant, IgG4-related variant, juvenile form, Hashitoxicosis, and painless thyroiditis (sporadic or post-partum) (1)

The prevalence of Hashimoto's thyroiditis was 7.5 (95%CI 5.7–9.6%), while in the low middle-income group the prevalence was 11.4 (95%CI 2.5–25.2%).(2)

The presentation may also be subclinical. Early symptoms may include fatigue, dryness in the skin, and weight gain ,decrease in bowel movements . More advanced and progressed symptoms may include decreased in sweating, intolerance to cold, deafness, peripheral neuropathic symptoms, depression, dementia, memory loss, muscle cramps, joint pain, hair loss, apnea, menorrhagia, and pressure symptoms in the neck from goiter enlargement such as voice hoarseness(3)

Currently there are three stages that are accepted regarding mechanism of pathogenesis of HT . In the early phase, antigen presenting cells (APC), mainly dendritic cells and macrophages, infiltrate the thyroid gland The infiltration may be induced by an environmental factor which causes cell damage and the exposure of thyrocytes specific proteins. Thyroid APC migrate to the lymph node where interactions occur between the APC cells, activated T cells, and B cells, leading to the induction of a variety of autoantibodies against thyroid-specific antigens.

In the final stage a massive destruction of the thyrocytes caused by autoreactive" T cells, B cells, and antibodies . It is a cell-mediated immune mechanisms, where there is production of antibodies against a variety of thyroid-specific antigens, such as thyroglobulin (TG) and peroxidase (TPO), but also the TSH receptor, sodium/iodine (NIS) symporter,19 and pendrin has been recently reported.(4)

Diagnosis of Hashimoto's thyroiditis can be done by various methods measuring blood levels of thyroid hormones (T3, T4) and thyroid-stimulating hormone (TSH). In Hashimoto's thyroiditis, TSH levels are usually elevated due to the thyroid gland's reduced ability to produce hormones. Specifically, tests for thyroid peroxidase antibodies (TPO antibodies) and thyroglobulin antibodies (TG antibodies) are done. Elevated levels of these antibodies indicate autoimmune thyroiditis, confirming the diagnosis of Hashimoto's. An ultrasound of the thyroid gland may be performed to assess its size, texture, and to detect any nodules or abnormalities. In some cases, particularly if nodules are found during ultrasound, a biopsy may be recommended to rule out thyroid cancer. This involves using a thin needle to extract a small tissue sample from the thyroid for examination under a microscope.

Clinical Presentation

A Female of 54 year age from Bengali tribe presented at the General medicine opd of a District hospital , Tripura .She belongs from low middle income group . Her Past Medical history was unremarkable with no history of any autoimmune disease .No such disease is present at her family.

The patient presented with general weakness with excessive perception of cold , insomnia ,decrease appetite ,vertigo ,palpitation, fatigue, anxiety , mental slowing along with persistent feelings of sadness or hopelessness. There is also associated of difficulty in word findings and periods of episodic confusions.

A through general physical Examination was performed and vitals were recorded. Patient was normosthenic built with body weight of 54 kg and BP recorded was 146/88 mmhg ,pulse 90 bpm. A diffuse enlarged thyroid gland of moderate size having firm consistency and freely movable measuring up to 6 x 3 cm. No associated regional lymphadenopathy. There is no

such clinical findings suggestive of any adjacent structure compression.

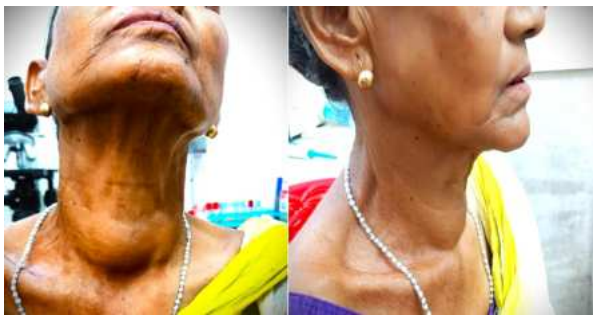

Figure : Showing diffuse enlarged thyroid gland in anterior and lateral view.

On Biochemical analysis of the blood sample taken from the patient after 8 hour of over night fasting shows following results:

TSH 89.6 uIU/mL , FT4 0.41 ng/dl, Total T4 2.48 ug/dl, Total T3 76.0 ng/dl , Anti TPO Antibody 998.0 IU/ml. Serum CRP test shows positive result. ESR 1st hour reading is 59 mm/hour. Hemoglobin is 8.3 gm/dl. Cholesterol- 280.0mg/dl Triglyceride- 180.0 mg/dl HDL – 59.0 mg/dl LDL – 185.0 mg/dl VLDL – 36.0 mg/dl. USG of Neck measures right lobe of 17.41 ml ,left lobe 10.53ml, isthmus 0.21cm .Both lobe of thyroid are enlarged in size .Echotexture is coarse and heterogenous in nature .Multiple small hypoechoic foci associated with multiple thin echogenic stripes are seen involving both lobes of thyroids giving a pseudo nodular appearance . Multiple well defined ,round to oval shaped ,hyper echoic lesions with peripheral vascularity are seen involving both lobes of thyroid ,largest measuring approx. 0.92 cm × 0.69 cm in size in right lobe of thyroid ,0.87 cm × 0.59 cm in size in left lobe of thyroid .No evidence of abnormal lymph node enlargement or abnormal mass lesion in seen at all levels . so, all the ultra sonographic feature suggestive of Multiple Adenometous nodules involving both lobes of thyroid (TIRADS II) with features of chronic thyroiditis .

TREATMENT Initially on first visit betahistine 16 mg thrice a day is started for the control of vertigo. In view of palpitation and increase heart rate with high blood pressure , Anti Hypertensive has been started , for hyperlipidemia statin Atorvastatin 40mg once daily at bed time started and the patient was hospitalized to stabilize the patient in a controlled environment . Tablet risperidone 2mg started after consulting with psychiatrist to control psychomotor symptoms . After getting the biochemical test of the patient ,Tablet levothyroxine 50 microgram once daily before breakfast has been started. Other symptoms were taken care of symptomatically . To eliminate negative nitrogen balance crystalline amino acid infusion is given. Risperidone was then withdrawn over a week with the recovery of her psychiatric abnormalities and then she was continued with tab thyroxin alone. She was discharged after about 14 days of hospitalization with the absence of any psychiatric symptoms.

DISCUSSION :

Not everyone with severe hypothyroidism will develop myxedema madness. The relationship between myxedema (severe hypothyroidism) and psychosis was strengthened by works of "Asher" who coined the term "myxedema madness,"[5] in the latter half of 20th century. Since then, numerous case reports have been published strengthening the existence of this entity.[6-8]

It typically occurs in cases where the thyroid hormone levels are extremely low and the condition has been untreated or undertreated for an extended period. Thyroid hormones are crucial for the normal functioning of the central nervous system (CNS), including neurotransmitter synthesis, metabolism, and receptor sensitivity. Thyroid hormones modulate the synthesis, release, and degradation of neurotransmitters such as serotonin, dopamine, and norepinephrine. Reduced thyroid hormone levels can lead to altered neurotransmitter levels and function, contributing to psychiatric symptoms such as mood disturbances, psychosis, and cognitive impairment.

In addition to the direct effects of thyroid hormone deficiency on neurotransmitter systems, myxedema madness may involve structural changes in brain tissue due to the accumulation of mucopolysaccharides.

CONCLUSION :

Understanding the multifactorial pathophysiology of myxedema madness is critical for its diagnosis and management. Treatment strategies should aim not only to replenish thyroid hormone levels but also to manage neurological and psychiatric symptoms effectively. This typically involves prompt initiation of thyroid hormone replacement therapy, often supplemented with supportive care and, in severe cases, antipsychotic medications to manage acute psychiatric symptoms. In conclusion, every patient presenting with psychiatric features having signs or symptoms suggestive of hypothyroidism should have a complete thyroid work-up to minimize the unnecessary long-term administration of antipsychotics. This case report demonstrates the potential reversible nature of psychosis of myxedema madness.

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