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Case Report

A case of long-term hypothyroidism converted to hyperthyroidism secondary to an autoimmune trigger

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ABSTRACT

Primary hypothyroidism is a common endocrine condition that is encountered. Graves disease and Hashimoto's thyroiditis are the most common autoimmune conditions in which conversion of hyperthyroidism to hypothyroidism is seen. Chances of conversion of hypothyroidism to hyperthyroidism is extremely rare. This case highlights that there should be a high index of suspicion for a possible conversion of hypothyroidism to hyperthyroidism. The etiology being an autoimmune switch by an external stimulus in genetically susceptible individuals. Hereby, presenting a case of 53-years female, who is a known case of type II diabetes mellitus, chronic kidney disease, nephrotic syndrome diagnosed with minimal change disease who presented with a hormonal profile showing hyperthyroidism. She had a history of hypothyroidism in the past for 12-years and was treated with levothyroxine and was off treatment for the past 2 years. Further evaluation showed presence of primary Sjogren's syndrome which has let to this conversion.

Keywords: Sjogren's syndrome, Primary hyperthyroidism, Autoimmune switch, Schimmer's test

INTRODUCTION

Primary hypothyroidism is the most common endocrine abnormality that is anticipated in females commonly. The most common cause being autoimmune thyroiditis while Graves' disease is the most common cause of hyperthyroidism.[1] Transformation from hypothyroidism to hyperthyroidism is considered relatively rare but commonly seen. This can be due to poor compliance of anti-thyroid medications frequently. Sometimes, this can occur due to autoimmune trigger.[2] Possible conversion can be due to over replacement of levothyroxine in few cases. The exact underlying etiology is not clear as there can be an autoimmune switch in genetically susceptible individuals.[1] We hereby present a case of 53-year-old female, known case of hypothyroidism, chronic kidney disease, nephrotic syndrome who presented with a clinical and hormonal picture of hyperthyroidism.

CASE REPORT

We hereby present a case of 53 years old female who presented to the outpatient department with complaints of joint pain, predominantly involving the small joints of both hands since one month. She also complained of generalized myalgia and fatigue for last three months with numbness and pricking sensation over both upper and lower limbs. She was diagnosed with hypothyroidism 12 years back and was on levothyroxine supplementation until 2019 after which she stopped taking medications. Patient was in euthyroid state in 2020 on routine evaluation. Patient had a past history of chronic kidney disease (stable) for past 10years. Renal biopsy was done eight years back and was diagnosed to have minimal change disease. Patient was diagnosed with systemic hypertension five years back and was in a normo-tensive state at the time of presentation.

Patient on arrival was conscious and well oriented to time, place and person. Vitals were found to be normal. Local examination of neck was done which showed diffuse enlargement of both lobes of thyroid gland. Central nervous system examination was unremarkable. Sensory system examination was unremarkable. Complete blood picture was normal. Thyroid profile showed a biochemical picture of hyperthyroidism with mildly elevated renal parameters. In view of unexplained peripheral neuropathy, serum vitamin B12 levels were done and was found to be within normal limits. RA factor and anti-CCP were negative. Ultrasound neck showed bilaterally enlarged glands with uniform surface. Ultrasound abdomen showed bilaterally shrunken kidneys with lost cortico-medullary differentiation. Nerve conduction study was done and was found to be normal. Thyroid stimulating immunoglobulin was found to be positive. ANA profile was sent to look for any autoimmune trigger for the conversation. ANA was 3+ positive with speckled pattern and was positive for anti-SSA. Schimmers test revealed dry eyes on both sides. Tectnitium^{99} radionucleotide scan showed mildly enlarged glands with increased uptake diffusely suggestive of autoimmune thyroiditis.

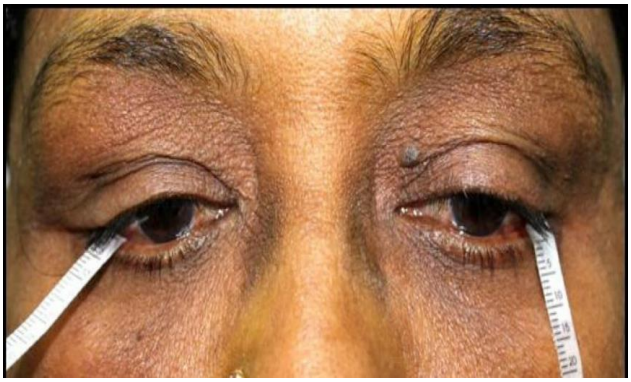

Figure 1: Schimmer's test of both eyes revealing dry eye.

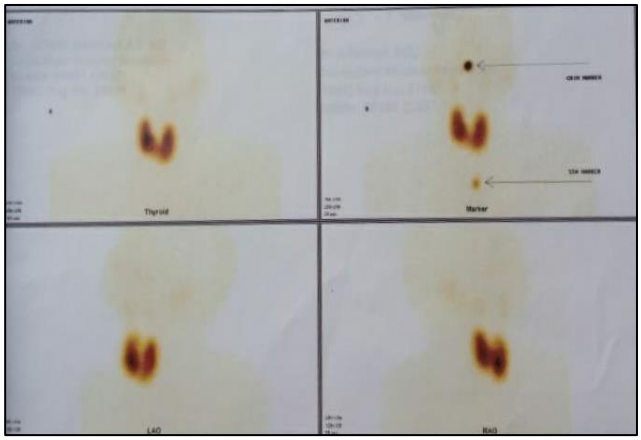

Figure 2: Technetium^{99} radionucleotide scan of thyroid.

Suggests diffuse homogenous uptake by both lobes with no evidence of toxic nodule or thyroiditis.

DISCUSSION

Conversion of hypothyroidism to hyperthyroidism is a well-known yet rare scenario in practice. Our patient presented with a clinical picture of hyperthyroidism with a long past history of hypothyroidism. Patient was off levothyroxine supplementation for almost 2 years and was in euthyroid state in between. In the setting of graves' disease, thyrotropin receptor antibodies are done. Few studies describe that the alterations in thyroid state were related to the balance in the activity of stimulating and blocking antibodies.[4] Several mechanisms have been postulated for the conversion including several genetic, environmental and others. According to several studies, presence of auto-antibodies such as thyroid stimulating antibodies, thyroid stimulation blocking antibodies may also lead to this conversion.[5]

Primary Sjogren's syndrome is ten times more common in patients with autoimmune thyroid disease. Autoimmune thyroiditis is frequently associated with primary Sjogren's syndrome. The most common clinical picture in patients with primary Sjogren's disease is hypothyroidism.[6] This is explained by the fact that salivary glands and thyroid glands share similar antigenic substances and are driven by similar genetic and environmental risk factors.[7] A wide range of central as well as peripheral neurological manifestations have said to been associated with primary Sjogren's syndrome, while the most common is pure sensory neuropathy.[8] Our patient was started on carbimazole and oral prednisolone, hydroxychloroquine, methotrexate and folic acid supplementation. Patient improved symptomatically and started showing good response at end of 6th month follow up.

CONCLUSION

Suspicion towards every possible conversion of hypothyroidism to hyperthyroidism should be kept in mind. Screening for autoimmune triggers is crucial in such scenarios. Several genetic and immuno-histopathological features such as peri-epithelial lymphocytic infiltration, oligoclonal B cell expansion and presence of human leukocyte antigen that play a major role in causing autoimmune tissue damage thereby leads to the conversion of biochemical profile. Autoimmune diseases like Sjogren are said to have a constellation of symptoms. Development of thyroid dysfunction in Sjogren is increasingly seen in female population and hence needs to be carefully evaluated.

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