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Case Report

Unsuspected Celiac Disease Severely Affects Levothyroxine Therapy in Hashimoto's Thyroiditis: a Case Report

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Abstract

Celiac disease (CD) is relatively common in western populations with estimated prevalence of approximately 1%. With the recent availability of sensitive and specific serological testing, many patients who were either asymptomatic or had subtle symptoms had shown CD. Over the past three decades, the pattern of CD presentation has altered. Many CD cases are now detected in adulthood during investigation of problems as diverse as anemia, osteoporosis, autoimmune disorders, unexplained neurological syndromes and infertility. Hashimoto's thyroiditis is a common thyroid disorder where hypothyroidism is treated by levothyroxine therapy. In our case report, we present how levothyroxine absorption was severely affected by celiac disease in a patient with Hashimoto's thyroiditis.

Keywords: Hashimoto's thyroiditis; Hypothyroidism; Celiac disease; Autoimmunity; Levothyroxine therapy

Introduction

Among autoimmune disorders, celiac disease (CD) has been increasingly reported in patients with autoimmune thyroid disease, type 1 diabetes mellitus, autoimmune liver diseases, and inflammatory bowel disease [1]. It has 1% to 19% prevalence in patients with type 1 diabetes mellitus, 2% to 5% in autoimmune thyroid disorders and 3% to 7% in primary biliary cirrhosis [2,3]. Though the pathogenesis of co-existent autoimmune thyroid disease and CD is unknown, they shared similar human leukocyte antigen (HLA) haplotypes and have been associated with the gene encoding cytotoxic T-lymphocyte-associated antigen-4[4-6].

Screening high risk patients for CD, such as those with autoimmune diseases, always has been a reasonable strategy. Gluten-free diet is known to reverse the complications of atypical CD like increased need for T4 and hence provide potential benefits to general health and perhaps increase life expectancy [7]. It also improves glycemic control in patients with type 1 diabetes mellitus and enhances the absorption of medications for associated hypothyroidism and osteoporosis.

Case Presentation

A 23-year-old female, presented with complaints of unintentional weight loss (6 kg in 3 months), hair loss (diffuse), and poor appetite past 6 months. There was no history of fever, cough, vomiting, loose stools, or pain in abdomen.

Investigation

On examination, she was a thin-built and a pale person with no edema, clubbing, and lymphadenopathy. Her thyroid examination was unremarkable. Other systemic examination was also within normal limits.

In August 2010, her laboratory reports showed Hb as 5.4mg/dl, MCV as 71.12 fl, TSH as 80µIU/l (0.34-5.6), FT4 as 0.12 (0.58-1.64), and FT3 as 0.23 (2.5-3.9). The anti-TPO antibody was high and iron profile revealed iron-deficiency anemia.

Treatment

In addition to 25 µg thyroxin, oral iron replacement therapy was instituted which subsequently improved her anemia. However, her requirement of thyroxin kept on increasing. Her TSH levels were never within the normal range despite increasing the thyroxin dose from 25 µg to 125µg. Even after consumption of 150 µg of thyroxin, her TSH levels remained mainly in the range of 300-400µIU/l

Table 1: Patient's Relationship between TSH levels, Thyroxine doses and Hemoglobin.

Date	TSH (µIU/ml)	Thyroxine Doses *(µg)*	*Hb* (g/dl)
Before Treatment at Max Hospital, Saket			
31-Aug-2010	80	0	5.4
16-Sep-2010	100	25	7.4
20-Oct-2010	175	50	11.2
12-Nov-2010	286	75	9.3
16-Dec-2010	354	100	-
After Treatment at Max Hospital, Saket			
3-Jan- 2011	486	125	9.9
5-Jan-2011	520	150	-
12-Mar-2011	520.3	250, gluten-free diet	9.7
9-Apr-2011	0.47	200	10.2
2-May-2011	35.32	150	11.6
28-May-2011	16.79	100	12.2
30-Aug-2011	0.04	50	13.4
25-Oct-2011	5.4	25	14.0

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(Table 1 and Figure 1). The patient was more concerned about her persistent elevated TSH levels. Hence, she was referred to Max Super Specialty Hospital, Saket. At the time (03-Jan-2011) of her visit to the hospital, her TSH level was 486μIU/l. She was highly compliant to her medications and used to take thyroxin empty stomach at 6 am in the morning. She was found positive for IgA anti-tissue transglutaminase (anti-tTG) antibodies. Upper GI endoscopy and duodenal biopsy revealed cryptic hyperplasia and villous atrophy, suggestive of celiac disease. Hence, she was put on gluten-free diet on 12-Mar-2011.

Outcome and Follow-Up

After a month (9-April-2011), she showed remarkable improvement. Her Hb improved to 10.2 mg/dl; TSH lowered down to 0.47μIU/l; gained 3 kg weight and felt better. Her levothyroxine dose was further reduced from 250 to 200μg. On her last follow-up on 25 October 2011, the Hb reached to 14 g/dl; TSH was stable at 5.4μIU/ml and the patient continued with 25 μg of levothyroxine.

Discussion

Celiac disease is a permanent intolerance of dietary gluten leading to mucosal damage in the proximal small bowel in genetically susceptible individuals, characterized by inflammation, crypt hyperplasia and villous atrophy which regress on withdrawal of gluten from the diet [8,9]. Recent population screening studies have shown approximate 1% prevalence of CD in Western countries [2]. Which may be due to under-diagnosis of the clinical condition or previously perceived rarity [9,10]. It should be considered in many clinical settings and must be detected earlier in order to prevent complications in later life. In atypical CD, there is an increased requirement for T4 due to its malabsorption which may provide the opportunity to detect atypical CD that was overlooked until the patients were put under T_4 therapy [3]. In a recent systematic review, Virilli and co-workers reported atypical CD as the pathogenetic factor underlying the increased demand of thyroxin and the effect was reversed by gluten-free diet [3,7].

In a 6-year prospective study, Corrao *et al.* followed 1,072 consecutive CD patients and found a standard mortality ratio of 0.5 in patients who complied with a strict gluten-free diet compared to a standard mortality ratio of 6.0 in the poor compliers [11]. Using the general practice research database, West *et al.* concluded that the patients with CD had the modest increase in overall risks of malignancy and mortality during their subsequent follow-ups [12].

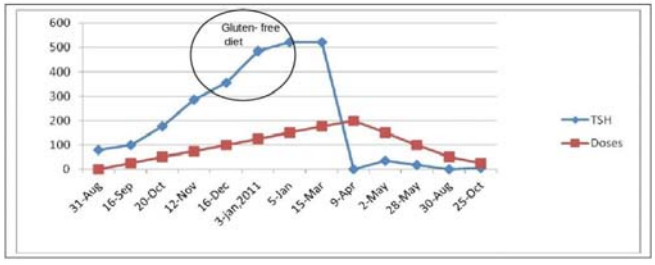

Figure 1: Showing changes in TSH and doses of thyroxine. Note the dip in the graph after initiation of gluten-free diet and also stabilixation of TSH on low doses of thyroxine once patient was maintained on gluten-free diet.

Learning Points

There is ample evidence of a strong association between CD and Hashimoto's thyroiditis. Some of these conditions share HLA haplotypes and non-HLA alleles, e.g., CTLA-4, which may underlie their pathogenesis.

- Patients with autoimmune thyroiditis should be routinely screened for CD in order to prevent symptoms and further complications (malabsorption, infertility, and osteoporosis) associated with the disease.

- Gluten-free diet plays a crucial role in regression of CD which improves the absorption of levothyroxine in Hashimoto thyroiditis and also improves the clinical condition of the patient in terms of thyroxin dose reduction and elevation of Hb levels.

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