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AN AUTOIMMUNE DISEASE OF THYROID HORMONE DYSFUNCTION: A CASE REPORT

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ABSTRACT

Iodine supplementation is associated with a greater incidence of autoimmune thyroid diseases. Iodine is an essential raw material for thyroid hormone synthesis. Adequate intakes of both iodine and selenium are required for optimal thyroid function. Iodine is an essential component of the thyroid hormones thyroxine (T4) and tri-iodothyronine (T3), and deficiency will impair synthesis of these hormones. Selenium is essential for the biosynthesis and function of the iodothyronine deiodinases that control the conversion of T4 to T3. A 45-years-old woman presented to the outpatient clinic with complaints of generalized swelling in the body, fatigue, and weakness with the irregular menstrual cycle and heavy flow of menstrual bleeding. At our first observation, with clinical signs of hypothyroidism: the regular pulse of 70 bpm and weight. Physical examination confirmed the blood pressure was 90/70mm of Hg. The patient was diagnosed with overt signs of hypothyroid with TPO antibodies quite higher with the normal range. FT4 and TSH also showed the hypofunction of the thyroid gland. The patient was already on medications (high dose of thyroxin 1000µg). The planned treatment was suggested and After one-month thyroid profile was done. After the therapy investigators observed significant changes in thyroid hormone function after the administration of 350 mcg iodine and elemental selenium 275mcg to a patient with underlying autoimmune thyroiditis. Thus, iodine and selenium supplementation reduce the dose of thyroxine to 350mcg.

CASE REPORT

Introduction

It has been suggested that iodine supplementation is associated with a greater incidence of autoimmune thyroid diseases. Iodine is an essential raw material for thyroid hormone synthesis. The minimum daily iodine intake that will maintain normal thyroid function is 150 µg in adults.[1] Iodine deficiency can result in goiter and hypothyroidism.[2] On the other hand, excess iodine intake may also lead to thyroid dysfunction as iodine exerts some effects on the normal and the sick thyroid gland.[3]

Adequate intakes of both iodine and selenium are required for optimal thyroid function (1–3). Iodine is an essential component of the thyroid hormones thyroxine (T4) and tri-iodothyronine (T3), and deficiency will impair synthesis of these hormones. Selenium is essential for the biosynthesis and

function of the iodothyronine deiodinases that control the conversion of T4 to T3. In addition, selenium-dependent glutathione peroxidases (GPxs) are implicated in the protection against oxidative damage to the thyroid gland. [4] Selenium deficiencies will decrease the conversion of T4 to T3, and it increases oxidant stress on the thyroid gland as a result of reduced GPx activity[5]. Both selenium and iodine deficiencies may exacerbate the effects of individual deficiencies such that selenium deficiency, in the presence of severe iodine deficiency, with the associated hyperstimulation of the thyroid and increased H_2O_2 production, results in reduced GPx activity and subsequent reduction in the clearance of H_2O_2.[5]

Case presentation:

A 45-years-old woman presented to the outpatient clinic with complaints of generalized swelling in the body, fatigue, and weakness with the irregular menstrual cycle and heavy flow of menstrual bleeding. Her medical history and family history were unremarkable but the daughter was hypothyroid. Previous endocrine tests revealed thyroid hormones and thyroid-stimulating hormone (TSH) plasma levels above the higher side of the normal range. A detailed medical history revealed that the patient was under medication and undergoing treatment for the hypothyroidism from last 5 years.

Investigations

At our first observation, with clinical signs of hypothyroidism: the regular pulse of 70 bpm and weight. Physical examination confirmed the blood pressure was 90/70mm of Hg. The patient was advised to undergo assessment of hematological parameters and hormonal parameters. The hormonal profile was of overt hypothyroidism, antithyroid peroxidase (TPO antibodies) was found to be positive (Table 1).

Ultrasonography of neck revealed no demonstratable abnormality with no increased or decreased echogenicity of the thyroid gland. No lymphadenopathy and no vascular abnormality is seen.

Table 1: Hematological and Hormonal Parameters

S. No	Parameters	Values	Normal range
1.	TSH	8.7	0.25-5.0µlu/Ml
2.	Free T_4	10.7	4.5-12.0 µg/dL
3.	T_3	102	60-200 ng/dL
4.	TPO	320	<60 unit/mL
5.	Iparathormone (iPTH)	0.34	11.12-79.5
6.	Hb	6.5	12-15g/dL
7.	PCV	25.3	35-52
8.	RBC	3.6	3.5-4.5millions/cumm
9.	MCV	69	76-96
10.	MCH	17.7	27-32pg
11.	MCHC	25.7g/dl	31.5-34.5

12.	TLC	5300	4000-11000
13.	RBS	96mmol	
14.	CALCIUM	9.6	8.5-11
15.	ESR	60	15
16.	Prolactin	31.34	2.8-29.2 ng/Ml

Past medical history:
Patient detailed medical history was taken and assessed thyroid profile carefully.

Table 2: Detailed medical history of the patient

Year	**Free T4 (thyroxine) (µg/dL)**	**T3 (triiodothyronine) (ng/dL)**	**TSH (µlu/mL)**	**Hb (g/dl)**	**Treatment and response to the treatment**
Sep 2013	24.02	<10	150	6.5	Firstly, the patient reported to the outpatient department with complaints of heavy menstrual bleeding and clot formation with normal 5-6 days of menstrual bleeding Advised tab thyronorm 100mg daily for 1month Banocide forte, Betonin syrup Antiparasitic medication diethylcarmazine Capsule trenexa at the time of menstrual bleeding Improvement in hematological parameters but thyroid profile shows an increase in TSH
Dec 2013	0.6	<10	60	9.5	Tab thyronorm 250µg
Feb 2014	55.46	30	16.10	10.2	Tab thyroxine 250µg daily
Dec 2015	24.02	30	36	10	Patient-reported with severe headache weakness and weight loss Tab thyroxine 450 µg
2016	11.2	40	126.82	10.5	No improvement was seen with 450 µg Suspected of malabsorption so loading dose of 1000 µg of Eltroxin started daily as evidence suggested of

					higher dose increases absorption.
2017	0.44	35	30.0	10	tablet ferrifort tablet shelcal, tablet calcitriol Tab eltroxin 1000ug for 2 months

Diagnosis, investigations, and treatment:

The patient was diagnosed with overt signs of hypothyroid with TPO antibodies quite higher with the normal range. FT4 and TSH also showed the hypofunction of the thyroid gland. The patient was already on medications (high dose of thyroxin 1000µg)

Treatment was planned for me. tab thyroxine sodium 200 MCG empty stomach for one month, ii. tab Caleat TH OD for one month (calcium citrate+ 1250 mg elemental selenium 200mcg+iodine 325mcg) after lunch iii. Carvic capsule for 1 month (alpha lipoic acid 200mg+chromium picolinate 200mcg+elemental selenium 75mcg) iv. Injection saturate after lunch for 5 days

After one-month thyroid profile was done on Dec 2018.

Table 3: Thyroid Profile After Treatment

S. No	Parameters	values	Normal range
1.	TSH	2.6	0.25-5.0µlu/mL
2.	Free T_4	8.6	4.5-12.0 µg/dL
3.	T_3	77.2	60-200ng/dL

Reports have been suggested patient responded quite well to the therapy and turn to euthyroid again with the control of clinical signs and symptoms.

Discussion:

To summarize: we observed significant changes in thyroid hormone function after the administration of 350 mcg iodine and elemental selenium 275mcg to a patient with underlying autoimmune thyroiditis. Thus, iodine and selenium supplementation reduce the dose of thyroxine to 350mcg.

Zagrodski et al. (2000)[6] have suggested that, in subjects with both Se and I deficiency, lack of observable differences in TSH and free T4 could be due to the overlapping effects of two processes: (1) decreased T4 secretion caused by I deficiency reflected by lower plasma free T4 concentrations; (2) an increase in free T4 associated with reduced iodothyronine deiodinase activity caused by Se deficiency. On the other hand, there may be other factors that influence thyroid hormone levels, including adaptations that occur in the pathways involved in thyroid hormone synthesis that act to maintain normal thyroid status (Arthur et al. 1999).[1]

The role of SE as an integral part of the iodothyronine deiodinase enzymes links Se and Iodine in a potentially important interrelationship, in which the degree of adequacy of one trace element may influence the metabolism of another. The iodothyronine deiodinase enzymes convert the prohormone thyroxine (T4) to the active form triiodothyronine (T3) required for normal growth and development, and for energy production and O2 consumption in cells. Type 1 deiodinase, abundant in liver and kidney, is sensitive to Se deficiency, which decreases deiodinase activity and therefore T4 to T3 conversion, resulting in a decrease in the T3: T4 ratio. On the other hand, type 2 and type 3 deiodinases are less sensitive to Se deficiency, indicating their importance for the maintenance of normal thyroid hormone levels (Arthur 1999).[7]

In addition, Se as the antioxidant enzyme glutathione peroxidase (GPx) may protect the thyroid gland from oxidative damage due to any excess H2O2 produced during thyroid hormone synthesis.[8] Thus, Se deficiency may exacerbate some effects of I deficiency and may have a role in the etiology

of I-deficiency disorders (Arthur & Beckett, 1999).[2]

Evidence also suggested that Se supplementation results in an increase in the selenoproteins, GPx and selenoprotein P (Thomson et al. 1993; Duffield et al. 1999) suggests that Se intake is insufficient for maximal activity of these proteins.[8,9,10]

Although we have not measured Se concentrations significantly effects on thyroid function have been observed, the combination of low Se status and mild I deficiency may be significant. A number of human studies have shown alterations in the T3: T4 ratio associated with low Se and Iodine status (Kvı´cala et al. 1995; Olivieri et al. 1996; Ravaglia et al. 2000).[11,12.]

Therefore, it is important to determine whether there are any detrimental effects of the marginal status of Se and I in our population. The present paper examines the importance of interaction between Se and I have been investigated, including (a) the relationship between Se status and thyroid status in this case and (b) the effect of Se supplementation on measures of thyroid status: thyroid-stimulating hormone (TSH), Tg and the T3: T4 ratio. The large sample size needed to authenticate the present findings.

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