



## Decreased Level of Fasting Blood Sugar in a Thyrotoxic Patient after Treatment with Methimazole- A case report

### ARTICLE INFO

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### ABSTRACT

Thyrotoxicosis is the name given to the clinical manifestations of the excess of thyroid hormones in the bloodstream. Thyrotoxicosis may induce increased fasting blood sugar (FBS) in patients of all ages, and has been reported only in few cases. The previous studies have shown that the prevalence of glucose intolerance in hyperthyroidism is 44-65% and may reverse to normal values after achieving euthyroid status. We have discussed about a 34-year-old woman who presented with thyrotoxicosis with enhanced FBS. The patient had mild elevated FBS (110 mg/dl) when she had severe thyrotoxicosis. After treatment with methimazole blood glucose level decreased to 85 mg/dl and by her third visit glucose level decreased to 82 mg/dl. It could be concluded that mild hyperglycemia may co-exist with thyrotoxicosis and reduce to normal when the patient becomes euthyroid. Thus, early detection and treatment of thyroid hormones over secretion can resolve the complications of both thyrotoxicosis and associated hyperglycemia.

**KeyWords:** Thyrotoxicosis; Methimazole; Fasting Blood Sugar.

## Introduction

Thyroid hormones are the important regulators of blood glucose [1,2]. Therefore, in hyperthyroidism and thyrotoxicosis which are commonly associated with excessive quantities of endogenous or exogenous thyroid hormones, glucose metabolism is impaired. Although, the exact underlying mechanisms for this impairment are not fully elucidated, a number of mechanisms have been provided including elevation in  $\beta$  cells mass, increase in intestinal glucose absorption, decrease in peripheral insulin sensitivity, decrease in the response of beta cells to glucose by insulin, and increase in insulin clearance [1,3]. The previous studies have shown that the prevalence of glucose intolerance in hyperthyroidism is 44-65% and may reverse to normal values after achieving euthyroid status [4,5]. We report here a patient with thyrotoxicosis and mild enhanced FBS.

## Case Presentation

A 34 years old Iranian woman was presented in the clinic with symptoms and signs of thyrotoxicosis. It took about two months from the onset of the disease until the patient's visit. The patient's weight was 71 kg in the first visit, which reached 73.5 kg in the second visit and remained the same in the third visit. The patient's blood pressure was in the range of 120 at each visit, which was not considered due to the fact that there were no significant changes. She was requested to have CBC including RBC, platelets, hemoglobin, hematocrit; FBS, thyroxine (T4) and thyroid stimulating hormone (TSH) were also measured (Table 1, Report 1). Thirty mg/day methimazole was prescribed for her. The patient came back after using this drug for six weeks and all the factors were measured again (Table 1, Report 2). By evaluation of clinical and biochemical parameters, the dose of methimazole was decreased to 10 mg/day for four weeks when she had another blood test for biochemical parameters. The results are presented as report 3. At this time 5 mg/day methimazole was prescribed for her as a maintenance dose and she was requested to refer to the clinic after 2 months. Therefore, results of different reports showed severe thyrotoxicosis patient with high serum level of T4 (24.9  $\mu\text{g}/\text{dl}$ ), low serum level of TSH

(0.01 mIU/L) and normal serum level free T4 (2.2 ng/dl) and mild elevated FBS (110mg/dl) in the first visit had. After treatment with methimazole, blood glucose and T4 levels in this patient decreased to 85 mg/dl and 8.2  $\mu\text{g}/\text{dl}$ , respectively. Glucose and T4 levels were normal and they were reported 82 mg/dl and 7.5, respectively in the third visit. To some extent we can claim that severe thyrotoxicosis can increase FBS mildly and taking methimazole will restore it to a normal range. Furthermore, methimazol treatment doesn't changed free T4 and TSH serum levels.

**Table 1:** Hematological parameters and hormone concentration in the thyrotoxic patient before and after treatment with methimazole

Test	Repo rt 1	Repo rt 2	Report 3
WBC ( $10^3/\mu\text{l}$ )	4.4	5.5	6.6
RBC ( $10^6/\mu\text{l}$ )	4.9	4.6	4.4
Platelets ( $10^3/\mu\text{l}$ )	259	257	242
Hemoglo bin (g/dl)	13.6	12.8	13.2
Hematocr it (%)	40.6	37.7	40.1
FBS (mg/dl)	110	85	82
T4 ( $\mu\text{g}/\text{dl}$ )	24.9	8.2	7.5
TSH (mIU/L)	0.01	0.02	0.03
Free T4 (ng/dl)	2.2	1.2	0.9

Serum levels of hematological parameters and hormone concentration in thyrotoxic patient before methimazole treatment (Report 1), after using the 30 mg/day methimazole for six weeks (Report 2) and after using the 10 mg/day methimazole for 4 weeks (Report 3).

WBC, white blood cells; RBC, red blood cells; FBS, fasting blood sugar; T4, thyroxine; TSH, thyroid stimulating hormone; mIU/L, milli International Unit/Liter.

## Discussion

The diagnosis of thyrotoxicosis has been standardized based on symptomatic severity and the levels of thyroxine (T4) and triiodothyronine (T3) [6]. Increase in the serum level of T4 and

decrease in the TSH level presented in the results of this study confirmed thyrotoxicosis in a woman who referred to the clinic before usage of any antithyroid drugs. While mild thyrotoxicosis may not cause any symptoms, more severe thyrotoxicosis shows major clinical manifestations such as: weight loss, increased heart rate, arrhythmia, tremor and intolerance to heat [6]. These symptoms occur as response to the increased amount of thyroid hormones and increased sensitivity of adrenergic receptors to catecholamines in hypermetabolic states. Accordingly, signs and symptoms of thyrotoxicosis including hand tremor, weight loss, warmth and sweating, anxiety, tachycardia, hyperreflexia and minimally enlarged thyroid gland were diagnosed in our patient who referred to the clinic. Moreover, in this case, low TSH and high T4 levels were combined with a mild hyperglycemia, one of the metabolic effects of excess thyroid hormones. Measurement oral glucose tolerance test and analysis of glucose and insulin curves in thirty-eight patients with hyperthyroidism and twenty-six healthy volunteers determined the prevalence of glucose intolerance in hyperthyroid patients was higher than healthy volunteers [3].

In our patient, mild elevated blood glucose was corrected after treatment with methimazole. Methimazole, as an anti-thyroid drug, reduces the production of thyroid hormones by interfering with the tyrosine iodination, by inhibiting the iodotyrosyl residues from the coupling and interfering with the oxidation of the iodide ion and iodotyrosyl groups. It has been shown that thyrotoxicosis and diabetes mellitus mutually influence each other. Diabetic patients compared with the normal population have higher incidence of thyroid disorders [7,8]. On the other hand, in hyperthyroidism glycemic control is worsened and insulin requirements are increased. Also, there are increased intestinal glucose absorption and probably increased insulin resistance. Indeed, thyrotoxicosis may unmask latent diabetes [3,9, 10]. An in vitro study determined methimazole improved increased metabolism of glucose and glutamine caused by thyroid hormones in normal lymphocytes [11]. Tene (Year) reported in patients with Graves' disease for four months

methimazole treatment increased insulin sensitivity from 3.47 to 6.39 mg/kg/min [12]

The finding of this study same as some other pronounced investigations demonstrated the importance of thyroid hormones in regulation of blood glucose. Furthermore, in this study, it was recognized for the first time that taking different doses of methimazole in a severe thyrotoxicosis case will restore FBS to a normal range.

## Conclusion

Mild hyperglycemia may co-exist with thyrotoxicosis and return to normal when the patient becomes euthyroid. Thus, early detection and treatment of thyroid hormones oversecretion could resolve the complications of thyrotoxicosis as well as associated hyperglycemia.

## Conflict of interest

The authors report no conflict of interest.

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