Annals of Thyroid Research

Open Access

Austin Publishing Group

Case Report

Hyperthyroidism due to Gestational Trophoblastic Disease - A Case Report

Topf A[1]*, Motloch L[1], Kopp K[1], Moritz M[1], Hoppe UC[1] and Pleininger T[2]

[1]Department of Cardiology, Paracelsus Medical University of Salzburg, Austria

[2]Department of Gastroenterology, Paracelsus Medical University of Salzburg, Austria

***Corresponding author:** Albert Topf, Department of Cardiology, Clinic of Internal Medicine II, Paracelsus Medical University of Salzburg, Salzburg, Austria

Received: July 20, 2020; **Accepted:** August 10, 2020; **Published:** August 17, 2020

Abstract

A 53-year-old female patient presented to our department with a symptomatic hyperthyroidism. After an inconclusive routine examination of the thyroid gland, an abdominal ultrasound and Human Chorionic Gonadotropin testing (ß–HCG) were done confirming the suspicion of a Beta-HCG secreting trophoblastic tumor-induced hyperthyroidism. ß–HCG can mimic Thyroid-Stimulating Hormone (TSH), has a potency 4000 times less than TSH, and therefore can stimulate thyroid hormone production and secretion.

While most thyroid abnormalities may be attributed to thyroid gland itself, the following report shows the importance of a ß–HCG screening in a hyperthyroidism with an unclear genesis.

Introduction

Hyperthyroidism is a common clinical problem. Although most abnormalities are caused by the thyroid gland itself, like the Graves' disease or thyroid autonomic dysfunction, it is important to be aware of other causes, as demonstrated by this case of a trophoblastic tumor-induced hyperthyroidism [1]. HCG, produced by trophoblastic tumor, can mimic TSH and consequently stimulate the thyroid hormone production and secretion [2]. The potency of HCG for TSH receptors is 4000 times less than TSH and hence, extremely high levels of HCG become necessary for an effect on thyroid function [3].

Evaluation of thyroid metabolism helped not only to find the cause for hyperthyroidism, but more importantly to find an existing tumor in the patient.

We present a case of a 53-year-old woman who presented to our department due to trophoblastic tumor-induced hyperthyroidism.

Case Report

A 53-year-old woman presented to our department with progressive leg edema, exertional dyspnea and nocturnal emesis. Additionally, she reported hypermenorrhea for lasting 10 days. Until this time, the patient had never experienced any health problems. She gave normal birth to two children. The patient underwent a regular gynecological care.

In the laboratory examination, severe hyperthyroidism (TSH < 0,01 mU/L), anemia (hemoglobin 8,8 g/dl) and an elevated pro B-type Natriuretic Peptide (BNP) level (1778 pg/ml) were diagnosed.

Further examination of the thyroid gland showed an inconspicuous morphology by ultrasound testing and the antithyroid autoantibody results were negative. The genesis of hyperthyroidism remained unexplained. Cardiac evaluation showed high output heart failure due to the severe hyperthyroidism.

Subsequent evaluation of the genesis of hyperthyroidism by an abdominal ultrasound and a Beta–HCG testing confirmed the suspicion of secondary hyperthyroidism by a ß–HCG-producing tumor. ß–HCG levels over 2 Mio U/L stimulated the thyroid hormone production and secretion.

MRI and PET – CT supported the suspicion of a gestational trophoblastic disease. Hysterectomy with a following histological examination showed an invasive mole. After tumor resection, the thyroid metabolism started to normalize.

Discussion

Symptomatic thyrotoxicosis is mostly associated with problems in thyroid metabolism itself. Interestingly, in our case, a ß–HCG-induced hyperthyroidism manifested with problems leading to thyrotoxicosis. With the exception of the 10-day hyper menorrhagia, the patient had no gynecologically-dependent problems.

High HCG levels stimulate the thyroid gland with suppression of pituitary TSH release [4]. HCG is α glycoprotein composed of α and β subunits. The α subunit is almost identical to that found in TSH. The sub-unit consists of a 92-amino-acid chain containing two nitrogen-linked oligosaccharide side chains [5]. The potency of HCG for TSH receptors is 4000 times less than TSH. Consequently, extremely high levels of HCG are usually required for an effect on thyroid metabolism to be observed [6]. Serum concentrations above 200,000 U/L have been demonstrated to suppress TSH in 67% of cases, and levels above 400,000 U/L promote suppression in 100% of cases [7,8].

Subclinical hyperthyroidism with inconspicuous T3 and T4 levels is well-known in normal pregnancy, but clinical manifestations are uncommon. A freedom of symptoms following changes in thyroid metabolism is less often reported in ß-HCG secreting gestational neoplasia and other germ cell malignancies [9,10]. Trophoblastic HCG production is not inhibited by increased levels of thyroid hormones. Therefore, a negative feedback mechanism is not existing. The thyroid function normalizes as the serum HCG sinks [11].

HCG-producing germ cell tumors may exist in both genders. HCG screening may help to diagnose the genesis of an unclear hyperthyroidism as demonstrated in this case report. The significance of HCG screening in hyperthyroidic metabolism must be evaluated

Citation: Topf A, Motloch L, Kopp K, Moritz M, Hoppe UC and Pleininger T. Hyperthyroidism due to Gestational Trophoblastic Disease - A Case Report. Annals Thyroid Res. 2020; 6(1): 251-252.

in future studies.

Conclusion

The reported case should highlight the awareness of extrathyroidal reasons for hyperthyroidism. Additional ß–HCG testing should be considered in hyperthyroidism with unclear genesis.

References

1. De Leo S, Lee SY, Braverman LE. Hyperthyroidism. Lancet. 2016; 388: 906–918.
2. Raj R, Mae Uy E, Hager M, Asadipooya K. Delivery of Euthyroid Baby Following Hyperthyroidism in Twin Gestation With Coexisting Complete Hydatidiform Mole. Case Rep Endocrinol. 2019.
3. Kennedy RL, Darne J. The Role of hCG in Regulation of the Thyroid Gland in Normal and Abnormal Pregnancy. Obstet Gynecol. 1991; 78: 298-307.
4. Koulouri O, Halsall D, Chatterjee K, Gurnell M. Pitfalls in the measurement and interpretation of thyroid function tests. Best Pract Res Clin Endocrinol Metab. 2013; 27: 745–762.
5. Szkudlinski MW, Fremont V, Ronin C, Weintraub BD. Thyroid-Stimulating Hormone and Thyroid-Stimulating Hormone Receptor Structure-Function Relationships. Physiol Rev. 2002; 82: 473–502.
6. Yoshimura M, Hershman JM. Thyrotropic Action of Human Chorionic Gonadotropin. Thyroid. 1995; 5: 425-434.
7. Lockwood CM, Grenache DG, Gronowski AM. Serum Human Chorionic Gonadotropin Concentrations Greater than 400,000 IU/L Are Invariably Associated with Suppressed Serum Thyrotropin Concentrations. American Thyroid Association. 2009; 19: 863-868.
8. Blick C, Schreyer KE. Gestational Trophoblastic Disease-induced Thyroid Storm. Clin Pract Cases Emerg Med. 2019; 3: 409–412.
9. Soldin OP. Thyroid Function Testing in Pregnancy and Thyroid Disease: Trimester-specific Reference Intervals. Ther Drug Monit. 2006; 28: 8–11.
10. de Almeida CED, Curi EF, de Almeida CRD, Vieira DF. Thyrotoxic Crisis Associated with Gestational Trophoblastic Disease. Rev Bras Anestesiol. 2011; 61: 604-609.
11. Walkington L, J Webster J, Hancock BW, Everard J, Coleman RE. Hyperthyroidism and human chorionic gonadotrophin production in gestational trophoblastic disease. Br J Cancer. 2011; 104: 1665–1669.