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# Hyperthyroidism secondary to a hydatidiform mole

Barbara Grzechocinska<sup>1</sup>, Malgorzata Gajewska<sup>1</sup>, Maciej Kedzierski<sup>1</sup>, Sylwia Gajda<sup>2</sup>, Piotr Jedrzejak<sup>1</sup>, Miroslaw Wielgos<sup>1</sup>

<sup>1</sup>1st Department of Obstetrics and Gynecology, Medical University of Warsaw, Poland
<sup>2</sup>Endocrine Oncology and Nuclear Medicine Clinic, M. Skłodowska-Curie Memorial Institute
— Cancer Centre, Warsaw, Poland

#### **ABSTRACT**

The case presented in the article is that of a 47-year-old female patient with hyperthyroidism induced by a hydatidiform mole. Attention was drawn to the necessity of preparing the patient for a procedure with drugs that stabilize the hormonal activity of the thyroid. The removal of the hydatidiform mole resulted in gradual normalization of thyroid hormone levels.

The trophoblast has a hormonal activity, secrete hCG (human chorionic gonadotropin). The hCG partial structural homology causes affinity to the TSH (thyroid stimulating hormone) receptor. The higher the weight of the trophoblast, the higher the production and concentration of hCG in the blood.

Therefore, gestational trophoblastic disease may be accompanied by hyperthyroidism. The problem is frequently described, however, due to the risk of developing thyroid storm, it cannot be overlooked [1].

Key words: gestational trophoblastic disease; hydatidiform mole; hCG; hyperthyroidism

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## **CASE STUDY**

A 47-year-old patient was admitted to the hospital due to uterine bleeding. The patient's general condition was good. The last menstruation started 53 days earlier. The patient was in her eighth pregnancy (5 births, 2 miscarriages in the history).

On gynaecological examination: the uterine body was enlarged, reaching 6 cm above the symphysis pubis. On TV ultrasound: a heterogeneous  $78 \times 77 \times 91$  mm 'Swiss-cheese pattern' area in the uterine cavity. Both ovaries have normal morphology.

Laboratory results are shown in Table 1.

The patient did not report any symptoms of hyperthyroidism. On physical examination the thyroid was not enlarged, the ultrasound examination showed its normal size, slightly reduced echogenicity without increased vascularization.

Table 1. Hormonal examination and antibody results before and during treatment					
	13.03.19 (Before the procedure)	18.03.19	9.04.19	22.07.19	13.05.20
betahCG (mIU/mL)	414,937	26,096	919.8	2.34	< 1.0
TSH (mIU/L) [0.33-4.59] <sup>1</sup>	0.008	< 0.005	0.011	0.05	0.36
fT3 (pmol/L) [3.78-5.98] <sup>1</sup>	16.0	6.62	4.0	4.5	3.34
fT4 (pmol/L) [12.1–19.6] <sup>1</sup>	60.7	34.4	18.3	18.1	16.3
ATPO (U/mL) <sup>3</sup> [< 60] <sup>2</sup>	< 28				
ATG (U/mL) <sup>4</sup> [< 60] <sup>2</sup>	< 28				
TRAb (U/L) <sup>5</sup> [< 1.0] <sup>2</sup>	< 1.0				

<sup>1</sup>Reference values for the laboratory are given in parentheses; <sup>2</sup>ATPO – Thyroid Peroxydase Antibodies; <sup>3</sup>ATG – Thyreoglobulin Antibodies; <sup>4</sup>TRAb – TSH ReceptorAntibodies

## Corresponding author:

Malgorzata Gajewska

1<sup>st</sup> Department of Obstetrics and Gynecology, Medical University of Warsaw, Poland e-mail: ma.gajewska@gmail.com

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Due to the patient's age, no further procreation plans, suspicion of complete hydatidiform mole, a decision was to excise the uterus.

Due to abnormal hormone concentrations, the patient needed to be prepared for surgery. Achieving euthyroidism, usually requiring time, was not possible in this case. Thiamazole at a dose of 20 mg every eight hours was activated, before the surgery hydrocortisone 100 mg was administered. Injections were repeated every six hours on the day of the surgery and continued the first day after the surgery.

The surgical procedure has run without complications. Histopathological examination results complete hydatidiform mole. No clinical signs of hyperthyroidism were observed postoperatively. The patient was discharged home on day four after the surgery in good general condition.

The patient is currently under observation with no signs of hydatidiform mole or thyroid disease.

#### **DISCUSSION**

Hyperthyroidism in the course of gestational trophoblastic disease is diagnosed in 25–64% of patients [2]. Nevertheless, the asymptomatic course is found in 5–10% of patients the risk of the storm should always be considered in every patient with hyperthyroidism [3]. It was found that severe complications of hyperthyroidism occur in about 20% of patients [4]. The described case indicates a direct relationship between  $\beta$ -hCG concentration and thyroid dysfunction. A very slow decrease in TSH concentration, with fairly rapid normalization of fT3 and fT4 concentrations after surgery.

The treatment of gestational trophoblastic disease with hyperthyroidism requires appropriate preparation of the patient to achieve euthyroidism and should be implemented as soon as possible after the diagnosis. There are no detailed guidelines for such cases in the literature. We recommended thionamides that inhibit thyroid peroxidase and glucocorticoids that inhibit the conversion of thyroxine to triiodothyronine. In monitoring the course of treatment of gestational trophoblastic disease it is important to pay attention not only to markers of treatment efficacy but also to clinical and biochemical markers of thyrotoxicosis.

## **Conflict of interest**

The authors state that there are no conflicts of interest to disclose.

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