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## Initial paraneoplastic presentation of advanced choriocarcinoma

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## **CLINICAL VIGNETTE**

### **Initial paraneoplastic presentation of advanced choriocarcinoma**

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## INTRODUCTION

Gestational trophoblastic neoplasia (GTN) includes invasive mole, choriocarcinoma, epithelioid trophoblastic tumor and placental site trophoblastic tumor. It occurs after either molar pregnancies (50%), term pregnancies (25%) or remaining gestational events (25%) [1]. In case of this neoplasm, an efficient diagnosis is based on the beta-subunit of human chorionic gonadotropin ( $\beta$ -hCG) serum levels. Diagnostic watchfulness is crucial and reproductive history should be analyzed, while performing differential diagnosis of various symptoms, as paraneoplastic syndromes and the tendency to metastasize are among the features characteristic for choriocarcinoma. We aim to increase efficiency of choriocarcinoma diagnosis based on characteristic paraneoplastic symptomatology. We want to stress clinicians to carefully note symptoms, that arise from other organ systems, for example, hyperthyroidism, hemoptysis, gastrointestinal bleeding, or presence of theca lutein cysts, that form as a result of ovarian overstimulation from high  $\beta$ -hCG levels.

## CASE STUDY

A 45-year-old female patient was admitted to the Gynecological Oncology Department with a suspicion of metastatic lesions in both lungs and liver. Earlier this year a patient was hospitalized due to severe thyrotoxicosis. At the time of admission nausea, vomiting, diarrhea, pelvic pain, palpitations, and significant fatigue were reported. Patients vital signs were as follows, blood pressure 100/50 mmHg, heart rate 113/min, blood saturation SO<sub>2</sub> 97%, and the results of laboratory tests are presented in Table 1. Taking into consideration clinical presentation and tests results, choriocarcinoma was diagnosed. After improving patients' overall condition, the first course of chemotherapy, etoposide, methotrexate, actinomycin D, cyclophosphamide, vincristine (EMA-CO) chemotherapy was administered. On the 10th day massive intestinal hemorrhage occurred at night. Despite conducting lifesaving procedures, the patient died. Essential information in medical interview: hypothyroidism since the age of 15 followed by hyperthyroidism after labor (Fig. 1)

## DISCUSSION

The similarity in biological structure between  $\beta$ -hCG and TSH allows  $\beta$ -hCG to interact with the TSH receptor and stimulate the thyroid gland, thus causing symptoms of hyperthyroidism [2]. Another presentation of choriocarcinoma is presence of theca lutein cysts, that form because of ovarian overstimulation from high  $\beta$ -hCG levels [3].

If the coexistence of the mentioned symptoms resulted in the control of beta  $\beta$ -hCG levels, it could enable early detection of GTN. Unfortunately, the  $\beta$ -hCG level (Tab. 1) was measured only in the advanced stage of the disease, which is characterized by distant metastases and their severe manifestations. Due to progress in effective treatment of early stages of the disease, advanced stages of choriocarcinoma are now rarely encountered. Unfortunately, the uncharacteristic clinical picture makes this disease a diagnostic challenge for clinicians. Moreover, severity of the disease course and sparse specific guidelines for effective chemotherapy often result in unsuccessful treatment of choriocarcinoma.

## **CONCLUSIONS**

Based on the presented case, we would like to draw particular attention to the fact that, not only patients with a history of hydatidiform or molar pregnancies should be registered and stay under clinicians' surveillance, but also patients after term pregnancies, as in their case such a diagnosis cannot be excluded. Overall cure rates can exceed 98% if the disease is diagnosed at the early stage [4]. However, omitting the elevated level of  $\beta$ -hCG may cause diagnostic bias due to several atypical symptoms in advanced disease.

## **Article information and declarations**

### **Ethics statement**

The patient's family was informed about the publication of clinical vignette and did not express any objections. The confidentiality of patient data was ensured at every stage of the work.

### **Author contributions**

Julia Rudnicka — study design, acquisition of data, data analysis and interpretation, case description, article draft (35%); Aleksandra Urban — study design, case description, data analysis and interpretation, article draft (30%); Julia Gorny — article draft (5%); Joanna Kacperczyk-Bartnik — article draft (5%); Anna Danska-Bidzinska — study design, revised article critically (10%); Ewa Romejko-Wolniewicz — concept, assumptions, revised article critically (20%).

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None.

## Conflict of interest

The authors declare that they have no conflict of interest.

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Laboratory tests:
CA 125 – 685 UI/l
β-hCG – 2.426.500 mIU/ml
fT3 – 31 pmol/l
fT4 – > 100 pmol/l
TSH – <0,005 mU/l
Antithyroid antibodies (ATG, ATPO, aTSHr) - negative

CA – cancer antigen; β-hCG – beta human chorionic gonadotropin; fT3 – free triiodothyronine; fT4 – free thyroxine; TSH – thyroid stimulation hormone; ATG – antithyroidglobulin antibodies; ATPO – antithyroid peroxidase antibodies; aTSHr – antithyroid stimulating hormone receptor antibodies

Table 1. Laboratory test results on admission

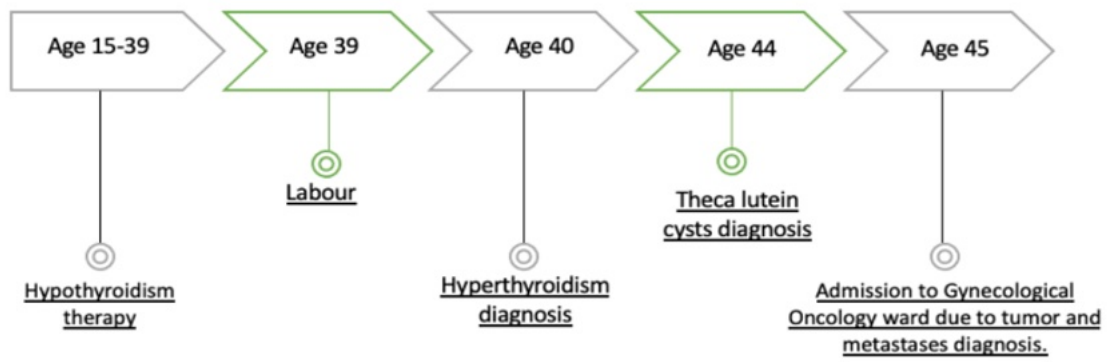


Figure 1. Chronological presentation of patient's medical history