

HYDATIDIFORM MOLE: DIAGNOSTICS AND PROGNOSIS

PhD, associate professor of the oncology department Yakubbekova S.S

Assistant of the Department of General Surgery and Transplantology

Hoshimov I.M.

Oncology Department 1st year Master's degree Hoshimova Sh. F.

Andijan State Medical Institute.

Abstract. Hydatidiform mole (HM) belongs to a group of diseases united by the term "trophoblastic disease" and developing from trophoblast tissue. It was first described by Hippocrates in 400 BC as "dropsy of the uterus". In North America and Europe, the total incidence of HM is 66-121 cases per 100,000 pregnancies [1, 2]. HM is characterized by a benign course and is classified as complete (CPM) and partial (PPM). Malignant trophoblastic disease is called gestational trophoblastic neoplasia (trophoblastic tumor), according to its histological structure it is divided into invasive HM, choriocarcinoma, placental bed trophoblastic tumor, and epithelioid trophoblastic tumor [1].

Key words: Hydatidiform mole, fertilization, trophoblastic tumor, chorionic gonadotropin

ПУЗЫРНЫЙ ЗАНОС : ДИАГНОСТИКА И ПРОГНОЗ

Кандидат медицинских наук, доцент кафедры онкологии Якуббекова

С.С

Ассистент кафедры общей хирургии и трансплантологии Хошимов И.М.

Кафедра онкологии 1 курс магистратуры Хошимова Ш. Ф.

Андижанский государственный медицинский институт.

Абстрактный. Пузырный занос (ПЗ) относится к группе заболеваний, объединенных термином «трофобластическая болезнь» и развивающихся из ткани трофобласта. Впервые она была описана Гиппократом в 400 году до нашей эры как «водянка матки». В Северной Америке и Европе общая заболеваемость ПЗ составляет 66-121 случай на 100 000 беременностей [1, 2]. ПЗ характеризуется доброкачественным течением и классифицируется на полную (СРМ) и частичную (РРМ). Злокачественное трофобластическое заболевание называется гестационной трофобластической неоплазией (трофобластической опухолью), по гистологическому строению подразделяется на инвазивную ПЗ, хориокарциному, трофобластическую опухоль плацентарного ложа и эпителиоидную трофобластическую опухоль [1].

Ключевые слова: язычковая родинка, оплодотворение, трофобластная опухоль, хорионический гонадотропин

Unlike neoplasia, HM occurs after aberrant fertilization, and not as a consequence of a previous pregnancy. It is characterized by the formation of abnormal chorionic villi with trophoblast hyperplasia. According to the karyotype, PZ is usually diploid (up to 80%), and CHZ is triploid. PZ is formed as a result of fertilization of an egg by a single sperm with the formation of a zygote containing only paternal chromosomes. Maternal chromosomes are lost during meiosis. Against the background of PZ, symptomatic hyperthyroidism may develop, associated with excessive stimulation of hCG (levels over 100,000 mIU/ml, persisting for several weeks) of the thyroid tissue. In some cases, this requires the administration of antithyroid drugs. Clinically, tachycardia, sweating, and tremor are noted. To relieve cardiac manifestations of hyperthyroidism, b-blockers are used [3]. A national study was conducted in Japan with 18 patients with PZ of one fetus.

Gestational trophoblastic neoplasia developed in 50% of patients, and metastases were found in 33% [2]; 13 patients wanted to continue the pregnancy, but in 10 it was terminated for medical reasons (preeclampsia, hemorrhage, intrauterine fetal death). Three women gave birth to healthy children before 32 weeks.

Hysterectomy is recommended for the treatment of trophoblastic neoplasia. This procedure significantly reduces the likelihood of developing trophoblastic neoplasia. In a study of 82 patients aged 40 to 49 years with PPH, gestational trophoblastic neoplasia developed in 54% of women after curettage and was absent after hysterectomy [23]. An indicator of a high risk of developing neoplasia is the hCG level $\geq 175,000$ mIU/ml (risk 60%) [4]. However, the probability of metastatic lesions remains after hysterectomy, amounting to 4% [24]. Prophylactic chemotherapy is administered after surgery in patients at high risk of developing neoplasia [1]. It is important to note that chemoprophylaxis does not affect subsequent fertility. Methotrexate or actinomycin D can be used [2,6]. Prophylactic chemotherapy helps to reduce the risk of developing neoplasia by 63% (relative risk 0.37, 95% confidence interval 0.24–0.57) [25]. Patients with Rh-negative blood are given a prophylactic dose of anti-Rh immunoglobulin after emptying the uterine cavity, since the fetal Rh factor antigen is expressed by trophoblast cells, among other things. After emptying the uterus (instrumental, childbirth), dynamic laboratory monitoring is necessary (Fig. 1) [9]. A decrease in the b-hCG level is a decrease of more than 10% compared to the previous result. An increase in the b-hCG level is defined as an excess of 10% or more compared to the previous result within 2 weeks. A b-hCG level is considered stable if it remains within $\pm 10\%$ compared to the previous result and continues to correspond to it over a 3-week period. b-hCG level

A hydatidiform mole, or molar pregnancy, is a pathological condition associated with abnormal development of placental tissue. This pathology usually occurs as a result of disorders in the early stages of conception and can lead to serious consequences for the woman's health, including placental cancer. In this regard,

early prevention of this condition plays an important role, especially in women with a predisposition to molar pregnancies. A hydatidiform mole, also known as a molar pregnancy, is an abnormal condition that occurs during pregnancy in which abnormal chorionic villi are formed. [4] This disease is mainly associated with disorders that occur during fertilization and subsequent implantation of the embryo. Diagnosis of hydatidiform mole in women is key to timely detection and prevention of possible complications. The main methods for diagnosing hydatidiform mole are ultrasound and analysis of the level of human chorionic gonadotropin (hCG) in the blood. Ultrasound examination can reveal specific signs such as "grape clusters" indicating the presence of abnormal villi. The hCG level in molar pregnancy significantly exceeds normal values, which also becomes an important diagnostic criterion. Clinical manifestations of hydatidiform mole can vary, but usually include symptoms such as bleeding, vomiting and abdominal pain. Consulting a doctor at the first signs of a disorder can significantly reduce the risk of developing serious complications such as chorionepithelioma. Thus, the diagnosis of hydatidiform mole in women is an important aspect in obstetrics and gynecology. High-quality and timely diagnostics allows not only to preserve the patient's health, but also to prevent possible complications associated with this condition.[5]

Early prevention of hydatidiform mole includes several key aspects. Firstly, it is important to conduct regular gynecological examinations to identify menstrual cycle dysfunction and other hormonal disorders. It is also advisable to pay attention to the history of previous pregnancies, since the presence of molar pregnancies in the anamnesis increases the risk of recurrence of this pathology.

In conclusion, predicting hydatidiform mole in women is a multifaceted task that includes not only medical but also sociocultural aspects. Sustained attention to this issue and the development of individualized approaches to prognosis and treatment will improve the quality of life of women and reduce the risk of complications associated with hydatidiform mole. Hydatidiform mole is part of a group of

diseases classified as gestational trophoblastic disease and develops as a result of aberrant fertilization. It can be complete or partial. GTD is characterized by a diploid karyotype, while PPD is triploid. GTD is characterized by higher b-hCG titers and an increased risk of developing malignant neoplasia of up to 15–20%. The main risk factors for trophoblastic disease are maternal age (over 35 years) and a history of the disease.

References

1. Altieri A, Franceschi S, Ferlay J et al. Epidemiology and aetiology of gestational trophoblastic diseases. *Lancet Oncol* 2003; 4: 670.
2. Azuma C, Saji F, Tokugawa Y et al. Application of gene amplification by polymerase chain reaction to genetic analysis of molar mitochondrial DNA: the detection of anuclear empty ovum as the cause of complete mole. *Gynecol Oncol* 1991; 40: 29.
3. Szulman AE, Surti U. The syndromes of hydatidiform mole. I. Cytogenetic and morphologic correlations. *Am J Obstet Gynecol* 1978; 131: 665.
4. Melamed A, Gockley AA, Joseph NT et al. Effect of race/ethnicity on risk of complete and partial molar pregnancy after adjustment for age. *Gynecol Oncol* 2016; 143: 73.
5. Vargas R, Barroilhet LM, Esselen K et al. Subsequent pregnancy outcomes after complete and partial molar pregnancy, recurrent molar pregnancy, and gestational trophoblastic neoplasia: an update from the New England Trophoblastic Disease Center. *J Reprod Med* 2014; 59: 188.
6. Hershman JM. Physiological and pathological aspects of the effect of human chorionic gonadotropin on the thyroid. *Best Pract Res Clin Endocrinol Metab* 2004; 18: 249.