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Case Report of Hyperthyroidism on Molar Pregnancy

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Abstract **Case Report**

The hydatidiform mole in its partial or total form is a common cause of thyrotoxycosis during pregnancy. The structural homology of the placental hormone choriogonadotropin (hCG) and the hormone thyroid stimulating hormone (TSH) produced by the pituitary gland is the cause. The diagnosis of hyperthyroidism secondary to Gestational trophoblastic diseases is made in the face of low or undetectable TSH, high T4l compared to high hCG level and a suggestive image on pelvic ultrasound. The clinical signs relating to thyropathies are often absent. This is a resolving table after uterine extraction.

Keywords: Hydatiform mole, molar pregnancy, hyperthyroidism.

INTRODUCTION

The hydatidiform mole in its partial or total form is a common cause of thyrotoxycosis during pregnancy. The structural homology of the placental hormone choriogonadotropin (hCG) and the hormone thyroid stimulating hormone (TSH) produced by the pituitary gland is the cause. We report a case of hyperthyroidism in molar pregnancy through an observation.

CASE REPORT

A twenty seven year-old patient with a history of intrauterine fetal death (IUFD) 5 years ago, who was admitted to maternity intensive care for incoercible vomiting with hydro-electrolytic repercussions on pregnancy estimated at 11 weeks with the presence of partial hydatidiform mole.

On examination we find a restless patient with dry mucous membranes, fine extremity tremors and tachycardia at 118bpm. There was no exophthalmos or goiter and the remainder of the exam was unremarkable.

On balance we have a low TSH at 0.01mui / ml (0.27-4.20), a high Ft_4 at 28.3pmol / L (12-22) as well as FT_3 at 7.9pmol / L (3.10 -6.80) all compared to high bHCG 540984 or +1.9 normal.

Obstretrical ultrasound shows an enlarged uterus, a flattened Intrauterine Fetal Death (IFUD) and an abnormally large placenta with a snowflake image suggesting a partial mole. We have no goiter or signs of thyroiditis on the cervical ultrasound.

The electrocardiogram (EKG) is unremarkable apart from sinus tachycardia at 120bpm.

An immunological assessment was carried out with negative income, in particular the anti TSH receptor and anti TPO Ab.

The management consisted of conditioning followed by the prescription of synthetic ant thyroid drugs and b-blocker after collegial discussion at the rate of carbimazole 30mg / d and propranolol 40mg / d to obtain euthyroidism before the uterine evacuation.

The course is marked on day 4 by clinical improvement with amelioration of symptoms and biological euthyroidism with T4L at 13pmol / l after uterine evacuation.

DISCUSSION

Gestational trophoblastic disease (GTD) includes a broad spectrum of placental pathologies characterized by cystic transformation of the chorionic

villi associated with trophoblast proliferation with excessive secretion of the hormone choriogonadotropin (hCG) [1, 2]. Their diagnosis is often suspected by a picture of a uterus increased in size compared to gestational age, an appearance suggestive on pelvic ultrasound and high plasma hCG levels [3, 4]. In our case we have a GDT in its partial mole form on ultrasound concomitantly with a level of bhCG close to twice the normal for the term. The lack of impact on uterine volume could be due to the partial nature of this mole in our patient.

Human placental specific hormone (hCG) is a glycoprotein hormone that has an alpha subunit identical to that of the thyroid stimulating hormone (TSH) as well as pituitary gonadotropins and a specific beta subunit [3, 4]. It is expressed strongly during pregnancy, but also physiologically outside pregnancy with very low noise and pathologically in tumors such as partial or total hydatidiform mole.

In our case, the clinical presentation was uncontrollable vomiting with some signs of thyrotoxic syndrome such as restlessness, tachycardia and fine tremors of the extremities. These signs are generally absent in a normal pregnancy despite high levels of hCG, peaking around the 10th-12th week of amenorrhea [5]. Under normal conditions, the plasma concentrations of the various glycoprotein hormones do not allow cross reactions [6].

The symptoms of hyperthyroidism associated with GTD are caused by the high concentrations of hCG acting on the TSH receptor in the thyroid and consequently the release of thyroid hormones [5].

Thyrotoxicosis is a classic and rare complication of GTDs, especially hydatidiform moles [7]. It is explained by a structural and functional analogy between (TSH) and hCG, which gives it thyrostimulin activity [3, 8].

In hyperthyroidism secondary to trophoblastic diseases, patients do not present the signs found in Graves' disease [5]. Thus in our case, the remainder of the clinical examination did not show signs in favor of an underlying thyropathy with an unremarkable morphological and immunological assessment.

The biological profile shows an increase in free T4 and free T3 with an undetectable TSH level [5]. as found in our case with suppressed TSH and high peripheral hormones compared to a high bhCG level for the term. The beta subunits of glycoprotein hormones although different include similar regions.

The presence of IUFD could be attributed to the direct effects of thyroid hormones on the fetus.

Hospitalization in intensive care and the use of plasmapheresis has been reported by Moskovitz *et al.*, [9]. In our case, the patient was certainly conditioned in an intensive care unit, but without resorting to plasmapheresis.

Treatment of such symptoms includes synthetic antithyroid drugs, treatment of peripheral effects (beta blockers), and, if applicable, the triggering event, which is molar pregnancy, by uterine evacuation [5]. In our case, treatment with ATS and b-blockers was in fact started before the uterine evacuation was performed.

In our case, the signs of hyperthyroidism had disappeared just after uterine evacuation due to the normalization of peripheral hormones. The course was favorable without complications.

CONCLUSION

The diagnosis of hyperthyroidism secondary to GTD is made in the face of low or undetectable TSH, high T4l compared to high hCG level and a suggestive image on pelvic ultrasound. It is a condition due to the structural homology between TSH and hCG. The clinical signs relating to thyropathies are often absent. This is a resolving table after uterine extraction.

Conflict of Interest: The authors declare no conflict of interest.

Authors' Contribution: All authors contributed to the development of this work.

REFRENCES

1. Boufettal, H., Coullin, P., Mahdaoui, S., Noun, M., Hermas, S., & Samouh, N. (2011). Les môles hydatiformes complètes au Maroc: étude épidémiologique et clinique. *Journal de gynécologie obstétrique et biologie de la reproduction*, *40*(5), 419-429.
2. Boufettal, H., Coullin, P., Mahdaoui, S., Noun, M., Hermas, S., & Samouh, N. (2012). Les môles hydatiformes partielles au Maroc: étude épidémiologique et clinique. *Eastern Mediterranean Health Journal*, *18*(7), 755-761.
3. Yoshimura, M., Nishikawa, M., Yoshikawa, N., Horimoto, M., Toyoda, N., Sawaragi, I., & Inada, M. (1991). Mechanism of thyroid stimulation by human chorionic gonadotropin in sera of normal pregnant women. *European Journal of Endocrinology*, *124*(2), 173-178.
4. Kimura, M., Amino, N., Tamaki, H., Mitsuda, N., Miyai, K., & Tanizawa, O. (1990). Physiologic thyroid activation in normal early pregnancy is induced by circulating hCG. *Obstetrics and gynecology*, *75*(5), 775-778.
5. Boufettal, H., Mahdoui, S., Noun, M., Hermas, S., & Samouh, N. (2014). Hyperthyroïdie au cours

d'une grossesse molaire. *La Revue de Médecine Interne*, *35*(3), 202-205.

6. Glinoer, D. (1997). The regulation of thyroid function in pregnancy: pathways of endocrine adaptation from physiology to pathology. *Endocrine reviews*, *18*(3), 404-433.
7. Rajatanavin, R., Chailurkit, L. O., Srisupandit, S., Tungtrakul, S., & Bunyaratvej, S. (1988). Trophoblastic hyperthyroidism: clinical and biochemical features of five cases. *The American journal of medicine*, *85*(2), 237-241.
8. Kokuho, T., Kuji, T., Yasuda, G., & Umemura, S. (2004). Thyroid Storm-induced Multiple Organ Failure Relieved Quickly by Plasma Exchange Therapy. *Therapeutic Apheresis and Dialysis*, *8*(4), 347-349.
9. Moskovitz, J. B., & Bond, M. C. (2010). Molar pregnancy-induced thyroid storm. *The Journal of emergency medicine*, *38*(5), e71-e76.