Gestational trophoblastic disease with multisistem complications

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ABSTRACT
Gestational trophoblastic disease (GTD) is a condition of uncertain etiology, comprised of a hydatidiform mole (complete and partial), invasive mole, choriocarcinoma, epithelioid trophoblastic tumour and placental site trophoblastic tumour. A partial hydatidiform mole develops when dispermy occurs, and the resulting conceptus is triploid. A 26-year-old woman (Gravida 2, Para 1, with one previous vaginal delivery of a normal female infant) was 16 weeks pregnant and was scheduled for emergency surgical treatment. She was diagnosed with a hydatidiform mole and eclampsia, and kept in our hospital for further treatment. Her pre-treatment beta human chorionic gonadotropin (β-HCG) level was extremely high at 1,082,900 mIU/ml. The obstetricians considered septic complications from the hydatidiform mole and we decided to perform an emergency Sectio parva. Two weeks after delivery, the serum β-hCG level was 16,341 mlU/mL and normalized gradually within two months without any cytotoxic therapy. Partial mole hydatidosa (PMH), as a milder form of GTD, can go along with malignant complications with fatal consequences.

Key words: gestational trophoblastic disease, eclampsia, chorionic gonadotropin beta subunit.

INTRODUCTION
Gestational trophoblastic disease (GTD) is a condition of uncertain etiology, comprised of a hydatidiform mole (complete and partial), invasive mole, choriocarcinoma, epithelioid trophoblastic tumour and placental site trophoblastic tumour. (1) The incidence of a complete hydatidiform mole is approximately 1 in 1,500 pregnancies in the United States. The incidence of a partial hydatidiform mole is approximately 1 in 750 pregnancies. A complete hydatidiform mole is usually due to an androgenetic diploid conception, in which a haploid sperm fertilizes an egg that lacks female chromosomes. (2) A partial hydatidiform mole (PHM) develops when dispermy occurs, and the resulting conceptus is triploid. (1)

CASE REPORT
In this case report we describe a patient with symptoms of eclampsia induced by molar pregnancy. A 26-year-old woman (Gravida 2, Para 1, with one previous vaginal delivery of a normal female infant) was 16 weeks pregnant and was scheduled for emergency surgical treatment. She was diagnosed with a hydatidiform mole and eclampsia, and kept in our hospital for further treatment. She had no medical or surgical history, and her vital signs upon admission were 70/100 mm Hg, 88/min-18/min-36.9°C. On admission she had irregular vaginal bleeding, lower abdominal pain, syncope, headache, excessive vomiting, transvaginal expulsion of grape-like vesicles, an abnormally enlarged uterus and features of eclampsia. Her pre-treatment beta human chorionic gonadotropin (β-HCG) level was extremely high at 1,082,900 mIU/ml (normal: 0.5-2.9 mIU/ml).

Her base line investigation showed Hb-132g/L, PLT-278x109/L, normal differential count, ABO/Rh B +ve, proteinuria 3+, dipstick and Thyrotropin (TSH) was 1.88 uIU/ml with normal free thyroxine (FT 4). Her blood sugar level, renal and liver function tests were all within normal limits. An ultrasound examination performed on admission showed a viable fetus with...
normal anatomy and placenta located at the posterior uterine wall, and a second multicystic molar-appearing placenta. The ovaries showed bilateral multilocular cystic masses with a dimension of 70x40 mm. The magnetic resonance imaging (MRI) of the brain showed hypodense lesions at the gray-white matter junction and diffuse cerebral edema primarily in the parieto-occipital lobe, frontal and inferior temporal lobes.

The obstetricians suspected eclampsia and magnesium sulphate and sodium thiopental were administered repeatedly, but they were not effective for controlling the blood pressure and heart rate. The obstetricians considered septic complications from the hydatidiform mole and it was decided that an emergency sectio parva under general anaesthesia would be performed.

On pathological examination, the placenta was 22x16x4 cm, weight 422.5 g. The microscopic findings consisted of partial mole hydatidosa with trophoblastic atypias, mild hyperplasia and spontaneous degeneration areas. The pathology diagnosis was gestational trophoblastic disease – partial mole hydatidosa (PMH).

At the end of the 40 minute operation, the patient was sent to the recovery room in an intubated state. Her estimated blood loss was 200 ml and 500 ml crystalloid was infused (figure 1).

In the Intensive Care Unit, and on the first day after the operation, the patient was put on mechanical ventilation and developed right side paralysis, amaurosis, hypoprothrombinaemia (40 g/L).

Postoperatively, she received parenteral therapy: antibiotics meropenem and metronidazol antimycotics aciklovir, human albumins, antiepileptics drug levetiracetam, crystalloid and colloids solutions. Seven days after the operation, subsequent investigations confirmed normal thyroid function tests, normal vision, normal blood pressure, no proteinuria and a normal chest X-ray.

Two weeks after delivery, the serum β-hCG level was 16,341 mIU/mL and normalized gradually within two months without any cytotoxic therapy and with no evidence of persistent or metastatic disease. Careful follow-up has so far shown no sign of persistent trophoblastic disease (table 1).

The patient has been transferred to the Institute of Physical Medicine and Rehabilitation “Dr. Miroslav Zotović” for physical therapy.

Table 1. Evolution of beta-HCG levels.

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Beta-HCG: beta human chorionic gonadotropin

REFERENCES