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RECURRENT MOLAR PREGNANCY

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Abstract : Gestational Trophoblastic Disease (GTD) originates from placental tissue and is among the rare human tumors that can be cured even in the presence of widespread metastases. GTD include a spectrum of interrelated tumors including complete and partial hydatidiform mole, invasive mole, choriocarcinoma, and placental site trophoblastic tumor, that have different propensities for local invasion and spread. Although most GTD develop after a mole, they can follow any antecedent pregnancy.Transvaginal ultrasound, routinary dosage of beta-hCG and current approaches to chemotherapy, let most women with malignant gestational trophoblastic disease to be cured and their reproductive function preserved. We present a case of 26 yrs old woman who presented with recurrent molar pregnancy

Keyword : gestational trophoblastic disease, human chorionic gonadotropin, chemotherapy

1. Introduction

Gestational trophoblastic disease (GTD) is a tumor originating from the trophoblast, which surrounds the blastocyst and develops into the chorion and amnion. The main types of gestational trophoblastic diseases are:

- Hydatidiform mole (complete or partial);
- Invasive mole;
- Choriocarcinoma;
- Placental site trophoblastic tumor.

The most common form of GTD is hydatidiform mole, also known as molar pregnancy. There are 2 types of hydatidiform moles: complete and partial. The *complete* hydatidiform mole is usually diploid and entirely androgenetic in origin. Most have 46,XX karyotype; a few have a 46,XY karyotype. A complete molar pregnancy consists of diffuse hydropic chorionic villi with trophoblastic hyperplasia, forming a mass of multiple vescicles. There is usually no evidence of a fetus and minimal embryonal development. The *partial* hydatidiform mole is usually triploid, with one maternal and two paternal haploid sets, either from dispermic fertilization or from fertilization with an unreduced diploid sperm. There is usually a fetus and a large placenta. The hydropic villi show a less florid appearance than is seen with a complete hydatidiform mole and are interspersed with normal chorionic villi. The fetus usually dies within a few weeks of conception, and a recent review did not identify any case in which a fetus of paternal (diandric) origin survived to term (1). Very rarely, a partial molar pregnancy develops with two maternal and one paternal haploid set (digynic). In these cases, the placenta is small, the villi show minimal hydropic changes, and the fetus is growth-restricted. Some of these pregnancies have been reported to result in live births, with subsequent early neonatal death (2). Of 3,000 women with partial hydatidiform moles, 0.1% had a choriocarcinoma. Persistent trophoblastic disease or malignant complications are much more common with a complete molar pregnancy than with a partial hydatidiform mole. The incidence of these complications is approximately 8% and 0.5% respectively, compared with a risk of 1:50,000 after a full-term pregnancy. Although recurrent molar pregnancies are a rare occurrence seen in 1 to 2%, it is clear that women who have had a previous mole have a higher risk of recurrence than the general population.

2. Case report

A 23 yrs old G6A5 married since 7 yrs previous 2 molar pregnancy 62 days of amenorrhoea, came with the complaints of spotting per vaginum. No history of lower abdominal pain. No history of medical illness. Her previous obstetric history includes
I- Vesicular mole, suction and evacuation done at 3MA in 2006. Patient did not turn up for follow up.
II- Partial mole, suction evacuation done at 2MA in 2007. Patient did not turn up for follow up.
III- Spontaneous abortion at 2MA, certified at private hospital
IV- Spontaneous abortion at 2MA, certified at private hospital
V- Spontaneous abortion at 2MA, certified at private hospital
VI- Present pregnancy.
On receiving, patient was mildly anemic, vitals stable, CVS, RS- clinically normal, abdomen soft, bimanual examination- cervix pointing upwards, uterus retroverted, 12 weeks size, bleeding present. Hb-10.2g%, urine albumin, sugar nil, FBS 80mg%, 1 hr 161mg%, 2 hr 134mg%, Serum HCG 5006 mIU/ml, TC- 11,200 cells/cumm, DC P62%, L31%, E4%, M3%,

RBC 3.52 million/cumm, Hb 10.7g%, PCV 32.5, Platelet 2.22 lakh/ cumm, ESR 35mm/hr, BT 2' 45" CT 4', Sr bilirubin 0.6mg%, blood group- B positive. Ultrasound abdomen and pelvis done - Normal size uterus 5.9*4.8cm, endometrial cavity is distended with hyperechoeic contents measuring 3.3*2.3cm size showing multiple small cystic spaces along with minimal fluid collection. No evidence of fetal pole/ yolk sac. No evidence of fibroid. Myometrial echoes normal. Both lips of cervix normal. Both ovaries normal sized, multiple follicles. Left ovary shows remnant of theca lutein cyst. Both adnexa free, moderate amount of free fluid in POD. Other organs normal. Imp- s/o molar pregnancy, for HCG correlation Suction evacuation done with one whole blood on flow. Chest X ray, liver function tests, thyroid profile, renal function tests were done. HCG a week later 5800mIU/ml, 2nd week 6100mIU/ml. Medical oncology opinion obtained. She was started on methotrexate regimen 50mg i.m on day 1,3,5,7 alternated with folinic acid 5mg. 3 cycles given. HCG reduced to 10mIU/ml. Three more cycles given. Discharged with advice for regular follow up with HCG monthly with oral contraceptives.

3. DISCUSSION

The cause of molar pregnancy is unclear; however, there are several risk factors. Molar pregnancies occur at extremes of the childbearing age. For women over 40 years of age, there is a 10-fold increase, compared with only 1.3-fold increased risk in teenagers.[5] Other factors postulated to increase the risk of HM have included diet, gravidity, and contraception.[6,7] The incidence of recurrent molar pregnancy ranges from 5- to 40-fold increase in the current literature.[2] In a report from the United Kingdom for women who had already had two molar pregnancies, the subsequent risk increases to 1 in 6.5 pregnancies.[4] This risk diminishes if there is a normal pregnancy following the HM. Familial predisposition has recently been evaluated. Familial recurrent HM are considered exceedingly rare, with only 21 families reported in the medical literature. In these cases, the HM are diploid, but biparental, rather than androgenetic in origin. These patients appear to have an autosomal recessive condition, causing them to have recurrent molar pregnancies and they have very little chance of a successful pregnancy. However, this patient had no known family history of recurrent HM. Genetic studies suggest mutations in the NLRP7 gene, also known as NALP7 gene, which is located on chromosome 19q13.3–q13.4, a maternal gene, as a cause of Familial biparental HM, and possibly responsible for causing recurrent spontaneous abortions, stillbirths, and intrauterine growth retardation.[8]. Women having a pregnancy affected by a complete or partial hydatidiform mole may be counselled that the risk of repeat mole in a subsequent pregnancy is about 1 in 60 Follow up of patients with HM by measuring serial -hCG levels is very crucial to allow early detection of persistent gestational trophoblastic disease (PTD) which has high potential to malignant change. Malignant transformation may be life-threatening to the mother and needs urgent treatment. Patients with complete molar pregnancies have an increased risk of PTD, considered to be 5% compared with patients with partial molar pregnancies where it is <1%.[5] Women who receive chemotherapy for GTD are likely to have an earlier menopause.[9] Furthermore, multi agent chemotherapy which includes etoposide increases the risk of developing secondary cancers, such as acute myeloid leukemia, colon cancer, melanoma, and breast cancer for those who survive more than 25 years.[10] These risks would necessitate long term follow-up of these patients treated with chemotherapeutic agents

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