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EARLY PREGNANCY FAILURE SECONDARY TO PARTIAL MOLAR PREGNANCY- A CASE REPORT AND REVIEW OF LITERATURE.

Obstetrics & Gynaecology

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ABSTRACT

Molar pregnancy or hydatidiform mole rarely occurs during pregnancy, belonging to a group of diseases known as Gestational Trophoblastic diseases (GTD) due to abnormal placentation and its potentially metastatic nature. The characteristic feature of a hydatidiform mole is fertilization abnormalities that lead to villous hydrops and hyperplasia of trophoblasts, with or without embryonic development. The overall incidence of molar pregnancy is around 1 in 1,200, whereas partial molar pregnancy occurs less commonly than complete molar pregnancy, with a reported incidence of 0.005% to 0.1% of all pregnancies. Sporadic overexpression of the paternal genome in hydatidiform moles is considered a fundamental genetic event, resulting in altered gene imprinting in molar trophoblasts – completely androgenetic in complete moles and diandric triploid in partial molar pregnancies. Although molar pregnancies are considered benign, they often have a premalignant nature and can become invasive and malignant. We hereby report a case of a 24-year-old Primigravida who presented with features of early pregnancy failure at 15 weeks of gestation secondary to partial molar pregnancy, with a very high level of HCG (>19,424mIU/ml).

KEYWORDS

Hydatidiform mole, Molar pregnancy, Gestational Trophoblastic diseases (GTD), Partial mole , Complete Mole, B- human chorionic gonadotropin

INTRODUCTION

Gestational Trophoblastic diseases (GTD) are defined as a group of tumors characterized by abnormal proliferation of trophoblasts, classified into Hydatidiform mole, Gestational Trophoblastic Neoplasia (GTN), Choriocarcinoma, Epithelioid Trophoblastic Tumor (ETT), and Placental Site Trophoblastic Tumor (PSTT), commonly occurring after molar pregnancy. [1,2] Abnormal proliferation of cytotrophoblasts and syncytiotrophoblasts produces Hydatidiform mole and Choriocarcinoma, whereas Intermediate Trophoblasts are commonly associated with ETT and PSTT.[3]

Hydatidiform mole has two subclasses: Complete molar and Partial molar pregnancy. Complete Mole is the most common type of molar pregnancy, occurring when an enucleated egg is fertilized by a haploid sperm or two sperms before duplication. Complete moles do not contain fetal parts, have paternally derived chromosomes with maternal mitochondrial DNA, and typically have a 46,XX karyotype (90%) or 46,XY karyotype (10%), with high levels of HCG. [3,4] Partial molar pregnancies are characterized by fertilization of a haploid ovum by a haploid sperm or by two sperms duplicating upon fertilization of a haploid ovum. Generally, a triploid karyotype (69,XXX, 69,XXY, 69,XYY) pattern is more common than a diploid karyotype, which occurs due to fertilization of an empty ovum by two sperms. Partial moles contain both maternal and paternal DNA and identifiable fetal parts.[3]

The risk factors for molar pregnancy include age extremes (>35 years or <21 years, with a 7.5 times higher risk in women >40 years), ethnicity, genetic etiology with a prior history of Hydatidiform mole (1%), and dietary factors like carotene and animal fat deficit diet, and smoking, which increases the risk of molar pregnancy. A history of spontaneous abortion increases the risk of molar pregnancy by 2 to 3 fold compared to women without spontaneous abortions. The recurrence rate of molar pregnancy in subsequent pregnancy is 15%-20%.[1,3,5]

The pathogenesis of Molar pregnancy is characterized by abnormal placental tissue development in the absence of a viable fetus, often presenting as oedematous immature placentas. Due to abnormal proliferation of chorionic villi, unusual transformation of trophoblasts occurs, which ultimately results in obliteration of vessels and fetal death.[6] Another theory suggests that dysmorphic villi and trophoblastic proliferation result in spontaneous stillbirth, and vasculogenic deficit in trophoblastic tissues decreases angiogenesis in early complete mole, associated with progressive accumulation of fluids with subsequent cystic space formation ("cistern"). [7]

Complete Molar pregnancy is often associated with the development of placental parts only, while partial molar pregnancy is associated with the development of fetal parts as well.[6,7] The clinical manifestations of Hydatidiform mole include heavy bleeding per vaginum in early weeks of pregnancy due to separation of molar tissue from decidua in the first trimester of pregnancy. Vaginal bleeding often appears dark brown or prune juice-like in color due to oxidation and liquefaction of accumulated blood products in the uterine cavity, and passage of vaginal tissues resembles grape-like vesicles or clusters.[8]

In the first trimester, common symptoms like nausea, vomiting (hyperemesis) are more pronounced due to high levels of HCG, and Pregnancy-induced hypertension (PIH) occurs. During the second trimester (14-16 weeks), signs and symptoms of hyperthyroidism (tremors, tachycardia), Theca lutein cysts, PIH, pre-eclampsia with or without proteinuria/end-organ dysfunction should be considered in the diagnosis of complete molar pregnancy, although some cases may be asymptomatic.[9] Rarely, respiratory distress secondary to pulmonary embolization of trophoblastic tissues may occur. In complete mole, the uterus size is larger than the period of amenorrhea, with absent fetal heart sounds, whereas in partial mole, the uterus size is smaller than the period of gestation. Partial mole often presents with similar clinical symptoms of spontaneous or threatened abortion, often presenting as missed abortion. Histopathological findings after surgical evacuation confirm the diagnosis of molar pregnancy.[10] After surgical evacuation, 15%-20% of cases with Hydatidiform mole will develop malignant transformations with invasive and metastatic potential.[3,10]

We hereby present a case of a 24-year-old Primigravida who presented with features of early pregnancy failure at 15 weeks of gestation secondary to Partial molar pregnancy with a very high level of HCG (19,424.1 mIU/ml).

Case Report

A 24-year-old Primigravida, married for 6 months, presented to our antenatal outpatient department with a history of 4 months of amenorrhea (15 weeks) and an ultrasound abdomen scan showing features of missed abortion, which was taken 1 month prior. She had regular menstrual cycles (3/28 days) with normal flow. Her past medical and surgical history was insignificant. On examination, her vitals were stable. Abdominal examination revealed a uterus size corresponding to 14 weeks of gestation. Bimanual examination confirmed the uterus size to be 14 weeks, with free fornices and no bleeding or discharge per vaginum. Basic investigations were sent and found to be within normal limits: Haemoglobin 12.1 g/dl, platelet count 2,65,000/mm³, TSH 4.2 mg/dl, and coagulation profile was

normal. Chest X-ray and ECG were taken. However, beta-HCG levels were found to be highly elevated at 19,424.1 mIU/ml.

A Trans abdominal ultrasound scan performed at our center revealed features of an irregular intrauterine gestational sac with a mean sac diameter of 30 mm, corresponding to 8 weeks and 4 days. No fetal pole or yolk sac was noted. An ill-defined, heterogeneously hyperechoic area was noted adjacent to the gestational sac, with internal vascularity on color Doppler. A provisional diagnosis of partial molar pregnancy was made. The patient underwent an emergency suction and evacuation procedure, during which multiple vesicles and products of conception were removed and sent for histopathological examination. Post-procedural beta-HCG monitoring was performed at intervals of day 3, day 5, day 7, and day 11, and values were found to be decreasing: 4,859.6 mIU/ml, 1,125.3 mIU/ml, 527.4 mIU/ml, and 31.1 mIU/ml, respectively, without any chemotherapy.

The histopathological findings confirmed the diagnosis of partial molar pregnancy, with features of retained products of conception, hydropic villi with central cistern formation, and focal trophoblastic proliferation. The patient's post-procedure period was uneventful, and she was discharged on the 5th postoperative day. She was followed up for 6 months, and no recurrence or retained tissues were detected.

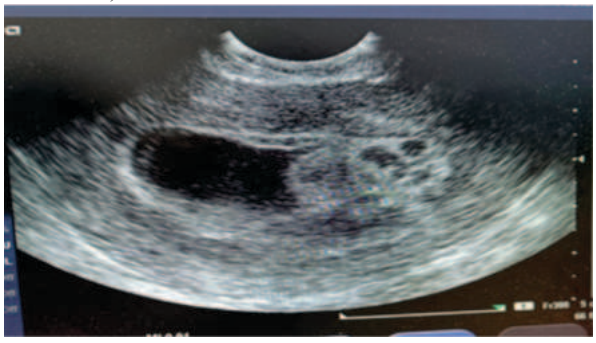

Figure 1. A Trans abdominal Ultrasound image of Partial Molar pregnancy at 15 weeks of menstrual age, showed 8 weeks of gestational age.

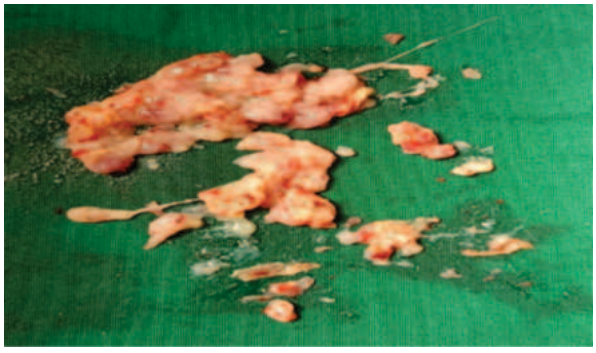

Figure 2. Macroscopic appearance of Multiple vesicles after Suction and Evacuation of Partial Molar pregnancy.

Figure 3. Specimen showing Multiple vesicles of Partial Molar pregnancy.

DISCUSSION

Partial molar pregnancy is a rare complication of all pregnancies, with a reported incidence of 0.005%-0.001%.[1] It often co-exists with an intrauterine fetus due to a triploid set of chromosomes. Due to the higher use of assisted reproductive techniques, its incidence has increased in recent times. The diagnosis of partial molar pregnancy should be considered in the first trimester using ultrasound with color Doppler and serial measurements of serum beta HCG. Clinical features include hyperemesis, heavy bleeding, high blood pressure, proteinuria, symptoms suggestive of pre-eclampsia, and hyperthyroidism due to high levels of HCG, which can mimic TSH. Due to the triploid nature of partial moles, pregnancy can be interrupted by congenital anomalies and severe intrauterine growth restriction due to abnormal placental function.[9,10] In our case, asymptomatic manifestations of partial molar pregnancy were observed. Prompt diagnosis and management of molar pregnancy can prevent complications like abortions, Malpresentations, preterm labor, persistent gestational trophoblastic disease, severe anemia, pulmonary edema, thromboembolic phenomena, and counseling should be given appropriately for regular follow-up with ultrasound and serial beta HCG monitoring.[10]

Complete moles have a 20% tendency of developing into an invasive mole or Choriocarcinoma, so early diagnosis with chorionic villous biopsy should be considered, while the risk is lower for partial moles. Ultrasonographic findings of molar placenta include focal and diffuse hydropic degenerative changes, enlarged placenta with cystic spaces, and a classic "snowstorm" appearance. Theca lutein cysts are also observed in molar pregnancy due to elevated levels of HCG causing ovarian stimulation. In complete moles, an echogenic intrauterine mass with multiple cystic spaces corresponding to hydropic villi is observed in first-trimester ultrasound.[11]

Partial moles are often diagnosed as missed or incomplete abortions, characterized by an enlarged, thickened placenta with fetal components, and amniotic fluid described as having a "Swiss cheese" appearance on ultrasound.[11] In some cases, a placental tumor like chorioangioma presents with a focal vascular placental lesion with a normal diploid fetus. Sometimes, it may coexist with a twin pregnancy. Vascular placental lesions like chorioangioma and placental mesenchymal dysplasia (PMD) should be differentiated from molar pregnancy. Chorioangioma shows sonographic features of a large feeding vessel or increased vascularity inside the tumor with the same pulsation as in the umbilical cord, which is absent in molar pregnancy. PMD is more common in female fetuses, with an incidence of 3.5%, and is characterized by a thickened placenta with hypoechoic areas, low velocity, or absent blood flow inside the placental lesion, whereas high-velocity blood flow is observed in chorioangioma and molar pregnancy.[11]

Although ultrasound is considered the primary modality of imaging, it has high false-negative and false-positive rates, specifically in partial molar cases, so histopathological diagnosis is mandatory for confirmation of diagnosis.[12] Quantitative measurements of beta HCG are significant markers for diagnosis and recurrence. In complete moles, pre-evacuation serum HCG values are higher than in normal pregnancy with the same gestational age or ectopic pregnancy, whereas HCG values are normal for gestational age in partial moles. In our case, HCG values were 19,424 mIU/ml, higher than expected for the period of gestation.

The treatment of molar pregnancy depends on its type, age, childbearing capacity, and uterine size. However, the mainstay of treatment is suction and evacuation for both partial and complete moles, regardless of uterine size.[13] Hysterectomy is an option for women over 40 years of age who have completed childbearing, as it reduces the risk of developing gestational trophoblastic neoplasia (GTN) and subsequent chemotherapy. Medical induction and Hysterotomy are usually not considered due to the risk of hemorrhage.[13]

According to ACOG recommendations, the post-evacuation HCG protocol should be adhered to in all cases for post-molar GTN, including HCG monitoring every week until non-detectable for three consecutive weeks, followed by monthly monitoring for six months. If HCG levels remain undetectable for six consecutive months, patients may be advised to conceive, and effective contraception should be recommended in the meantime. In our case, serial HCG monitoring was performed for three consecutive months, after which the patient

was lost to follow-up.[14] Recent Cochrane reviews do not recommend prophylactic chemotherapy for high-risk complete moles. Based on FIGO criteria, the diagnosis of post-molar GTN is made by serial HCG follow-up, including a plateau level of HCG of four consecutive values over three weeks, an HCG increase of >10% of three consecutive values over two weeks, or persistent detectable serum HCG.[15]

CONCLUSION

Prompt diagnosis and management of molar pregnancy by a multidisciplinary team should be considered in a tertiary care center to provide comprehensive, patient-centered care, optimizing treatment outcomes and enhancing the overall patient experience. In future more studies are required in the field of genetic and molecular mechanisms, treatment protocols for improving patient outcomes.

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