Twin Gestation with One Vesicular Mole and One Normal Foetus

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Abstract

Gestational trophoblastic disease refers to pregnancy related trophoblastic proliferative abnormalities. Hydatidiform mole is the most common type of gestational trophoblastic disease. Very rarely Hydatidiform mole can occur in association with a live twin foetus the incidence being 1 per 22,000 and 1 per 100,000 pregnancies.¹ This is a case report of twin gestation with complete mole in one sac along with a normal foetus in the other which progressed to term followed by delivery of a normal neonate.

Introduction

Gestational trophoblastic disease is a spectrum of disorders ranging from Hydatidiform mole to post molar gestational trophoblastic neoplasia and is characterised by abnormal trophoblastic proliferation with varying risk of metastasis. Hydatidiform mole is divided into complete vesicular mole with absence of identifiable embryonic tissue and partial mole wherein the molar tissue coexists with a foetus which often has genetic and morphological abnormalities. Twin gestation with one molar pregnancy and other live foetus is an extremely rare condition. Differentiation between partial mole and a multifoetal gestation with complete mole with normal foetus is of utmost importance. Studies have shown high risk persistent gestational trophoblastic disease in patients with twin gestation and complete mole pregnancy.²

Case Report

A 30 year old lady gravida 3, para 1, living 1, abortion 1 married since 7 years with 28 weeks of pregnancy following intrauterine insemination presented at our antenatal clinic with complaint of one episode of spotting per vaginum without associated abdominal pain. Patient gave history of a similar episode of spotting per vaginum in first trimester for which she was investigated and diagnosed with subchorionic haemorrhage. Patient had responded to conservative management during that episode.

Her first pregnancy was a spontaneous abortion at 2 months of pregnancy. The pregnancy was also conceived following intrauterine insemination. Her second pregnancy was again conceived following intrauterine insemination. The pregnancy continued to term and elective caesarean delivery was done in view of a breech presentation. The current pregnancy was her third pregnancy.

On examination her general condition was fair. She was afebrile with vital parameters normal. Per abdomen examination revealed uterus corresponding to 30 weeks of gestation with the foetus in longitudinal lie and cephalic presentation. The foetal heart sound was localised on Doppler. The uterus was relaxed. On per vaginal examination, the external os was patulous and the internal os was closed.

Her haemoglobin was 11.6 gm%, complete blood count 12200/cmm, platelets adequate, urine routine normal, fasting blood sugar 89 gm%, TSH 1.3 U/L and her HIV, HBsAg and VDRL......
nonreactive. Coagulation parameters (PT, PTT, serum fibrinogen) were within normal limits. Serum beta hCG was 31,400 U/L. Ultrasonography revealed twin gestation with one sac filled with multiple cystic structures and a second sac showing a live intrauterine foetus corresponding to 21 weeks of gestation with no obvious congenital anomaly (Fig. 1). A diagnosis of twin gestation with one molar pregnancy and other normal live foetus was made.

After counselling and consultation with the patient and her husband, it was decided to continue the pregnancy. Her antenatal checkup was undertaken every week with clinical monitoring of foetal and maternal well being. Ultrasonography was repeated every two week. Follow up scans showed normally growing live foetus along with sac filled with vesicular moles. There was no repeat episode of antepartum haemorrhage. At 32 weeks of gestation, coagulation profile revealed raised serum fibrinogen concentration (569 mg%). Other coagulation parameters (PT, PTT, platelet count, D-dimer) were within normal limits. Haematologist’s opinion was taken and diagnosis of pregnancy-induced hyperfibrinogenaemia was made.

Patient underwent an elective caesarean delivery at 39.1 weeks of gestation giving birth to a female foetus weighing 2.5 kg. Second sac was filled with vesicular mole (Fig. 2). The neonate was morphologically normal and had a normal karyotype.

Both placenta and vesicular mole were sent for histopathology examination. Molar tissue weighed 350 gm and microscopic diagnosis of a complete mole (Fig. 3) was made as there was no evidence of umbilical cord, membranes or foetus. Post partum beta hCG was 38,000 U/L. The beta hCG was repeated at weekly intervals and, seven weeks following delivery, serum beta hCG had reached non-pregnant levels and a chest X-rays (PA and lateral view) were normal.

Discussion

Gestational trophoblastic disease is a spectrum of interrelated conditions including complete and partial hydatidiform mole, placental site...
trophoblastic tumour and choriocarcinoma. Complete hydatidiform moles have a 46 XX karyotype with both sets of chromosomes derived from the male gametes. Partial mole pregnancies are triploid and extra set of paternal chromosomes. Complete moles are usually diagnosed earlier in pregnancy. The incidence of complete and partial mole was found to be 1 per 1,945 and 1 per 695 pregnancies respectively. It is important to differentiate between a partial mole with viable pregnancy from a twin gestation with one molar pregnancy and other normal foetus in view of different foetal prognosis (increased risk of foetal abnormalities in partial mole) and risk of persistent gestational trophoblastic disease (increased in complete vesicular mole) in each condition.

Stellar et al have described 8 cases of twins with molar pregnancy. They found five out of eight patients developed persistent gestational trophoblastic neoplasia.

However, a recent study in U.K. showed no increase in risk of persistent gestational trophoblastic neoplasia in a twin gestation with vesicular mole compared to a singleton complete vesicular mole.

Successful outcome of twin gestation with vesicular mole and foetus have been reported in studies. In view of the complications associated with a complete vesicular mole (PIH, embolization, antepartum haemorrhage, torsion of theca lutein cysts, thyrotoxicosis and risk of persistent and metastatic disease), continuation of pregnancy should be only considered after extensive counselling of the patient. Careful monitoring of the pregnancy, maternal parameters, coagulation profile and TSH has to be undertaken. Delivery has to be conducted in a tertiary centre in view of risk of embolisation and postpartum haemorrhage. Monitoring of beta hCG levels till normalisation is necessary. Chemotherapy is not warranted except in the presence of factors suggesting an increased risk of persistence or recurrence of gestational trophoblastic disease.

References
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