HYDATIFORM MOLE WITH A COEXISTENT FOETUS

by

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Hydatiform mole (HM) may either be partial or commonly complete. Partial HM is not incompatible with birth of a viable child and this depends on the degree of involvement of the placenta. Incidence of HM with a coexistent foetus has been reported to be 1 in 10,000 (Bowles, 1943) to I in 200,000 pregnancies (Beischer, 1966). It presents in two forms; (a) a single molar placenta or (b) a dizygotic twin in which the placenta of the viable foetus is normal but other undergoes molar degeneration. In partial HM with single molar placenta the foetus often shows multiple congenital anomalies (Beischer, 1966). Subsequent choriocarcinoma is highly unlikely in the presence of coexistent foetus with HM (Park, 1971).

On account of its rarity, we present here 2 cases of single molar placenta with coexistent foetus.

CASE REPORTS

Case 1

I.P., a 19 year old primigravida was seen at the Nehru Hospital of Postgraduate Institute of Medical Education and Research, Chandigarh (PGIMER) on 27 February, 1979 with 32 weeks of amenorrhoea (LMP 14 July, 1978), severe pre-eclamptic toxaemia (PET) with onset at 28 weeks and suspected molar pregnancy. She was in early labour. She had felt foetal movements from 20 weeks to 28 weeks, and had hyperemesis till 26 weeks of pregnancy.

On examination, she had edema, her B.P. was 160/110 mm of Hg. She was on 8 hourly Aldomet and Adelphane. Fundus showed grade III hypertensive retinopathy. Uterus was of 32 weeks size, foetal parts were not made out on palpation and foetal movements were absent. Foetal skeleton was not visualised on plain X-ray of abdomen.

Investigations revealed a Haemoglobin of 11.6 g/dl., urine albumin ++, blood urea 37 mg/dl, uric acid 7.5 mg/dl, creatinine 1 mg/dl, Liver function tests and blood sugar were normal.

She delivered spontaneously a macerated stillborn male of 550 g. with foetal ascites, syndactaly and sacrococcygeal sinus. Three fourths of placenta (weight 350 gm.) and grape-like vesicles. Histological examination gave a diagnosis of molar placenta. She was discharged on methyldopa and propranolol for her hypertension. She was followed at regular intervals for a period of 5 months and was well with her hypertension under control. Serum human chorionic gonadotrophin was negative on three occasions.

Case 2

S.K., a 30 year old female, 5th gravida (P 4+0+0+4), was admitted on March 26, 1979. She was ammenorrhic for 35 weeks and presented with severe vaginal bleeding. She had tremors of hands, palpitations and warm

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and moist skin. There was pedal edema and the blood pressure was 150/100 mm. of Hg.

On examination of abdomen the uterus was of 35 weeks size but foetal heart sounds were not heard. There was evidence of pulmonary edema.

Investigations showed a haemoglobin of 12.8 g./dl., urine albumin ++, urea 20 mg/dl, creatinine 1.3 mg/dl, uric acid 6 mg/dl and serum proteins were 4.3 g/dl with a albumin of 2.4 g/dl. The platelet count was 30,000/mm and prothrombin index was 54 per cent. One hour after artificial rupture of membranes, she delivered a fresh still born foetus (Fig. 1) smeared with meconium and weighed 2.5 kg. A complete autopsy revealed no congenital malformation. Placenta weighed 1250 gm and the cord length was 30 cm. Microscopic examination confirmed the diagnosis of molar placenta. Pulmonary edema disappeared after the evacuation of the placenta. Her blood pressure came down to normal. The patient was followed up for 12 weeks and was symptomless and serum human chorionic gonadotrophin was negative on two occasions.

Discussion

On the basis of histopathological examination and chromosomal studies HM have been classified as partial or complete (Vassilakos *et al* 1977). Recently complete HM have been shown to be androgenic in origin (Kajii and Okama, 1977; Jacobs *et al* 1978). The reasons for a very high propensity of complete HM to develop choriocarcinoma and almost a negligible risk of malignancy in partial HM remain unknown. The presence of foetal movements helps to differentiate partial from complete HM on clinical examination. Toxaemia and hypertension are often associated with this entity. Antenatal diagnosis of partial HM is difficult but pre-eclamptic toxaemia (PET) before 24 weeks of pregnancy, signs of severe PET, excessive vomiting, disparity in uterus size and gestational age and vaginal bleeding at any time of pregnancy with a coexistant foetus should arouse suspicion of molar change of placenta (Jones and Lauersen, 1975). Modern techniques of transabdominal injection of contrast media (Torres and Pelegrina, 1966) and ultrasonic scanning (Donald, 1968) can identify the condition with reasonable accuracy.

There is an increase of thyroid function with or without clinical hyperthyroidism in cases of HM and choriocarcinoma. Pulmonary edema may follow as seen in one of our cases (Herschman and Higgins, 1971). The thyroid functions return to normal after evacuation of HM. A thyroid stimulator, which differs from long acting thyroid stimulator, has been isolated from the molar tissue and has been named molar thyroid stimulating factor (Herschman *et al* 1970). Unfortunately, the thyroid functions could not be done in our cases. Though cases of partial HM presenting as antepartum haemorrhage due to placenta previa have been reported (Beischer, 1966; Kachroo and Khanum, 1971), a case like our case 2 with accidental haemorrhage accounting for still birth has not been reported so far.

The treatment of HM with a coexistent foetus is immediate termination of pregnancy. Hysterotomy is rarely necessary. Adjuvant chemotherapy is not recommended since these cases of partial HM with coexistent foetus have a negligible chance of malignant transformation.

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