COMPLETE HYDATIDIFORM MOLE COEXISTING WITH A LIVE FETUS

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ABSTRACT

Hydatidiform mole co-existing with a normal fetus is very rare. We report a case of a 36 year old woman Para 4+0 who presented with amenorrhoea of twenty four weeks duration, vaginal bleeding , abdominal pain and pre-eclampsia. Ultrasound examination revealed a hydatidiform mole coexisting with a normal living fetus. The patient underwent a caesarean section at twenty eight weeks for maternal distress due to unbearable abdominal pain. The baby died after seven days. Post operatively she had an eclamptic fit and developed oliguria and persistent trophoblastic disease which were all successfully treated.

Key words: hydatidiform mole, living fetus, perisitent trophoblastic disease.

INTRODUCTION

Hydatidiform mole is the mildest of a spectrum of placental tumours known as gestational trophoblatic diseases. They are common in South East Asia , the older woman [1] and may result from in vitro fertilization [2] or ovulation induction [3] .There are two types the complete and the incomplete .The former has 46 chromosomes all of paternal origin while the latter is usually triploid with 69 chromosomes 23 of them being of paternal origin [4] .The placenta is completely replaced by tumour in the complete mole and no fetal parts are seen. In the incomplete or partial mole, placental distortion is partial and the fetus is present but may be abnormal [5] and often dies in the first trimester[6] .

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CASE REPORT

Mrs O.O a 36 year old Gravida 5, Para 4 +0 with three children alive presented in the outpatient clinic of Imo State University Teaching Hospital on 23/6/11.Her last menstrual period was 28/01/10 (Gestation 20weeks+6days).Her pregnancy had been uneventful until seven days before presentation when she started bleeding per vagina. This was accompanied by lower abdominal pain .The pregnancy was conceived naturally.

On examination she was pale, afebrile, not jaundiced or dehydrated and had no oedema or lymphadenopathy. Her chest was clinically clear. Her pulse rate was 104 beats per minute and her BP was 170/100 mm Hg. In the abdomen there was no organomegaly and the uterine fundus was consistent with 24 weeks gestation. Other systems were essentially normal. A speculum examination of the cervix showed a healthy closed cervix with blood oozing out of the cervical os. Investigations showed a packed cell volume of 22 percent while urinalysis showed protein 500mg /dl. Ultrasound revealed a normal viable male fetus with no obvious abnormalities, in longitudinal lie at gestational age of 25weeks 6days. Liquor volume was adequate. A normal postero-fundal placenta was seen with a coexisting intrauterine mass with numerous vesicles giving a snow storm appearance Fig 1. A diagnosis of viable intrauterine pregnancy coexisting with a hydatidiform mole with superimposed pre-eclampsia and anaemia was made. Serum beta-HCG was 217.5 mIU/litre (Normal= 2-50), and chest X-ray was normal. A conservative approach was adopted after counseling the patient. The patient was treated with analgesics for pain, haematinics and had multiple blood transfusions to correct the anaemia. Alpha methyldopa was given for the pre-eclampsia and corticosteroids were given for lung maturation. Bleeding and pain continued intermittently. Serial detailed ultrasound scans of the fetus revealed no fetal abnormalities. On 26/7/11 the pain became very severe and could no longer be controlled by analgesics. A presumptive diagnosis of abruptio placenta was made. An hysterotomy was done on account of maternal distress. A normal placenta (Fig 2) which weighed 280gms and was 17cm at its largest diameter was seen. The molar vesicles filled about three quarters of the uterus and weighed 210gms .The estimated blood loss was 1000mls.The baby weighed 0.75 kg with Apgar scores of 7,and 4 at one and five minutes respectively . It was macroscopically normal but died in the special care nursery on the seventh day. Post mortem was declined. The histology revealed hydropic villi with extensive stromal oedema some of which had central cisterns. Though minimal amount of proliferative trophoblast was seen in the sampled area, no normal villus or fetal membrane was seen. Overall features were those of complete hydatidiform mole. Post partum the patient developed oliguria with a urinary output of 100mls on the first day .Over the next twenty four hours it rose to 2500mls . She also had an eclamptic fit as blood pressure rose to 220/120 mm of Hg. This was treated with magnesium sulphate , hydrallazine and alpha methyldopa. She also developed blurring of vision secondary to exudative retinal detachment. The patient subsequently did well and HCG level was normal <5mIU/litre at the time of discharge on 22/8/11.The patient was advised to come for follow up but defaulted .On the twenty -seventh day post op she presented again with a history of losing fresh blood per vagina. A diagnosis of persistent trophobalstic disease was made. Chest X-Ray was normal while serum HCG level was 547IU/litre (Normal <5IU /litre) .She was successfully treated with a single eight day course of chemotherapy using methotrexate and folinic acid on alternate days and was discharged to follow up on 17/9/11. She has remained free of the disease but has been unable to keep her follow up appointments.

DISCUSSION

Hydatidiform mole coexisting with a viable fetus is a very rare complication and an incidence of 1 :10,000-1:100,000 has been quoted [7] .There are two possibilities in such situations . It could either be an incomplete mole with a viable fetus or a twin pregnancy in which one of the fetuses has undergone molar degeneration. Presentation may be varied and includes vaginal bleeding and abdominal pain as in this patient. It may also be suspected when pre-eclampsia develops very early [8], when the uterus is larger than dates as was the case here, in hyperemesis or when pregnancy is associated with hyperthyroidism. High HCG levels seen during screening for Down's syndrome may also lead to its diagnosis [9,10]. Diagnosis may be made by ultrasound as in this patient but it should be noted that missed abortion and degenerating fibroid may give a similar picture on ultrasound and should be excluded [11].The major dilemmas in the management of this condition are whether the fetus is normal, whether there are serious maternal complications and the risk of developing persistent trophoblastic disease. Although severe maternal problems were present in this case, it was felt that they could be controlled until the fetus reached the age of viability while the normality of the fetus was partially confirmed by ultrasound. Hydatidiform moles when

associated with a viable fetus are more likely to cause persistent trophoblastic disease [12,13] than ordinary molar pregnancies. Also complete mole is more likely than incomplete mole to cause persistent trophoblastic disease [14] and the level of HCG has a role to play in determining the risk of developing persistent trophoblastic disease [15]. Determination of whether a mole is complete or partial mole is therefore desirable .This may not always be possible by ultrasound but may be done by cytogenetic tests on chorionic villi sample ,amniocentesis or fetal blood sampling [14]

.These facilities were not available to us. For cases that present before the age of fetal viability the problem is to decide whether to terminate immediately or manage conservatively until there is a reasonable chance of fetal survival. Some authors [16,17] favour immediate termination of the pregnancy while others favour continuation of the pregnancy in the absence of fetal abnormality and uncontrollable pre-eclampsia [18,19]. The latter argue that continuation of the pregnancy is not associated with an increase in incidence of persistent trophoblastic disease[14,18-20] which has been reported even when immediate termination was done [21,22]. In a study[18] the incidence of persistent trophoblatic disease was more (19.2%) in those in which termination of pregnancy was immediate than in those in which it was not done(9.1%).In this patient a conservative approach was adopted until severe maternal complications forced a caesarean section.

This case highlights some of the challenges faced in the management of such cases in the developing countries. The inability to salvage babies of low birth weight in most developing countries would mean prolonging such pregnancies to near term to guarantee survival. The baby who weighed 750gms would probably have survived in a developed country. Secondly poverty continues to make the delivery of optimal services impossible. The patient could not afford the cost of medications or tests. Consequently only three HCG assays were done in the management of this patient and follow-up was unsatisfactory because the patient did not have the resources for multiple hospital visits. Fortunately a home visit confirmed that the patient remains well to date.

In conclusion, as there is no clear-cut evidence that prolonging a pregnancy is associated with increased risk of persistent trophoblastic disease, provided the baby is normal and there is no life threatening maternal complication, a conservative approach is recommended in order to get the baby to a gestation where fetal survival is certain.

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