INTRODUCTION:

Partial molar pregnancy with live foetus is exceedingly rare, and is frequently associated with significant morbidity. It is a benign form of Gestational Trophoblastic Disease, in which a normal haploid ovum is fertilised by two sperm or a diploid sperm, resulting in a triploid chromosome set. The incidence of coexisting live foetus in partial molar pregnancy is 0.005% to 0.01%. We describe a case of a partial mole singleton foetus, diagnosed in the second trimester, complicated by severe early onset preeclampsia.

CASE REPORT:

A 30-year-old G2P1 presenting for her routine antenatal appointment at 16 weeks gestation with small APH was found to be hypertensive and have severe nephrotic range proteinuria. Her early pregnancy period was uncomplicated, with no abnormalities present in her first trimester ultrasound or routine investigations. The patient had experienced a recent upper respiratory tract infection, but was otherwise healthy with no significant medical history. Her first pregnancy resulted in emergency caesarean section for foetal distress. Our patient commenced hypertensive therapy, and further assessment indicated early onset severe preeclampsia, requiring admission to hospital. In hospital, the patient experienced ongoing mild hypertension despite treatment, headache, nausea and vomiting, and both facial and lower limb oedema. Additional input from Nephrology was sought, and a renal ultrasound was performed to investigate potential causes of renal origin. The ultrasound detected live intrauterine foetus at 16 weeks but a markedly thickened cystic placenta suggesting a partial mole.

Arrangements were made for transfer to a tertiary obstetrics service for renal monitoring and continuation of pregnancy, however an acute decrease in foetal heart rate was noted, resulting in imminent foetal demise. The patient was commenced on Misoprostol to induce miscarriage at 16 + 3 weeks. The patient delivered a non-viable male foetus, and a grossly abnormal placenta, with severe hydropic change consisting of chorionic fronds and vesicles (as pictured).

Figure 1. Photograph of the placenta

Subsequent histopathological assessment of placental tissue confirmed a partial mole and abnormal molecular karyotype of the foetus was triploidy 69 XXY. The patient recovered well and her subsequent pregnancy was uncomplicated, with no abnormalities present in her first trimester ultrasound or routine investigations. The patient had experienced a recent upper respiratory tract infection, but was otherwise healthy with no significant medical history. Her first pregnancy resulted in emergency caesarean section for foetal distress. Our patient commenced hypertensive therapy, and further assessment indicated early onset severe preeclampsia, requiring admission to hospital. In hospital, the patient experienced ongoing mild hypertension despite treatment, headache, nausea and vomiting, and both facial and lower limb oedema. Additional input from Nephrology was sought, and a renal ultrasound was performed to investigate potential causes of renal origin. The ultrasound detected live intrauterine foetus at 16 weeks but a markedly thickened cystic placenta suggesting a partial mole.

DISCUSSION:

As a rare and poorly understood condition, partial molar pregnancy with coexisting live foetus remains a complex clinical diagnostic and management challenge. In the case reported, the twelve week ultrasound was normal, which presents a unique diagnostic dilemma; as placental changes were not evident until the second trimester when the patient presented with complications, management was subsequently delayed. Furthermore in a partial mole, the serum beta-hCG level may be lower compared to in complete mole; as a result, the clinical presentation of a partial molar pregnancy may demonstrate less features associated with beta-hCG stimulation, including pre-eclampsia. This has implications on diagnosis and management. There are few documented cases of preeclampsia in hydatidiform moles, with previous analysis noting an incidence of 3.5%. This case is particularly interesting due to the unexpected early onset of severe pre eclampsia at 16 weeks gestation and first trimester absent of features suggestive of hydatidiform pregnancy.

REFERENCES: