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Case Report

Placentomegaly in Pregnancy with Primary Postpartum Haemorrhage leading to Caesarean Hysterectomy: A Case Report of a Prevented Feto -Maternal Death in a Tertiary Hospital in Akure

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Abstract
Placentomegaly is an important ultrasound finding in pregnancy. It may be asymptomatic or sometimes cause life threatening complications than a normal sized placenta and could also serve as a clue for poor feto-maternal outcome. We report a 29-year-old G2P1+0 (1 Alive) now P2+0 (2 Alive) with one previous caesarean section and an ultrasound diagnosis of placentomegaly at a gestational age of 35weeks+4days who had a repeat caesarean section at a gestational age of 35weeks+ 5days on account of reduced fetal movements. She was delivered of a live male 2.6 kg neonate with APGAR score of 8 at one minute and 9 at 5 minutes of life and placenta weight of 1.6 kg. Mother had primary postpartum haemorrhage (PPH) secondary to uterine atony. She subsequently had a caesarean hysterectomy due to protracted bleeding and failure of all other interventions to stop the bleeding with a high index of suspicion of a gestational trophoblastic disease especially an invasive mole as the cause of the bleeding. The uterine and placenta tissue were sent for histology which confirmed invasive(destructive) mole (chorioadenoma destruens). Maternal and perinatal mortalities were prevented following prompt intervention and a high index of suspicion.

Keywords: *Placentomegaly, Primary PPH, Caesarean Hysterectomy, Successful Feto-Maternal Outcome.*

INTRODUCTION:
Placentomegaly is an enlarged placenta which means it's thicker or weighs more than normal. It can be caused by conditions like maternal anaemia, infections, hydrops fetalis (Rhesus iso-immunization), macrosomia, or multiple gestation [1]. While sometimes, it's a sign of a highrisk pregnancy, in many cases it is simply an oversized placenta that may not cause more complication than a normal one. It can also be an indicator of more serious complications like partial molar pregnancy, invasive mole (Chorioadenoma destruens) and placenta site trophoblastic tumour (PSTT) [2]. The destructive type (invasive mole) penetrates the uterine wall and can lead to uterine rupture and haemorrhage. The fetal complications include intra-uterine growth restriction (IUGR) [1] and fetal demise.

The placenta weighs about 500-600g which is about $1/5^{th}$ -$1/6^{th}$ of a normal fetus that weighs between 2500 -3500g at term [3, 4]. When the placenta is abnormally large such as in maternal diabetes mellitus, it is combined with newly formed vessels and increased surface area as a compensatory mechanism [5]. In PSTT and invasive mole, it can result in uncontrollable PPH due to uterine atony [2]. The treatment of primary PPH can be medical or surgical interventions. The medical management include the use of oxytocics such as oxytocin, ergometrine or heat stable carbetocin for the prevention of PPH and the additional use of tranexamic acid if PPH develops [6]. While the surgical intervention includes the use of uterine ballon tamponade, B Lynch brace sutures, uterine artery ligation and hysterectomy [6].

CASE REPORT:

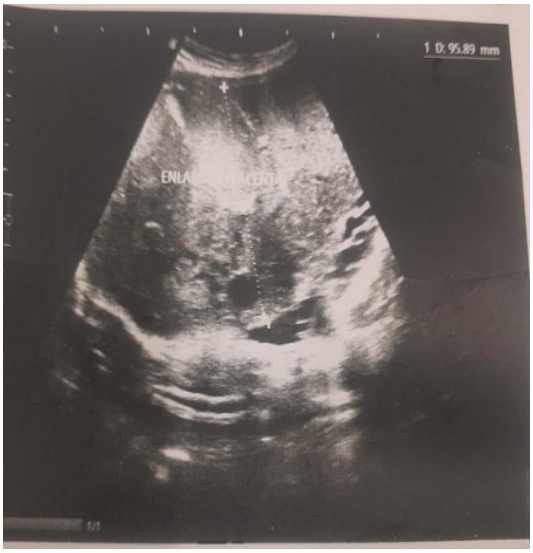
This was a case of a booked 29- year- old G2P1(1 Alive) with 1 previous Caesarean section who was unsure of her last menstrual period (LMP). She was admitted from the antenatal clinic (ANC) on the 8th of September 2025 with an ultrasound scan done outside our facility which revealed a singleton live fetus with placentomegaly at 35weeks and 4 days.

The estimated fetal weight (EFW) was 2.9kg. The placenta was anteriorly sited and enlarged in size measuring 9.59cm (Normal < 4cm) as shown in Figure 1. Her blood group was AB rhesus D +ve, retroviral screening (RVS), hepatitis B surface antigen (HBsAg), and HCV results were non-reactive. Urinalysis was negative for glucose and protein, her genotype was AA and her packed cell volume was 39%. A repeat ultrasound scan was done in the Antenatal ward which also revealed a single viable fetus at 35weeks+ 2 days. The EFW was 2.6kg, amniotic fluid index (AFI) was 20.9 cm. There was a large placenta with multiple areas of calcifications as shown in Figure 2. On the basis of the placentomegaly which was confirmed from the second opinion ultrasound scan; Fasting blood sugar and 2hours post-pandrial was ordered and results were 3.3mmol/L and 4.7mmol/L respectively (Normal). Serum B human chorionic gonadotrophin (B-HCG) was also ordered and the result was normal. The patient could not afford the cost of human placenta lactogen (HPL) because it was expensive. The patient was subsequently counselled on elective Caesarean section (ELCS) which she consented. Two units of blood was then grouped and cross-matched for her.

On examination, her general clinical condition was satisfactory. She was not pale, her pulse rate was 82 bpm and her blood pressure was 100/80mmHg. On abdominal examination, her symphysio-fundal height was 35cm, non-tender, fetal heart was heard and regular. She was compliant with her routine antenatal drugs and was monitored closely on the ward.

She however complained of reduced perception of maternal fetal movements the next day and she had emergency caesarean section (EMCS) on the 9th of September 2025 with the delivery of a live male 2.6kg neonate with APGAR score of 8 at one minute and 8 at 5 minutes of life. Other intra-operative findings included a massive placenta which weighed 1.6kg which was more than half of the baby as shown in Figure 3. There was no intra-operative(partum) bleeding as haemostasis was secured as seen in Fig 4 after uterine closure and intravenous heat stable carbetocin 100ucg was instantly administered after delivery of the baby. However, there was torrential bleeding from the external cervical os during vulva toileting and 1g of intravenous tranexamic acid was given immediately and intravenous oxytocin 40units in 500mls of normal saline was put up. Also, 800microgram of misoprostol was inserted into the rectum. The bleeding however persisted and intravenous tranexamic acid 500mg was repeated after 30minutes but to no avail. Then, condom catheter was attempted but the torrential bleeding flushed it out of the endometrial cavity as seen in Fig 5. The estimated blood loss (EBL) was 1.5L. At this stage we quickly requested for more units of fresh whole blood and one unit of fresh frozen plasma (FFP). Two consultant Obstetricians quickly resorted to re-laparotomy and bimanual uterine compression in preparation for B-Lynch suture but was abortive (Figure 5). The patient eventually had total Caesarean hysterectomy with bilateral salpingo-oophorectomy. The EBL for the second procedure was 1.5L and the total blood loss (TBL) was 3L. She had 6 units of blood and one FFP. She was counselled on intra-operative findings and the extent of the surgery.

The post-operative PCV was 26%. She was discharged home on haematinics on the 14th of September 2025 in stable condition. The uterus and its appendages and the placenta tissue were sent for histology and both revealed invasive(destructive) mole (chorioadenoma destruens). The patient was adequately counselled on the histology report and subsequently referred to the Oncologist for further expert management.



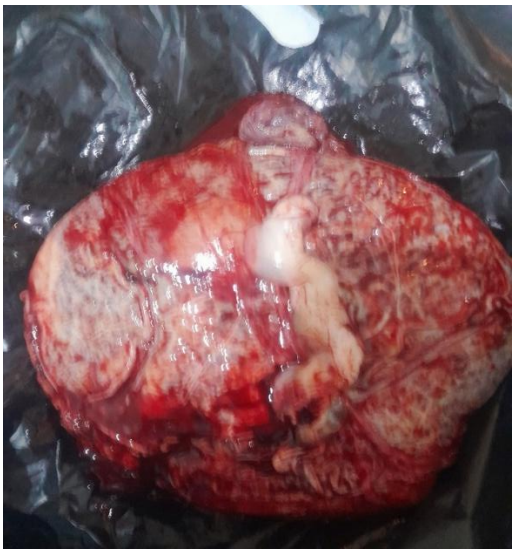


Fig 1

Fig 2

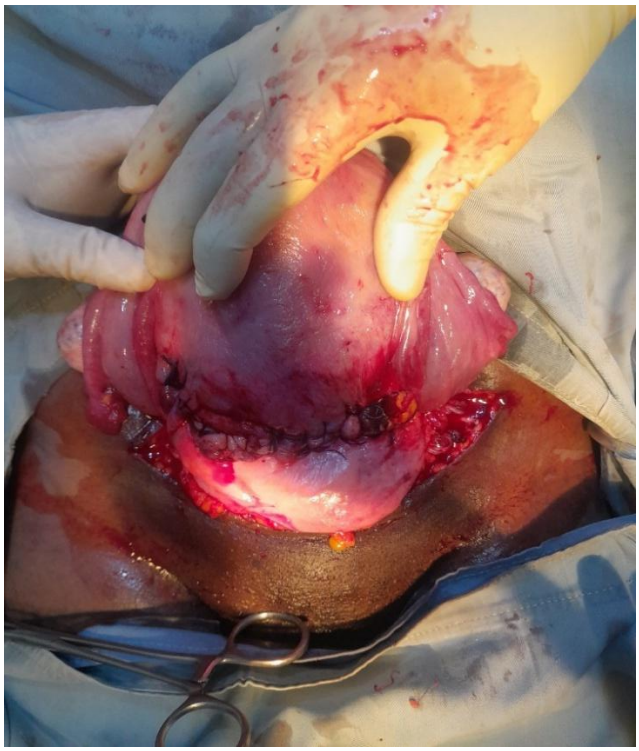


Fig 3

Fig 4

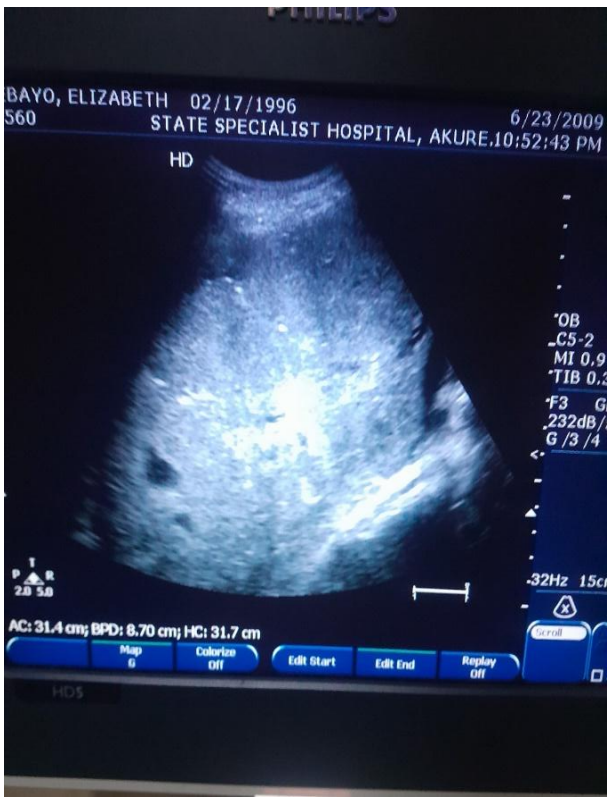


Fig 5

Fig 6

DISCUSSION:

We presented a booked 29-year-old G2P1 now P2+0 (2 Alive) woman who was managed for primary postpartum haemorrhage secondary to uterine atony which occurred following placentomegaly which was diagnosed in the antenatal period at a gestational age of 35 weeks. The antenatal and postpartum feto-maternal complications were anticipated and a repeat obstetric scan was done as depicted in the diagram (Fig 6). The fetus was compressed to one side by the large placenta with increased risk of fetal compromise/demise. There were also multiple areas of calcifications on the placenta. This could be a sign of pregnancy failure with a sign of imminent fetal loss in the absence of a timely intervention [7]. The patient was scheduled for elective Caesarean section and while on admission, she complained of reduced fetal movements the next day this was confirmatory and she had EMCS at 35weeks +5 days with the delivery of a normal birth weight neonate with good APGAR score.

The patient had primary PPH and finally had total Caesarean hysterectomy with bilateral salpingo-oophorectomy. This treatment is the gold standard for trophoblastic tumour and thereby limit the need for multiple courses of chemotherapy [8]. The decision was promptly taken by 2 consultant Obstetricians bearing in mind the risk of leaving a cervical stump behind which can cause persistent bleeding and even cancerous growth [9]. Although, this is more technically difficult in the face of massive obstetric haemorrhage because of the inherent complications such as ureteric injury and the rest. It was courageously and painstakingly done because of the profuse bleeding; we had trophoblastic tumors as the first differential on the list of our differential diagnosis and among this is PSTT which is relatively resistant to chemotherapy and produce human placental lactogen (HPL) [10]. The other differential diagnosis such as diabetes mellitus and rhesus iso-immunization were already ruled out from the investigations we ordered for. The histology report confirmed the diagnosis we had in mind which was invasive mole therefore the stress and the decision was worthwhile.

The decision to deliver this baby at a gestational age of 35 weeks which was close to term was timely and had contributed to the successful fetal outcome reported in this baby. Also, the maternal complications that we anticipated and the prompt intervention contributed to the successful maternal outcome.

CONCLUSION:

Placentomegaly may be an early sign of maternal complications before clinical symptoms or other adverse outcomes become apparent warranting enhanced prenatal, intrapartum and postpartum follow up of potentially affected pregnancies.

IMPLICATION OF THIS CASE REPORT

This condition is not just theoretical as seen in the texts. It is real and can occur in any environment or settings. Adequate preparations and management plan should be put in place to avert imminent feto-maternal complication. The most senior doctors or obstetricians should take charge rather than booking such for the junior colleagues to operate.

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