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Unveiling the Enigma: Chronic Concealed Abruption Disguised as Placentomegaly

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Abstract

Here we present a case of Rh-negative Primigravida with 22 weeks gestation who presented to us with USG report of a live fetus with FGR, placentomegaly with oligohydramnios without any clinical symptoms and later diagnosed to be a case of chronic abruption oligohydramnios sequence (CAOS). Initially, all differentials of Placentomegaly like placental chorioangioma, placental mosaicism, chronic concealed abruptio placenta or placental tumours such as placental mesenchymal dysplasia were considered. Regular antenatal workup was carried out for the patient which revealed an incidental heart disease probably of rheumatic origin and the patient was managed accordingly. The patient was planned for termination of pregnancy in view of subsequent anhydramios in the midst of which the patient developed cardiac complications. The patient was stabilized and taken up for emergency hysterotomy which intraoperatively revealed retroplacental clots and altered blood as evidence of premature separation of placenta clinching the diagnosis of CAOS. Since CAOS can lead to fetal morbidity and mortality, decision-making has to be carried out regarding accelerating fetal lung maturity and neuroprotection or termination of pregnancy after carefully weighing out the fetomaternal outcomes.

Keywords*: Gestation, Placenta, Placentomegaly, Pregnancy.*

Introduction

Placentomegaly is an increase in placental thickness more than 4 cm at any stage in pregnancy. The thickness could be homogenous or heterogenous depending on the cause of placentomegaly [1]. Differential diagnosis of placentomegaly include placental chorioangioma, placental mosaicism, chronic concealed abrouptio placenta or placental tumours such as placental mesenchymal dysplasia. Placentomegaly along with FGR is seen in maternal chronic hypoxia, placental mesenchymal dysplasia [2, 3]. We hereby report a case of Rh negative primigravida at 22 weeks of gestation with heart disease complicating pregnancy presenting with USG report of placentomegaly, oligohydramnios and FGR and later diagnosed to be a case of chronic concealed abruption. CAOS, or Chronic Abruption-Oligohydramnios Sequence, is a clinical condition characterized by ongoing vaginal bleeding and reduced levels of amniotic fluid. This reduction in amniotic fluid is attributed to a chronic form of placental abruption, where the placenta separates partially or completely from the uterine wall before delivery. The presence of chronic placental abruption in CAOS is believed to contribute to several adverse outcomes, notably preterm labor and the development of neonatal chronic lung disease [4].

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Case Report

A 26 year old RH negative Primi of 22 weeks gestational age, booked elsewhere class 2 obesity with a BMI of 37.9 came with USG report showing severe oligohydramnios and placental thickness of 5.3 cm indicating placentomegaly. Target scan could not identify anomalies in view of severe oligohydramnios. The patient had a history of bleeding PV at 15 weeks gestation and was conservatively managed with oral and injectable progesterone. Clinically per abdomen finding revealed uterus corresponds to 28 weeks (more than the period of amenorrhea), the uterus was found to be tense but not tender.

Since a target scan could not be done and to understand the placentomegaly, MRI Pelvis (fetal MRI) was done and showed a gravid uterus with single intrauterine gestation with fetus in a transverse lie and placental findings showed intra placental T1- focal hypointense, T2- focal heterogeneous hypointense-hyperintense area/ lesion in the anterior segment of the placenta (fundal, upper and mid body) (predominately hyperintensity) of size 35x125x 168 mm, volume - 367 cc, central few areas of hypointensities (? Normal placenta) with peripheral smooth margin (layering appearance) (STIR- Heterogenous hyperintense hypointense, DWI- no reduced diffusivity, ADC - high) suggesting Placentomegaly with suspicious neoplastic etiology in the anterior segment of the placenta. Intraplacental T1- Two focal hypointense, T2 focal hyperintense areas in the posterior segment of the placenta (upper and mid body) of largest size- 10x15 mm, volume - 367 cc, central homogenous, peripheral irregular margin (STIR- hyperintense, DWI- no reduced diffusivity, ADC - high)- suggesting likely placental lakes in the posterior segment of the placenta.

Baseline investigations were done which showed the patient had moderate anemia. Routine antenatal echocardiography done showed features of Thickened doming AML, Thickened Restricted PML, Mild Mitral stenosis, MVA/PHT– 2.5-2.6 cm2, Gradient Across Mitral Valve (PG –26mmHg, MG - 18mmHg), Severe subvalvular narrowing,Trivial aortic regurgitation. Cardiology opinion was obtained, and the patient was managed accordingly.

The following day USG scan was done and a severe reduction in liquor index showing anhydramnios with the presence of fetal cardiac activity. Hence patient was planned for termination of pregnancy given anhydramnios. Patients were given oral mifepristone following which patient developed leaking pervaginum - tobacco-coloured fluid noted. Patients started on antibiotic prophylaxis.

Meanwhile, the Patient developed breathlessness, bilateral crepitations and tachycardia suggestive of pulmonary edema-cardiology, pulmonology and medicine opinion was obtained and started on injectable Furosemide. The patient developed pain abdomen and per abdomen examination revealed a tense and tender uterus and bleeding PV noted. After stabilising the patient, due to unfavourable cervix and cardiac complications, Patient was taken up for emergency hysterotomy under GA. Intraoperatively, greenish tinged ascitic fluid was present and same sent for cytology which showed negative for malignancy. Upon uterine insicion, about 500ml of altered blood and retroplacental clots seen gushing out as evidence of premature placental separation. Fetus- no signs of life after delivery, 1 tight loop of cord around neck strangulating the baby noted. Baby appeared macerated and weighed 240 g, retroplacental clots present all around the edge of the placenta, premature placental separation noted, cord was blood stained. Placenta weighed to be 100 grams without blood clots.

Patients were transfused with 1-unit PRBC, 4 units of FFP. Postoperatively patient was stable and shifted to ICU for post op

monitoring. Patients started on heparin for DVT prophylaxis. Patients were given Anti D and cabergoline. Patient was extubated and provided with NIV support in view of bilateral crepitations. HRCT chest was done, and the findings were suggestive of acute respiratory distress syndrome and were managed accordingly with multidisciplinary approach. Patient improved symptomatically, weaned off NIV and was stepped down from ICU. Cardiology review was obtained and advised to continue Penicillin and diuretics. Patient progressed well and became symptomatically better. Fetal HPE showed no significant abnormalities.

HPE of placenta showed **Placenta – Retroplacental hematoma with intra placental extension, villous infarction and intervillous hemorrhage. Features being consistent with Abruption.**

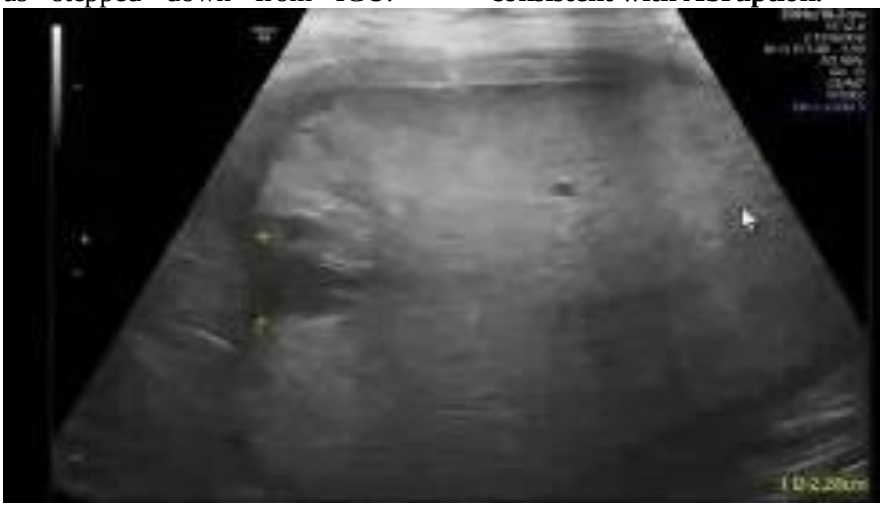

Figure 1. Placentomegaly of 5.2cms Thickness in USG

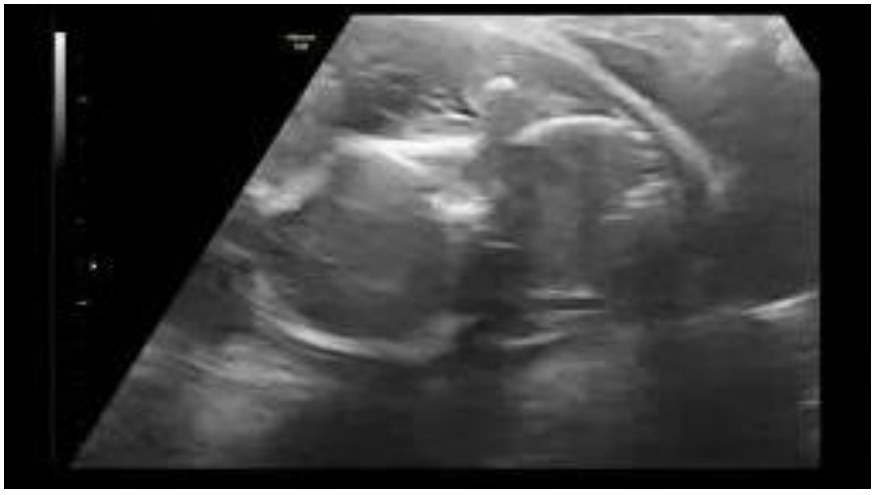

Figure 2. Fetus Crumpled up near the Lower Pole of Uterus with Severe Oligohydromnios

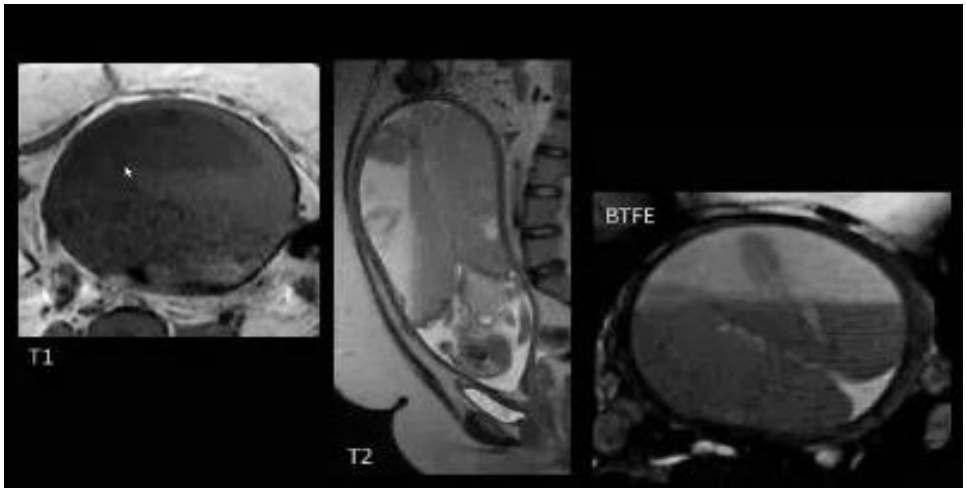

Figure 3. MRI - Axial and Sagital Views of Placenta

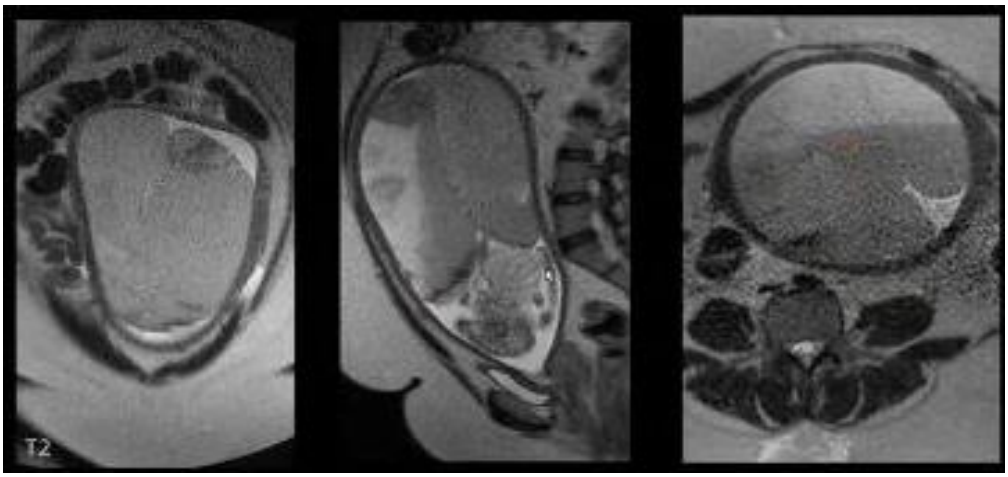

Figure 4. MRI showing Single Intrauterine Gestation with Crumpled Up Fetus in Transverse Lie with Central Opacities in Placenta

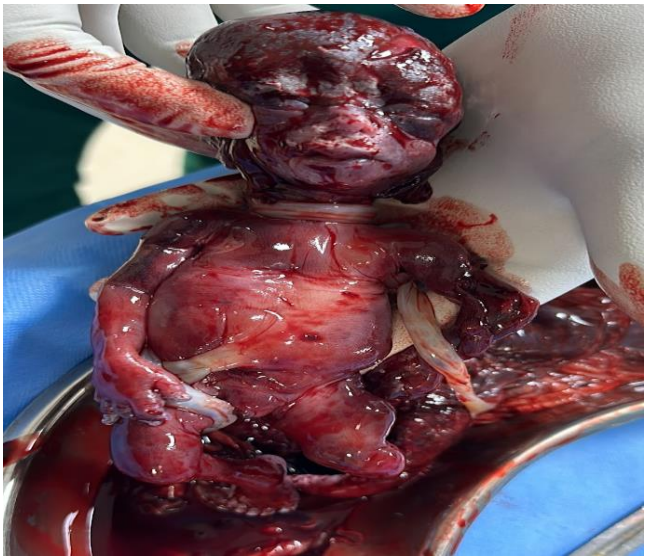

Figure 5. Macerated Fetus

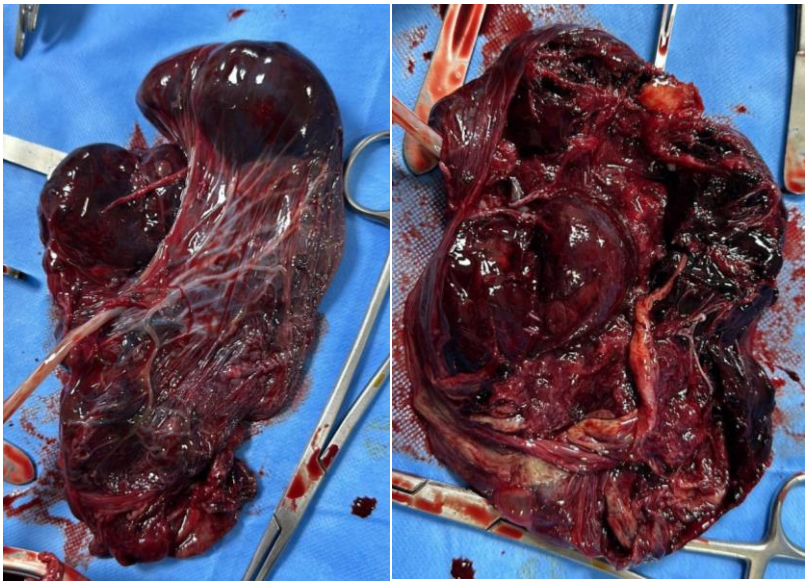

Figure 6. Placenta with Retroplacental Clots

Discussion

Abruption becomes apparent when vaginal bleeding is visible, otherwise, it remains concealed as the hemorrhage accumulates behind the placenta [5]. Concealed abruption has an incidence of 11%-20% [6]. Factors that lead to the above syndrome chronically include maternal vasculopathy, anemia, hypertension (chronic or preeclampsia), smoking, intake of drugs, nutritional deficiency, uterine malformations and tumors, supine hypotension syndrome, antiphospholipid syndrome, and thrombophilias [7]. Almost 18%-20% are present with no clinical symptoms, just like in the present case posing a diagnostic dilemma [5]. Ultrasound is the main modality of diagnosis in cases of concealed abruption as it does not present without any clinical symptoms. This case was a clinical enigma because of a large lesion in ultrasound, located beneath the placenta pushing the fetus to one side of the uterus, leading to a differential diagnosis of placental tumours in the form of placentomegaly. On USG, abruption can be missed or mistaken for other lesions [8]. Similar confusion arose in our case, prompting the need to consider the possibility of other lesions. One hypothesis addressing ambiguities suggests that retroplacental hemorrhages can be categorized into two main types: arterial and venous. According to this theory, arterial hemorrhage leads to acute placental abruption, while venous hemorrhage leads to marginal and chronic abruption. In the central two-thirds of the placenta, arterial blood flow and pressure are at their highest. Consequently, placental abruptions are more likely to occur centrally, resulting in indentation and rupture of the basal plate, often leading to rapid delivery and acute fetal decompensation [9,10]. Conversely, venous hemorrhages from distended veins tend to remain peripheral or marginal. These peripheral veins, previously referred to as the marginal sinus, have less support compared to central veins. Since venous pressure is lower than arterial pressure, venous hemorrhages are less destructive and may not prompt immediate delivery. Prolonged venous bleeding within the placenta contributes to the formation of this subchorionic hematoma, causing a separation between the amnion and the chorion from the decidua. Consequently, there is a reduction in blood and nutrient supply to the amnion and chorion. This can result in placental dysfunction, leading to suboptimal fetal renal perfusion, which may contribute to oligohydramnios [11]. This hypothesis helps explain the chronic nature

and less destructive characteristics of the abruption in our case. This condition poses significant clinical challenges due to its link with premature birth and potential lung damage in newborns [12].

The underlying mechanism behind CAOS lies in recurrent bleeding within the uterus, leading to the formation of subchorionic hematomas. In 75% of patients diagnosed with CAOS, an ultrasound typically reveals a subchorionic clot [11]. Hemorrhage occurring in the peripheral vein of the placenta rather than the arterial hemorrhage can lead to the development of CAOS. Another school of thought is that this peripheral venous hemorrhage triggers the presence of hemorrhagic amniotic fluid and DCH (decidual cast-off hemorrhage), which contribute to the degeneration and weakening of the amnion. Consequently, the primary pathology of CAOS is thought to be premature rupture of membranes rather than chronic abruption which could be seen in some cases [4].

Conclusion

Placentomegaly and CAOS can lead to diagnostic dilemmas and prove troublesome in managing the condition. As seen in our case, the radiological investigation may not be conclusive, and a strong clinical correlation is recommended. This case also highlights the importance of prompt recognition and multidisciplinary management of complex obstetric and medical conditions to optimize patient outcomes. Since many women may present late with IUGR/IUD due to the insidious onset of this condition, Patients should be educated to do regular antenatal checkups to reduce maternal and fetal morbidity and mortality.

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