

## Case Report

## Impending Stillbirth of an IUGR Fetus in a Preeclamptic Woman and the Role of Doppler Sonography: A Case Report

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Received: 20.07.2025

Accepted: 17.09.2025

Published: 23.09.2025

**Journal homepage:**<https://www.easpublisher.com>**Quick Response Code**

**Abstract:** Stillbirth is more common in low and middle-income countries like Tanzania. The risk of stillbirth is high in intrauterine growth-restricted fetuses. Placental insufficiency is associated with intrauterine growth restriction as well as the risk of both preeclampsia and stillbirth. We present a 36-year-old preeclamptic woman with sonographic features of placental insufficiency, intrauterine growth restriction, whose obstetric outcome was a stillbirth. The obstetric Doppler findings indicated severe placental insufficiency and absent end-diastolic flow in the middle cerebral artery, which we consider a sign of impending fetal death/stillbirth.

**Keywords:** Stillbirth, Preeclampsia, Intrauterine Growth Restriction [IUGR], Fetal Doppler, Placental Insufficiency, Pulsatility Index [PI], Resistance Index [RI], Cerebral Placental Ratio [CPR], Absent End-Diastolic Flow in the Middle Cerebral Artery.

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### INTRODUCTION

Stillbirth refers to a baby born at or beyond 28 weeks of gestation or if weight is > 1000 grams when gestation age is not known, with no sign of life. An estimated 2.6 million stillbirths are recorded worldwide yearly, out of which 98% of stillbirths occur in low and middle-income countries, of which over 67% occur in sub-Saharan Africa (SSA) and Southern Asia. The highest stillbirth rate [SBR] observed in East Africa is in Tanzania, which is noted to be 25.9 per 1000 live births (Blencowe *et al.*, 2016)

It is known that Preeclampsia is a risk for stillbirth; both stillbirth and preeclampsia are associated with several placental abnormalities/pathologies, which are also implicated in placental insufficiency (Gibbins *et al.*, 2016). Placental insufficiency is reflected as intrauterine growth restriction [IUGR] which is diagnosed sonographically when the estimated fetal weight (EFW) is less than the 3rd percentile of the normal fetal growth for a given gestation age or if the estimated fetal weight [EFW] and or abdominal circumference is less than the 10<sup>th</sup> percentile with deranged/abnormal Doppler (Salomon *et al.*, 2019). The

presence of abnormal obstetric Doppler indices reflects abnormalities in maternal-fetal hemodynamics. Integration of fetal Doppler studies provides an objective assessment of fetal hemodynamic circulation. It can be used clinically to identify women at risk for stillbirths and neonatal mortality (Choorakuttil *et al.*, 2023). Absent end-diastolic flow in the middle cerebral artery [MCA] has rarely been reported as an indicator of impending fetal death since most of the published case reports are of end-diastolic reversal of the middle cerebral artery. Here we report a case of impending stillbirth of an IUGR fetus in a pre-eclamptic woman and the role of obstetric Doppler studies in the prediction of impending stillbirth.

### CASE PRESENTATION

A 36-year-old female was brought to the Radiology department [Kahama Municipal Council Hospital] for an obstetric ultrasound examination to evaluate fetal viability. She is non-diabetic, G5P3L3 with a clinical GA of 33 weeks, and was admitted due to complaints of headache, dizziness, blurred vision, and progressive decrease of fundal height and fetal movements. No Per vaginal bleeding, Per vaginal

leakage, or abdominal pain. Her first antenatal visit was at 16 weeks at which Blood pressure was normal. She had 3 visits, of which the last visit was 1 month [before current admission]. The blood pressure was high [178/110mmHg, and she was given methyldopa 1000mg bid, and nifedipine 20mg once daily.

**On Examination:** Conscious, cooperative, not pale, not jaundiced, no lower limb edema, BP 189/97mmHg, fetal heart rate was not adequately heard using a fetal scope, so the client was sent for an obstetric ultrasound examination.

**LAB:** Urine dipstick was positive for protein, CBC was normal, HB-14.5g/dl, Group O Rh+ve  
 Serum creatinine 97.3µmol/L, UREA=2.4µmol/L, ASAT=122U/L, ALAT=89.9U/L

**Obstetric Ultrasound** examination revealed [done at 10:20 AM] an average ultrasound age [AUA] of 29weeks+2days [which is less than clinical GA, Gestational age [GA] by trans cerebellar diameter-33weeks+2days which was congruent with clinical GA indicating that the clinical GA was correct, estimated fetal weight[EFW]of [1225 +-179]g which was less than the 3<sup>rd</sup> centiles of the normal fetal growth curve indicative of IUGR[Figure 1], fetal heart rate [FHR] was

fluctuating between 86bpm to 104bpm but regular in the pattern.

Doppler examination revealed; a high flow resistance index (RI) and pulsatility (PI) above the 95<sup>th</sup> centiles in the uterine artery (figure 2A,&C and 3A, B&C) and above 90<sup>th</sup> centiles in umbilical arteries(figure 4A, B&C)[diastolic reversal], high flow resistance of middle cerebral artery[MCA](absent diastolic flow) (figure 5A, B&C), Based on the findings, a diagnosis of placental insufficiency, intrauterine growth restriction [IUGR] with impending intrauterine fetal death /stillbirth was made, and recommended urgent attention, for which a decision to induce labour with a balloon catheter was planned[contraction stress test was not done due to limited availability of cardiotocography machine.

**Treatment:** Included magnesium sulfate loading dose of 4g, maintenance dose of 5g every 4 hours for 24 hours, methyldopa 2g bid, and nifedipine 20mg bid, 10 hours after ultrasound examination labor had not started, BP was 156/94 mmHg, pulse rate 77bpm, and fetal heart rate was un recordable. She delivered seven hours later with the fetus's Apgar score of zero and with a baby weight of 1100g [Almost the same to the sonographically estimated fetal weight]. She had a postpartum hemorrhage due to a cervical tear, which was managed successfully.

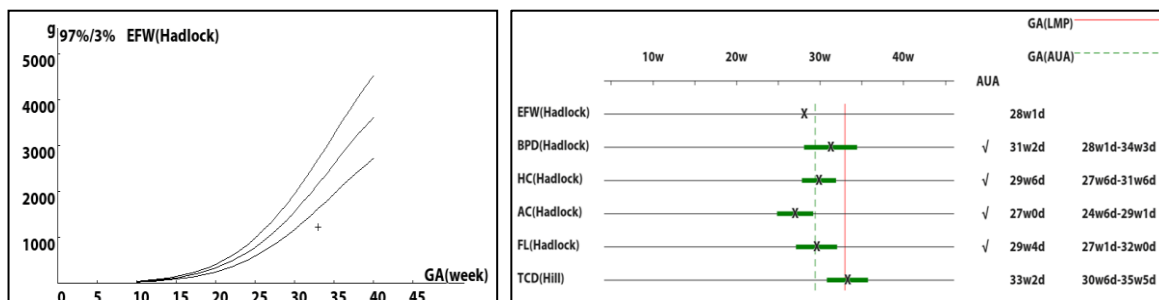


Figure 1: Fetal growth curve [left]Showing estimated fetal weight of less than 3<sup>rd</sup> percentile of the normal growth weight per gestation, fetal growth compare [right] showing GA by all fetal biometry and average GA being less than the clinical GA [red line], except the Trans cerebellar determined gestation age which is congruent o the clinical gestational age

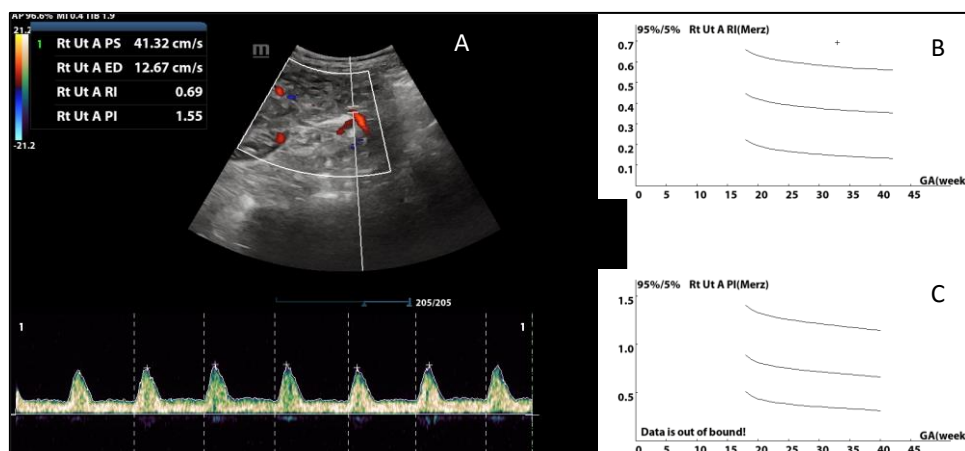


Figure 2: Right uterine artery color and spectral Doppler [A], showing early diastolic notching, [B], high uterine artery resistance index [RI-0.69] above 95<sup>th</sup> percentile and C]. very high pulsatility index [PI-1.55] above 95<sup>th</sup> percentile and beyond the limit of 1.5

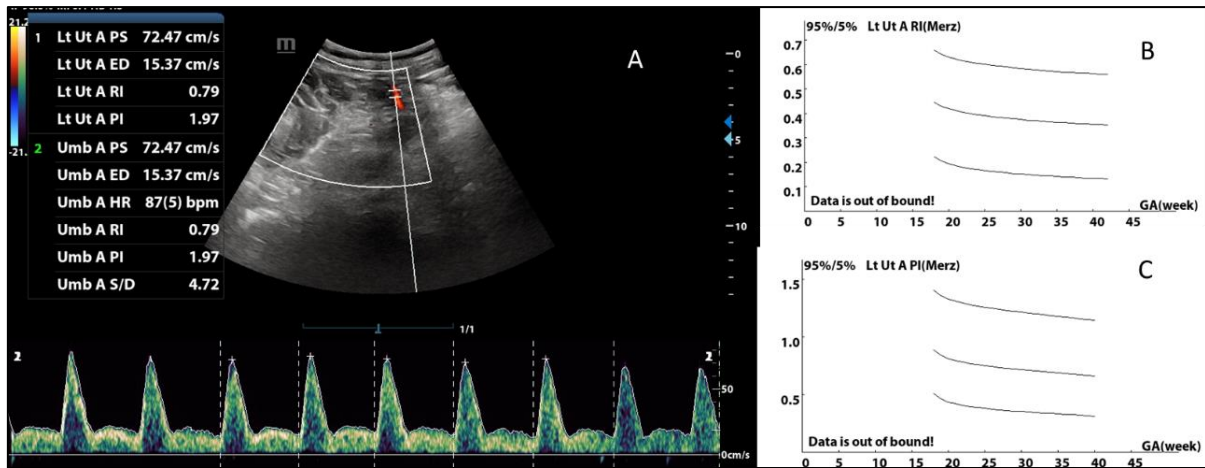


Figure 3: Left uterine artery color and spectral Doppler [A], showing early diastolic notching, [B]. very high resistance index [RI-0.79] above 95<sup>th</sup> percentile and beyond the limit of 0.79 and C) very high pulsatility index [PI-1.97] above 95<sup>th</sup> percentile per age and beyond the limit of 1.5

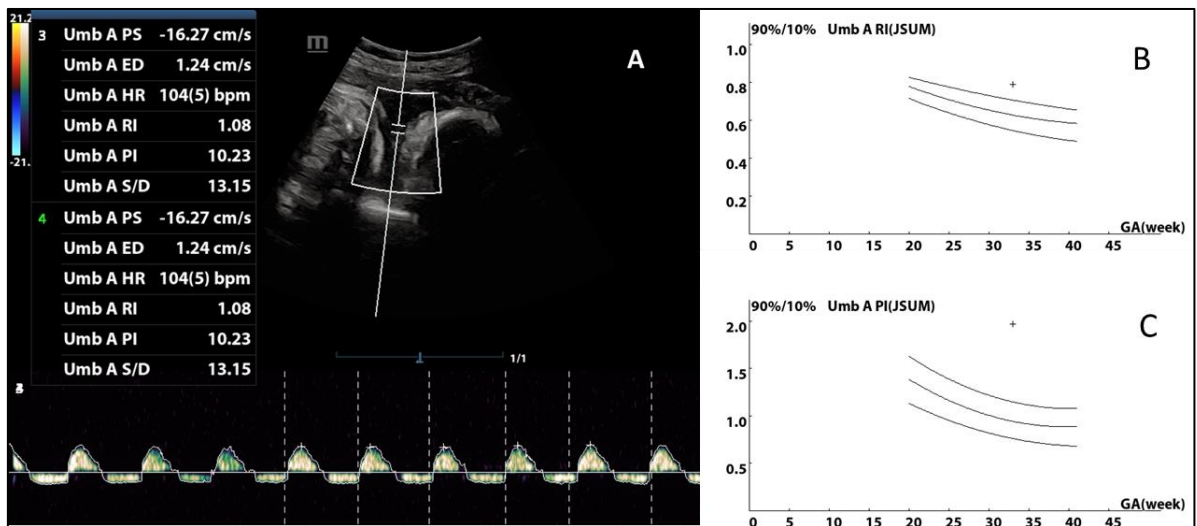


Figure 4: Umbilical artery color and spectral Doppler [A], showing diastolic reversal, [B]. very high resistance index [RI-0.108] above 90<sup>th</sup> percentile and beyond the limit of 0.1 and C). very high pulsatility index [PI-10.23] above 90<sup>th</sup> percentile per age and beyond the limit of 2.0 [almost five times high than the cut off limit]

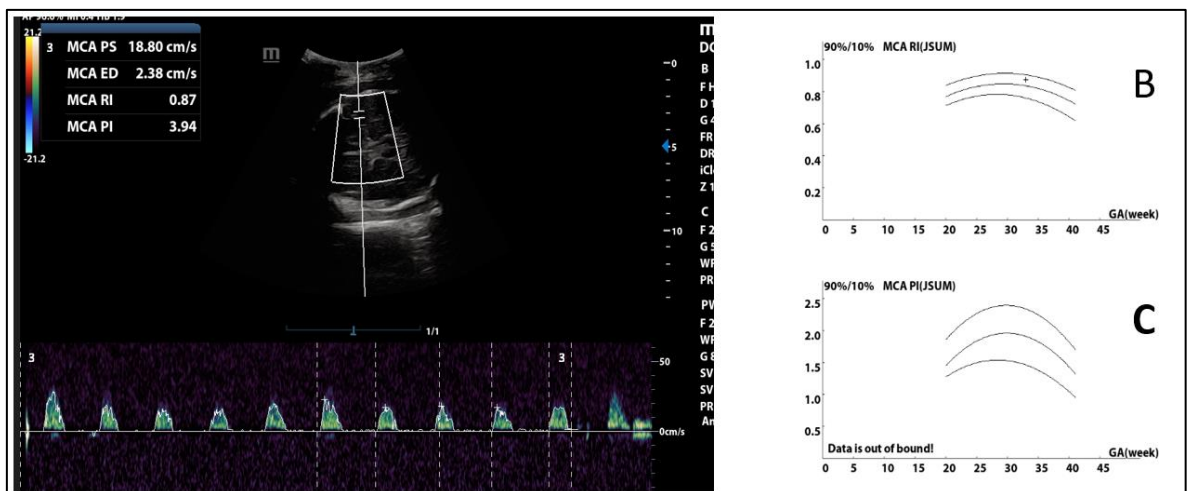


Figure 5: Middle cerebral artery color and spectral Doppler [A], showing absent diastolic flow, B), normal resistance index [RI-0.87] within 90<sup>th</sup> to 10<sup>th</sup> percentile and C), Very high pulsatility index [PI-3.94] above 90<sup>th</sup> percentile per gestational age and beyond the limit of 2.5

## DISCUSSION

Prompt correct diagnosis of impending fetal death and timing of delivery is a key to the prevention of stillbirth in preeclamptic women with placental insufficiency and an IUGR fetus.

According to (Setiawan *et al.*, 2023), both preeclampsia and IUGR are associated with inadequate quality and quantity of the maternal response to placentation. (Aviram *et al.*, 2019) showed that maternal vascular malperfusion is higher in early-onset IUGR, i.e., before 33 weeks, which is consistent with our case. The extent of placental damage determines the severity of obstetric Doppler abnormalities, according to (Morrow *et al.*, 1989). Umbilical artery end-diastolic flow becomes reversed when 60 to 70% of the placental bed is damaged and becomes insufficient. This is similar to our case, [Figure 4A].

The severity of placental insufficiency determines the possibility of stillbirth. The relative risk ratio for stillbirth in pregnant women with preeclampsia varies with gestational age; at 34 weeks, the risk of stillbirth is approximately seven times higher than in normotensive women, while at 28 weeks, the risk is 35 times higher (Harmon *et al.*, 2015). Since our client's clinical gestational age was 33 weeks, and the ultrasound age was 29 weeks. The risk of fetal death was in the range between 35-fold to 7-fold higher.

According to (Spinillo *et al.*, 2009), in IUGR fetuses, the occurrence of absent/reversed end-diastolic blood flow in the umbilical artery [UA] is associated with a significant increase in risk of perinatal and neonatal death or severe brain damage. On this basis, our client had an increased chance of complications to stillbirth or early neonatal death as demonstrated by the Doppler findings in Figure 4A.

According to (Yalti *et al.*, 2004), the cerebral-placental ratio [CPR] is a good predictor of neonatal outcomes and can be utilized to identify fetuses at risk of morbidity and mortality. It is more sensitive and specific in the prediction of poor neonatal outcomes in patients with severe preeclampsia (Abdelrazik Abdelfattah, Mohammed Moustafa, and Mohammed Taha 2020). In these cases, a cerebral-placental ratio of less than 1 is associated with more adverse outcomes [ie, increased perinatal morbidity and mortality] (Yalti *et al.*, 2004). The ratio becomes less than 1 owing to the fetal physiological response to hypoxia, where brain-sparing mechanism issues such that the impedance to flow in the middle cerebral artery becomes low to compensate for fetal hypoxia when there is decreased placental perfusion [(Yalti *et al.*, 2004; Dhand, Kansal, and Dave 2011). However, this is contrary to our case, the ratio IS 0.38 but not associated with brain sparing due to vasodilatation since the PI in the umbilical artery is extremely high, giving a small CPR despite high cerebral

flow resistance. The CPR, however, does not have a cut-off point indicative of impending fetal death.

According to (Dhand, Kansal, and Dave 2011), in the setting of severe hypoxia in an IUGR fetus, brain sparing precedes normalization of MCA pulsatility index a week before fetal death and becomes reversed a day before fetal death occurs. In our case fetal heartbeat was not heard at around 8:00 pm [approximately 10 hours from the time obstetric Doppler was performed, however, the MCA pulsatility index had not reversed but was deficient almost absent diastolic flow impressing the fact that an absent or low MCA diastolic flow in the setting of fetal hypoxia is as well an indication of terminal hemodynamic breakdown preceding fetal death. We could not exactly establish the time fetal death occurred, similarly, we can't tell how long the fetus had stayed with the absent diastolic flow before the time obstetric Doppler was performed. However, it can be deduced from this that, absent MCA end diastolic flow is a stage toward end diastolic reversal and is indicative of failure of circulatory autoregulation. [(Spinillo *et al.*, 2009) pointed out that MCA end diastolic reversal is uncommon and difficult to detect in clinical practice and has a poor prognosis, reflecting a failure of fetal adaptive mechanisms to intrauterine hypoxia. so, possibly a similar impression can be made to the absent MCA diastolic flow since timing to detect it can be difficult due to the less predictable course of cardiovascular sequences of response to fetal hypoxia in pregnancy complicated with preeclampsia and intrauterine growth restriction, as pointed out by (Mari, Hanif, and Kruger 2008). So, what is the explanation for absent MCA diastolic flow?

According to (Setiawan *et al.*, 2023) under conditions of prolonged hypoxemia there is an increase in intracranial pressure due to cerebral edema which is responsible for increased cerebral vascular resistance resulting in an end-diastolic reversal in MCA, we think the same explanation applies to the absence of end-diastolic flow in the MCA with the difference lying on the difference in the degree of cerebral edema whereas there cerebral edema is high in end-diastolic reversal than absent end diastolic flow. It is important to note other causes of increased intracranial pressure, which include intracranial hemorrhage, severe anemia, and fetal.

## CONCLUSION

An absent end diastolic flow in the middle cerebral artery in the setting of reversed end diastolic flow in umbilical arteries and high flow resistances in uterine arteries is one of the terminal hemodynamic events preceding fetal death. All preeclamptic women with sonographic evidence of intrauterine growth restriction must undergo obstetric Doppler studies to assess the fetal hemodynamic status to allow appropriate timing of delivery to minimize the occurrence of stillbirth.

**Funding:** No funding received.

**Conflict of Interest:** There are no conflicts of interest.

**Informed Consent:** verbal consent was obtained from the patient.

#### Author's Contributions

Dr. Fredrick Malunde: writing original manuscript, Dr. Philip Laizer: clinical patient management. Dr. Alfred Mushumbusi: Manuscript review and editing, Dr. Sylvia Nsato.: manuscript review and editing; All authors have read and agreed to the published version of the manuscript.

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**Cite This Article:** Fredrick Malunde, Philip Laizer, Alfred Mushumbusi, Sylvia Nsato (2025). Impending Stillbirth of an IUGR Fetus in a Preeclamptic Woman and the Role of Doppler Sonography: A Case Report. *EAS J Radiol Imaging Technol*, 7(5), 120-124.

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