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Protocol Based Feto-Maternal Monitoring of Early-Onset Fetal Growth Restriction: A Case Report

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Abstract

Evaluation of fetal growth is one of the critical objectives of prenatal care. Fetus failing to reach its genetic potential is known as fetus growth restriction (FGR). FGR is associated with not only acute neonatal consequences but also long-term metabolic syndrome with risk to multiple organ system. Here is the case report of a 24 year pregnant woman diagnosed with FGR pregnancy at 25+4 weeks gestation and with serial monitoring pregnancy was continued till 34+2 weeks of gestation when its terminated because of 0 Modified biophysical profile (BPP) and gross absence of maternal perception of fetal movement to achieve a neonate with a good outcome. Future prospective studies need to investigate risk factors for infants who are SGA. If reliable prediction can be achieved, there is potential to reduce future perinatal morbidity and mortality, and long-term consequences among SGA babies. Antepartum fetal surveillance using multimodality like ultrasonography with dopplers, BPS, NST, cCTG can be helpful in deciding timing of delivery for best neonatal outcome.

Keywords: ***Fetal growth restriction; Small for gestational age; Cerebro placental ratio; Doppler velocimetry; Biophysical store***

1. Introduction

Fetal growth is dependent on many factors like maternal (demographic, social, dietary intake, maternal habitus, habits, illness), placental, and fetal genetic and chromosomal factors [1]. A SGA fetus is one when its size (biometric evaluation) falls below a predefined threshold for its gestational age. The most common definition of SGA is EFW or AC below the 10th percentile of given reference ranges. Nevertheless, other thresholds have been described, such as the 5th and 3rd percentiles (the latter approximating 2 SD) or a *Z*-score of -2. A fetus failing to reach its genetically predetermined growth potential is known as fetus growth restriction [2]. Early FGR is FGR diagnosed at or below 32 weeks and differs from late-onset FGR in clinical manifestations [3]. There are disturbing uteroplacental functions in early FGR [4]. The acute neonatal consequences of FGR

include metabolic and hematological disturbances, and disrupted thermoregulation. Respiratory distress (RDS), necrotizing enterocolitis (NEC), and retinopathy of prematurity (ROP) too may contribute to perinatal morbidity. FGR babies are at significantly increased risk for type 2 diabetes mellitus (T2DM), obesity, hypertension, dyslipidemia, and insulin resistance (the so-called metabolic syndrome, MS). MS ultimately leads to the premature development of cardiovascular diseases. In addition, short stature in children and adults, premature adrenarche, and the polycystic ovarian syndrome (PCOS) are endocrinological sequelae of IUGR. Early onset growth delay and prematurity significantly increase the risk for neurological sequelae and motor and cognitive delay [5,6].

2. Case History

A 24-year-old primigravida, hailing from the local population of Chhattisgarh, a housewife and belonging to middle socioeconomic class, in non-consanguineous marriage, having primary education, with BMI of 23.5 kg/m^2 referred from a district hospital to tertiary care center at 22 weeks of gestation given baby weight not appropriate for gestational age that was elicited by symphysio-fundal height and also ultrasonographically by Hadlock formula. She had two antenatal visits at a district hospital. This pregnancy was a spontaneous conception after a married life of 9 months. Her previous menstrual cycles were regular at an interval of 28-30 days. A dating scan at 10+4 weeks of gestation corresponded with EDD. Early anomaly scan, Dual marker & Quad test were not done. There was nothing significant in her past medical, surgical & family history. She took a balanced diet without any calorie deficit and adequate protein intake. Her detailed anomaly scan done at 22 weeks was suggestive of an intrauterine fetus of gestation age 19+3 weeks with a weight of 350 grams (<10 percentile), adequate amniotic fluid & fetus without any gross congenital anomaly. TORCH profile and all other routine antenatal investigations were in a normal range. No apparent reason for FGR could be elicited from her detailed clinical history and lab investigations. She was on weekly follow-up in OPD. Her growth scan with Doppler done at AIIMS, Raipur at 25+4 weeks showed a period of gestation as 22+3 weeks with estimated fetal weight (EFW) to be 470 grams (<1 percentile) &mean uterine artery PI artery 99 percentile suggestive of Stage 1 FGR (FIG. 1).

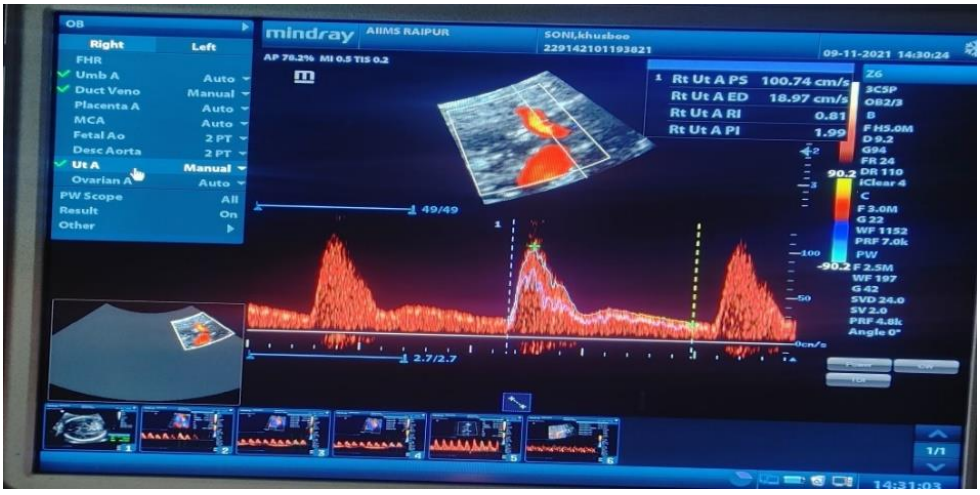


FIG. 1. **Showing uterine artery PI>99%.**

At 32 weeks gestation, she got admitted to the Antenatal ward for daily feto-maternal surveillance. Along with vital monitoring, Blood sugar charting, her daily NST, and biweekly Dopplers done for fetal monitoring. She received a high-protein diabetic

diet with adequate hydration. At 33+4 weeks of gestation, absent flow in the umbilical artery was noted, after which monitoring was done with ductus venous waveform (FIG. 2).

FIG. 2. **Showing AEDF in UA.**

At 34+2 weeks,reversal of umbilical artery flow along with the reversal of a wave was noted in ductus venosus waveforms.(FIG. 3 & 4) .

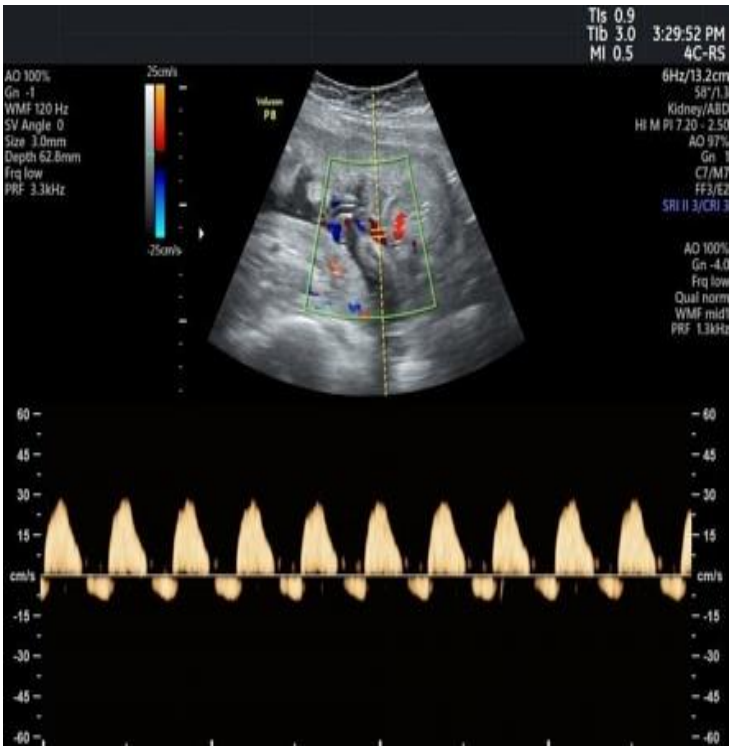


FIG. 3. **Showing REDF in UA.**

FIG. 4. **Showing DV a wave reversal.**

In NST, there was persistent decreased variability and late decelerations (FIG. 5).

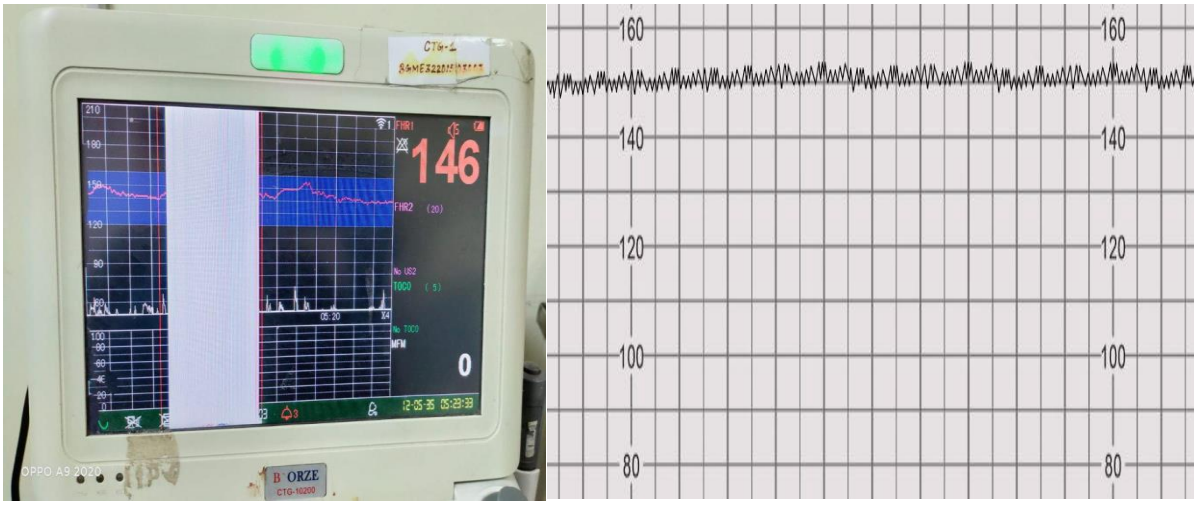


FIG.5. **Showing decreased beat to beat variability in NST.**

TABLE 1. **Depicting her Serial USG parameters.**

GROWTH SCAN	**Doppler Parameters**

POG	USG POG	AFI	EFW Gm (%)	Mean Ut Artery PI (%)	UMB ARTERY PI (%)	MCA PI (%)	CPR PI	DV PI (%)
25+4	22+3	11.33	470.6 (<1)	1.7(>99)	1.11 (35)	2.14 (35)	1.93(55)	
28+2	23+6	10.25	624.9 (<1)	2.45(>99)	1.18 (59)	1.56 (9)	1.32(16)	
32+1	26+2	5.12	866 (<1)		1.2 (76)	1.3 (3)	1.08	
32+6	26+3	5.1	882 (<1)		1.7(99)	1.02 (1)	0.82	
33+3	29+2	3.1	892 (<1)		Absent	1.1(1)		0.47
33+6	29+4	1.5	904 (<1)		Absent	1.1 1(1)	0.64	PI>95%
34+2	29+6	Absent	930 (<1)		Reversal	1.05 (<1)	0.4	A wave reversal

Emergency LSCS done at 34+2 weeks of gestation because of 0 modified biophysical profile with an absence of maternal perception of fetal movement & a preterm, alive male baby of weight 1.025 kg delivered by vertex. Intraoperatively liquor was minimal but clear. The baby cried immediately after birth. The APGAR score of the baby at 5 and 10 minutes were 6/10 and 8/10. The baby shifted to NICU because of low birth weight and prematurity. Cord blood ABG was done immediately after birth showed a pH OF 7.2. After one 1week,the baby started taking maternal milk. After four weeks of admission to NICU, the baby was discharged at the weight of 1.66 Kg. The baby was healthy at the time of discharge, and after three months of follow-up, the baby is doing well. After 6 months of follow up the baby has gained weight of 4.6 kgs and with no neurodevelopmental delay.

3. Discussion

There two main phenotypes of FGR differ in many aspects, such as prevalence, gestation age at onset, placental histopathological findings &Doppler velocimetric profile [7,8].

Delphi consensus has categorized FGR in the absence of congenital anomalies as early and late FGR; details have been described below [9].

Early FGR: GA <32 weeks

AC/EFW <3rd centile or UA-AEDF Or

1. AC/EFW <10th centile combined with

2. Ut A-PI >95th centile and/or
3. UA-PI >95th centile

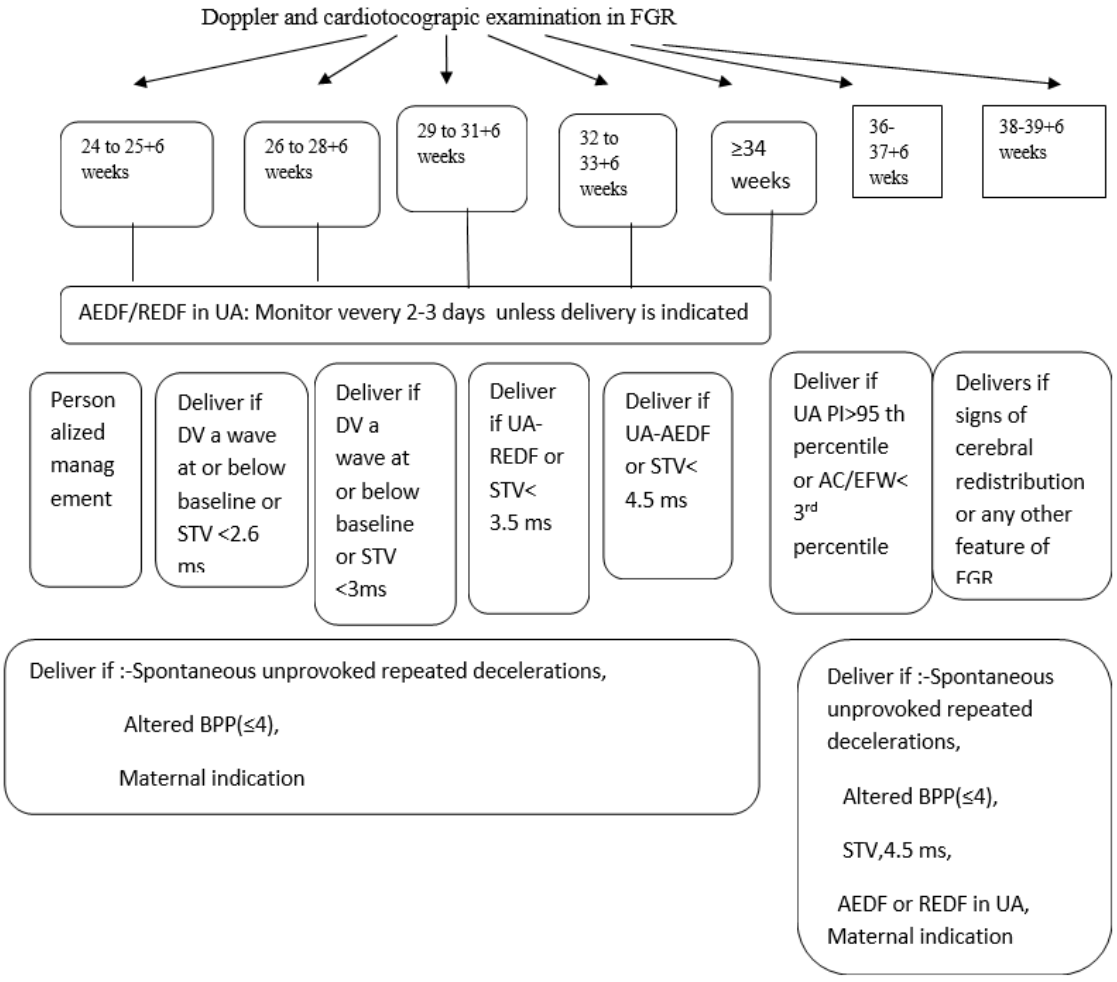
Late FGR: GA ≥ 32 weeks

AC/EFW <3rd centile Or at least two out of three of the following

1. AC/EFW <10th centile
2. AC/EFW crossing centiles >2 quartiles on growth centiles
3. CPR <5th centile or UA-PI >95th centile.

Doppler velocimetric studies play a very important role in the management of FGR [10]. CPR is both used as diagnosis and surveillance of late FGR and used as a guide to decide the termination of pregnancy in late FGR [11].

Following is the flowchart by ISUOG prepared using many guidelines and trial which guides us judiciously to monitor and manage both early and late FGR and decide time of termination of pregnancy [12].



ACOG & SFM recommends magnesium sulfate in pregnant women at high risk of preterm birth (within 24 hours) upto 32 weeks of gestation for neuroprotection in fetus [13].

Antenatal corticosteroid (ACS) in form of Inj Dexamethasone or Inj Betamethasone are given to the pregnant women for fetal lung maturity. These are the recommendations for injectable corticosteroids:

For gestational age <22+0 weeks, ACS are generally not considered as there are few primitive alveoli till this period on which the drug exerts its effect.

At 22+0 to 22+6 weeks of gestation, ACS can be considered in pregnant patients in whom delivery is anticipated in the next seven days [14].

At 23+0 to 33+6 weeks of gestation all guidelines recommend administration of ACS in pregnant patients who are at increased risk of preterm delivery within the next seven days.

NICE guideline recommends considering ACS to pregnant women at **34+0 and 35+6 weeks of gestation** who are in suspected, diagnosed, or established preterm labor; are having a planned preterm birth; or have preterm prelabor rupture of membranes [15].

ACOG recommends administration of ACS for women with a singleton pregnancy at **34+0 to 36+6 weeks of gestation** at imminent risk of preterm birth within seven days [16].

SMFM recommends a two-dose course of ACS to pregnant women at **34+0 to 36+6 weeks of gestation** at high risk for preterm birth within seven days [17].

4. Conclusion

With vigilant feto maternal monitoring, pregnancy should be tried to prolong as much possible that results in lesser NICU stay because of prematurity and other complications or sometimes ameliorate need of NICU. The short- and long-term prognosis are improved with above measures. In our case, early FGR was diagnosed very early but with serial feto maternal monitoring, we prolonged the duration of pregnancy that results in a good baby outcome. Future prospective studies need to investigate risk factors for infants who are SGA. If reliable prediction can be achieved, there is potential to reduce future perinatal morbidity and mortality, and long-term consequences among SGA babies.

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