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Original Research Article

Diagnosis, Classification and Management of Fetal Growth Restriction: A Practice Update

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Abstract:

Background & Methods: The aim of the study is to Diagnose, classify and manage Fetal growth restriction: A practice update. All eligible pregnancies underwent serial sonographic evaluation of fetal weight at 2-weekly intervals until birth and all normally formed fetuses underwent evaluation of amniotic fluid volume. biophysical profile scoring (BPP) and multi-vessel Doppler UA, middle cerebral artery (MCA), ductus venosus (DV), umbilical vein (UV), aortic isthmus (Aol) and myocardial performance index (MPI) at every subsequent contact with the research sonographers.

Results: We found 27% in composite & 12% no morbidity. 02% each in maternal diabetes respectively, we found, 08% in male & 03% in female of Perinatal Morbidity, 1.2% in male & 0.1% in female in Perinatal Mortality.

Conclusion: Significant advances have been made in the understanding the complex etiology and pathophysiology of FGR. This knowledge will certainly aid the clinician to optimize antepartum monitoring and timely planned delivery of FGR infants. Biophysical tests and multivessel Doppler have predictive abilities in detecting academia but their strengths need further evaluation. The temporal correlation between the commonly used tools and preterminal fetal status remains unclear. There are emerging recommendations to combine the use of multiple monitoring tools in models to improve the prediction of adverse outcome. Nonetheless, there is still no definitive cure for FGR and the management strategies for pregnancies complicated by FGR are based on limited evidence. Well-designed randomized clinical trials are much needed that specifically target the different management options.

Keywords: Diagnosis, Classification, Management & Fetal.

Study Design: Observational Study.

1. Introduction

Fetal growth restriction (FGR) refers to a fetus that has failed to achieve its genetically determined growth potential and affects up to 5–10% of pregnancies[1]. Fetal growth restriction is associated with an increase in perinatal mortality and morbidity.

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The evaluation of fetal growth is one of the key objectives of prenatal care. Fetal growth depends on several factors, which includes like uteroplacental function, maternal disease, maternal cardiovascular function or cardiac disease, maternal nutrition, altitude, smoking, illicit drug use, and presence of pathological conditions, such as infection, aneuploidy and some genetic conditions [2].

However, uteroplacental insufficiency or dysfunction represents one of the most frequent causes of abnormal growth in an otherwise normal fetus. Impaired fetal growth is associated with an increased risk of perinatal mortality and morbidity, and long-term adverse infant outcome1. Overall, growth-restricted fetuses have a higher rate of complications associated with prematurity, like poor neurodevelopmental outcome, increased risk of non-communicable diseases in adulthood, such as hypertension, metabolic syndrome, insulin resistance, Type-2 diabetes mellitus, coronary heart disease and stroke[3]. Prenatal recognition of fetal growth restriction (FGR) is a major factor identified in strategies aimed at preventing stillbirth, in which up to 30% of cases are associated with FGR or small-for-gestational age (SGA) in the late third trimester [4].

This study provides definitions of FGR, previously referred to as intrauterine growth restriction, and SGA, and describes the best possible management options based on current data and knowledge [5-7]. For the purposes of this study, we assume that the pregnancy is singleton, pregnancy dating has been carried out correctly (preferably by first trimester ultrasound) and that there are no fetal pathologies, such as aneuploidy, congenital malformation or infection.

2. Material and Methods

Present Study was conducted at Mahaveer Institute of Medical Science for 01 Year on 200 cases. All eligible pregnancies underwent serial sonographic evaluation of normally formed fetus by fwt weight, AFI at 2-weekly intervals until birth and biophysical profile scoring (BPP), multi-vessel Doppler UA, middle cerebral artery (MCA), ductus venosus (DV), umbilical vein (UV), aortic isthmus (Aol), umbilical artery, myocardial performance index (MPI) at every subsequent contact with the research sonographers. In cases of absent (AEDF) or reversed end-diastolic (REDF) in the UA, the patient was admitted to hospital and daily electronic fetal heart rate (CTG) monitoring with computerized analysis of short term variation was carried out.

Corticosteroids to promote fetal lung maturation and reduce perinatal morbidity were administered as a single course between 24 and 36 weeks gestation if preterm delivery was expected likely within one week. Type and dosing regimens of antenatal corticosteroids were as per the local protocol of the participating study centre. Timing of administration and all decisions regarding mode and timing of delivery were at the discretion of the clinician managing each case. Adverse perinatal outcome was defined as a composite outcome of IVH, periventricular leucomalacia (PVL), hypoxic ischaemic encephalopathy (HIE), NEC (Necrotising Entero Colitis), BPD (Broncho Pulmonary Dysplasia)

Delivery indications were recorded on a pre-specified delivery outcome datasheet which was completed by the midwife or physician attending the delivery. The gestational age, antenatal steroids & date and time of delivery and birth weight. infant sex were was recorded together with

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the indication for delivery. This indication of delivery was divided into (i) fetal indications such as non-reassuring fetal heart rate testing, reduced fetal movements, abnormal Doppler findings, oligohydramnios, abnormal biophysical profile or poor interval growth velocity and (ii) maternal indications which included antepartum haemorrhage (APH), pre-eclampsia (PET) / gestational hypertension (GH), diabetes or preterm premature rupture of membranes (PPROM)/ suspected chorioamnionitis. In addition. intrapartum delivery indications included prolonged first and second stage of labour also included. Other variables like Onset of labour, mode of delivery (spontaneous vaginal, vacuum-assisted, forceps-assisted. elective and emergency cesarean delivery), any maternal complications and fetal status at birth (arterial and venous cord pH, Apgars scores, NICU admission) were also collected and transferred to a central consolidated web-based database together with any prenatal ultrasound information.

All cases were prospectively recruited and medical records of all mortalities were reviewed retrospectively for the descriptive analysis of perinatal mortality cases. Comparisons was made to the entire cohort.

The perinatal mortality rate (PNMR) was calculated by the number of still births and neonatal deaths per 1000 live birth and stillbirths (all births were 24 weeks gestation and weighed ≥500 grams) The PNMR was excluded for congenital abnormalities.

Placental abnormalities were categorised according to Redline entena1/4 into villous developmental abnormalities (distal vinous dismaturity/ immaturity. distal villous hypoplasia/ accelerated villous maturation abnormal placental shape and Position), maternal vascular pathology, fetal vascular pathology and inflammatory lesions (acute choriamnionitis chronic unknown aetiology). Maternal vascular injuries included Infarction, retroplacental haemorrhage and increased perivillous fibrinoid degeneration. Fetal vascular Injuries Included true cord knots, cord hypercoiling, abnormal cord insertion & single umbilical artery.

3. Result

Table No. 1: Maternal characteristics

	Composite Morbidity	**No Morbidity**	**P Value**
Maternal Age	32.4±6.3	29.7±6.1	0.331
BMI	27%	12%	0.047
Maternal Diabetes	02%	02%	0.712
GA at Delivery	31.5±4.1	37.9±2.3	0.093

In our study we found 27% in composite & 12% no morbidity. 02% each in maternal diabetes respectively.

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Table No. 2: Indication Delivery of FGR

Indication	Mean Gestational age at Delivery	Mean recruitment to Delivery Interval	P Value
Poor Interval Growth	36.4±2.3	6.8±4.8	0.527
Hypertension	33.7±6.4	4.6±3.8	0.049
Previous Cesarean Delivery	37.9±1.6	7.5±3.7	0.311
Diabetes	36.7±1.1	8.2±7.4	0.744

Table No. 3: Comparison of Perinatal Outcome according to Infant Sex

Outcome	Male 78 (39%)	Female 122 (61%)	P Value
NICU admission	(27)35%	(29)24%	0.001
Perinatal Morbidity	(06)08%	(04)03%	0.001
Perinatal Mortality	(01)1.2%	(0.1)0.1%	0.043

We found, 08% in male & 03% in female of Perinatal Morbidity, 1.2% in male & 0.1% in female in Perinatal Mortality.

4. Discussion

Since approximately 9% of pregnancies are affected by FGR and another 9% by fetal overgrowth, the clinical and societal impact of abnormal fetal growth is significant. Accurate detections of FGR & fetal overgrowth can be challenging[8-11].

At present there is no effective treatment to reverse the course of FGR and macrosomia except delivery. FGR is probably the condition among the obstetric entities with the greatest variation in clinical practice, in terms of monitoring, management strategies and plan of delivery. Prenatal recognition of FGR remains a major challenge in daily obstetric practice[12-14]. Current focus of measurements lies on the nutritional component of fetal deprivation as this can be inferred from size measurements. By using the expression ‘fetal growth restriction’ it is implied that the nutritional component of the deprivation is the biggest threat.

However, the most important outcomes, including the devastating outcome of perinatal mortality, are caused by a deprived oxygen status of the fetus rather than starvation and, unfortunately, we are currently unable to measure fetal serum oxygen levels[15]. New techniques for in vivo assessment of fetal oxygenation, such as the magnetic resonance blood oxygen level dependent (BOLD) effect, are currently being investigated as part of NIH-sponsored Human Placenta Project. Similarly, methods to distinguish healthy large fetuses from overgrown foetuses or to identify overgrown fetuses with appropriate weight are lacking, especially in the absence of maternal diabetes. Beyond the obvious obstetrical concerns of obstructed labor, detecting the latter group with ‘normal size’ is necessary so interventions can be developed to mitigate the associated long-term health risks for the overgrown newborn. Future efforts should focus on the development of a more accurate diagnostic approach that considers fetal body proportion, composition, and metabolic characteristics [16].

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5. Conclusion

Significant advances have been made in the understanding of the complex etiology and pathophysiology of FGR. This knowledge will certainly aid the clinician to optimize antepartum monitoring and time delivery of FGR infants. Biophysical tests and multivessel Doppler have predictive abilities in detecting academia but their strengths need further evaluation. The temporal correlation between the commonly used tools and preterminal fetal status remains unclear. There are emerging recommendations to combine the use of multiple monitoring tools in models to improve the prediction of adverse outcome. Nonetheless, there is still no definitive cure for FGR and management strategies for pregnancies complicated by FGR, are based on limited evidence. Well-designed randomized clinical trials are much needed that specifically target the different management options.

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