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FETAL CARDIAC FUNCTION ASSESSED BY M - MODE ANNULAR EXCURSION IN IUGR FETUSES

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Conflicts of Interest: Nil

ABSTRACT:

Introduction: Fetal growth restriction (FGR) affects 5-10% of pregnancies and is associated with increased risk of perinatal morbidity, mortality and long term complications. Heart is the central organ in the prenatal adaptation to placental insufficiency and fetal hypoxia leading to cardiac remodeling. It causes changes in cardiac shape subclinical cardiac dysfunction and vascular remodeling. Longitudinal myocardial motion is a sensitive early parameter for the indication of subclinical dysfunction in utero and can be assessed by M-mode. Here in this study we evaluated MAPSE for identifying high risk group among IUGR fetuses who could be targeted for early detection of cardiovascular dysfunction.

Materials and Methods: Foetal echocardiacography was done to evaluated mitral valve displacement by M - mode and was compared between IUGR and normal growth babies. Neonatal outcome were also recorded and compared

Results: 40 cases each were taken in the study and the control group. IUGR fetuses had abnormal fetoplacental doppler parameters. Mitral annular plane systolic excursion was reduced in IUGR babies as compared to normal fetuses.

Conclusion: IUGRfetuses present signs of subclinical cardiac dysfunction. Early identification of high risk groups among the IUGR fetuses, who are at an increased cardiovascular risk can be identified by applying functional fetal echocardiography in routine practice.

Keywords: FGR, Fetoplacental Doppler, M - mode, MAPSE.

1. Introduction

Fetal growth restriction (FGR), also known as intrauterine growth restriction (IUGR) is defined when estimated fetal weight is below 10^{th} percentile for a given gestational age. It is a pathological process that modifies the growth potential of the fetus and restricts its intrauterine development.[1] It affects 5-10% of pregnancies and is associated with increased risk of perinatal morbidity, mortality and long term complications.[2] However, which are seen only in a fraction of children. Therefore, focused approach is necessary for the selection of subjects at a higher risk.[3]

Clinically, FGR is suspected when serial measurement of symphyseal - fundal height shows a lag of 4 weeks. It is only a physical screening test with its poor sensitivity and specificity.[4] Ultrasound with Doppler studies is the benchmark for accurate pregnancy dating, diagnosis and management of FGR. Doppler is used to determine vascular resistance and end organ function. Doppler assessment is mainly done of umbilical artery (UA), middle cerebral artery (MCA), and ductus venosus (DV).[5]

The rapid cell proliferation and differentiation during fetal growth are sensitive to even smallest changes that damage the environment leading to permanent alterations in structural and functional constitution, which may persist into the adult life.[6] Hence, IUGR can cause multifactorial damage to fetuses and neonates.[7] The sequence

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of hemodynamic adaptation starts with placental failure and ends at postnatal cardiovascular dysfunction.[8] This signifies that heart is the central organ in the prenatal adaptation to placental insufficiency and fetal hypoxia. Chronic pressure/volume overload together with hypoxia in utero have been postulated as the potential underlying mechanistic pathway of cardiovascular remodeling in FGR.[9] Cardiac remodeling leads to systolic and diastolic dysfunction with preserved ejection fraction.[10] Remodeling causes changes in cardiac shape (more globular morphology), subclinical cardiac dysfunction (increased heart rate and reduced stroke volume, myocardial peak velocities and longitudinal motion), and vascular remodeling (increased blood pressure and carotid intima media thickness). These findings suggest that IUGR induces primary cardiac changes, which could explain the increased predisposition to cardiovascular disease in adult life.[11]

Fetal echocardiography has developed over the past 20 years as the primary non invasive modality to evaluate fetal cardiac anatomy, function and hemodynamics. Longitudinal myocardial motion is a sensitive early parameter for the indication of subclinical dysfunction in utero.[12] It is assessed by long - axis displacement (LAD) of the artrioventricular (AV) valve annulus or by measuring annular displacement from end diastole to end systole (i.e. relative descent of the annulus). It is the most commonly used parameter, because it is an easy and reproducible method to measure myocardial motion. It is investigated by means of tissue doppler imaging (TDI) or M - mode imaging. As TDI is highly demanding and requires expertise and specific equipment and software,[13] M-mode is used because of its easy procreation and performability. M - mode is most suited to right ventricle examination because of the longitudinal orientation of the deep right ventricle muscle fibers, as compared to the mainly circumferential arrangement of left ventricle muscle fibers.[14] In the fetus, M-mode can be applied to measure the annular displacement of mitral and tricuspid valves,(TAPSE and MAPSE, respectively) and correlates well with systolic ventricular performance. Hence, TAPSE/MAPSE in IUGR fetuses are sensitive markers of cardiac dysfunction as they reflect global longitudinal function.

In this study, mitral annular systolic excursion : MAPSE by M-mode have been evaluated for identifying high risk group among IUGR fetuses who could be targeted for early detection of cardiovascular dysfunction.

2. Material and Method :

It is a hospital based prospective and comparative analytical study. It is performed in the Department of Obstetrics and Gynecology, in collaboration with Department of Cardiology at SMS Medical College and attached hospitals, Jaipur for a period of 1 year. The study included two groups : A and B consisting of 40 women each with singleton pregnancy with gestational period of more than equal to 28 wks with IUGR and with normal fetuses respectively. Pregnancies with medical disorders or congenitally malformed fetuses were excluded. Informed written consent was taken from the women willing to participate in the study. Approval from Institutional Research, Review Board and Ethical Committee was taken. Detailed history and examination with all relevant routine investigations were done. Ultrasound was done to diagnose IUGR (Abdominal circumference $<10^{th}$ percentile). Doppler & biometry measurement were performed. Fetal Biometry included: - Femur length, Bi-parietal diameter, Abdominal circumference, Liquor amount and Estimated fetal weight

Fetal peripheral circulation included : - a)Umbilical artery (UA) Doppler pulsatility index (PI), resistance index (RI) was measured on a free-floating loop of the umbilical cord. b)Middle cerebral artery (MCA) pulsatility index (PI), resistance index (RI) was measured in a transverse view of the fetal head at the level of its origin from the circle of Willis. c) Cerebroplacental ratio – obtained by dividing MCA-PI / UA-PCI.

In Fetal echocardiography annular m – mode displacement as MAPSE (Mitral annular plane systolic excursion) TAPSE (Tricuspid annular plane systolic excursion) and SAPSE (Septal annular plane systolic excursion) were measured for both the intrauterine growth restricted and appropriate gestational age fetuses. This was done by the same cardiologist in real-time using a 2–6-MHz linear curved-array transducer in an apical or basal four-chamber view, by placing the cursor at

right angles to the atrio ventricular junction, marked by the valve rings at the mitral, tricuspid and basal septum, respectively. Maximum amplitude of motion was taken as the extent of displacement between end-systole and end-diastole (measured in mm). For both measurements, insonation by the ultrasound beam was kept at an angle of $<30^{\circ}$ to the orientation of the ventricular wall or the interventricular septum, with no angle correction.

3. Results:

Table 1: Baseline and Perinatal Characteristics of the Study Groups

Characteristics	Case (n = 40)	Control (n = 40)	p-value
Maternal Age			
Maternal Age (yrs)	25.03 ± 3.51	25.70 ± 3.78	0.410
Haemoglobin (gm/dl)	9.67 ± 0.91	9.61 ± 1.12	0.810
BMI (kg/m^2)	19.71 ± 1.90	21.91 ± 2.09	0.009
Primipara	21 (52.50%)	17 (42.50%)	0.272
Fetoplacental USG			
GA at USG (wks)	35.95 ± 2.52	35.70 ± 1.74	0.607
EFW (kg)	1.80 ± 0.44	2.23 ± 0.42	<0.001
AFI (cm)	3.75 ± 1.86	7.63 ± 1.29	<0.001
Mean UA-PI	1.486 ± 0.402	0.780 ± 0.228	<0.001
Mean MCA-PI	1.694 ± 0.526	1.840 ± 0.471	0.195
CPR	1.224 ± 0.602	1.835 ± 0.590	<0.001
BSE	19 (47.50%)	0 (0.00%)	<0.001
Pregnancy Outcome			
Pre-eclampsia	10 (25.00%)	0 (0.00%)	0.002
GA at Delivery (wks)	37.35 ± 1.35	39.00 ± 0.91	<0.001
Birth Weight (kg)	1.96 ± 0.37	2.86 ± 0.22	<0.001
Caesarean Section	23 (57.50%)	12 (30.00%)	0.02
5 min APGAR Score <7	5.88 ± 1.28	7.18 ± 0.98	<0.001
Days in NICU	5.65 ± 6.94	0.13 ± 0.52	<0.001
Perinatal Mortality	1 (2.50%)	0 (0.00%)	1.0

The above table shows characteristics of the study groups. Baseline characteristics were similar in both study and control group, except BMI which was significantly lower in cases as compared to the controls. Gestational age at ultrasound was comparable in both groups, while all fetoplacental Doppler parameters showed significantly jeopardised in the IUGR group. The growth-restricted group showed a significantly higher prevalence of pre-eclampsia, lower birth-weight centile and longer neonatal hospitalization in the neonatal intensive care unit as expected. Also as compared to the controls, IUGR fetuses were delivered mainly by Caesarean section and with poor 5-min Apgar scores.

Table 2: Distribution of Cases According to MAPSE

MAPSE (in mm)	Case		Control	
	No.	%	No.	%
2 - 4	4	10.00	1	2.50
4.1 - 6	20	50.00	19	47.50
6.1 - 8	15	37.50	18	45.00
8.1 - 10	1	2.50	2	5.00
Total	**40**	**100**	**40**	**100**

$\chi^2 = 2.432$ *d.f. = 3* *p = 0.665* *NS*

The above table shows distribution of cases according to MAPSE values in IUGR and control group. Mitral displacement of 4.1 - 6 mm was observed in 50% of the cases both in the case and control group. 40% IUGR fetuses and 50% normal fetuses had MAPSE from 6.1-8 mm. 10% fetuses in the study group had a longitudinal displacement of tricuspid valve <4mm as compared to only 1 in the controls.

Table 3: Distribution of Cases According to Mean MAPSE

MAPSE	Case	Control	p-value
Mean ± SD	5.824 ± 1.220	6.113 ± 1.195	0.288, NS
Median	6.000	6.080	
Min-Max	2.7 - 8.6	4.0 - 8.3	
IQR	5.34-6.5	5.298 - 6.923	

Mean MAPSE in cases was 5.824 ± 1.220 mm and median was 6 mm whereas in control group mean was 6.113 ± 1.195 mm and median was 6.080 mm. Though MAPSE was found to be decreased in the IUGR group as compared to the controls, the difference between the two groups was not statistically significant.

4. Discussion:

Many studies have reported that due to chronic hypoxia and elevated placental resistance in IUGR fetuses, there is both pressure & volume overload on fetal heart. This leads to more globular shape, decreased longitudinal function and impaired relaxation of fetal heart with a preserved ejection fraction. All these changes finally results into subclinical systolic & diastolic dysfunction.

Our results were similar to those by Cruz-Lemini M et al (2013)[13] who studied annular M-mode displacement in IUGR fetuses. They found that MAPSE was significantly reduced in IUGR fetuses (3.9 ± 1.2 mm) as compared to the control group (5.5 ± 0.8 mm).

Our results were similar to that by Crispi F et al (2014)[15] who studied post systolic shortening of myocardium in FGR fetuses. They observed reduction in MAPSE among the IUGR group (4.7 ± 1.4 mm) as compared to control group (5.5 ± 1.4 mm), but the difference was not statistically significant. Cruz-Lemini M et al (2014)[3] calculated cardiovascular score in IUGR fetuses to predict infant hypertension and arterial remodelling. The value of MAPSE in IUGR group was 5.0 ± 1.1 mm, while among the control group was 5.5 ± 1.3 mm. Our results were similar to their results. Our findings also correlated with the work of Perez-Cruz M et al (2015)[16] who observed significant reduction in MAPSE in IUGR group (4.9 ± 0.9 mm) as compared to control group (6.0 ± 1.0 mm) while assessing cardiac function in IUGR and SGA babies.

The study by Cruz-Lemini M et al (2016)[17] on cardiovascular remodelling in intrauterine growth restriction babies also found that there is reduction in MAPSE in IUGR group (5.0 ± 1.0 mm) as compared to the controls (5.3 ± 1.2 mm). Our results were similar to this study. Our results were comparable to those by Rodriguez-Guerineau L et al (2018)[7] who evaluated cardiovascular adaptation in IUGR babies. The study concluded that MAPSE is decreased in IUGR babies when compared with control group (6.1 v/s 6.9 mm).

5. Conclusion:

IUGR fetuses present signs of subclinical cardiac dysfunction and have have cardiac remodelling even in the absence of severity predictors in comparison with normally grown fetuses which can be characterised by prenatal functional echocardiography. It includes techniques like longitudinal axis motion in M – mode that can be used to detect subtle cardiac dysfunction. It is more feasible, reproducible and requires a lower technical demand in comparison to TDI for monitoring these fetuses. Early identification of high risk groups among the IUGR fetuses, who are at an increased cardiovascular risk, would benefit in long run by prompt adoption of lifestyle changes and preventive strategies of cardiovascular disease. Fetal echocardiography is currently used mainly in

the research work. Further studies in IUGR and other fetal conditions are required to validate our findings. Thus fetal echocardiography in routine clinical practice could lay new paths for targeted intervention to reduce the risk of cardiovascular disease and improve their health in the long run.

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