Original Article

The Impacts of Maternal Gestational Diabetes Mellitus (GDM) on Fetal Hearts*

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Abstract

Objective To evaluate the fetal cardiac function in gestational diabetes mellitus (GDM) pregnancies under different maternal glycemic controls.

Methods Forty four GDM mothers received 78 fetal echocardiographic evaluations at three gestational periods (<28, 28-34 and ⩾34 weeks) and were divided into poorly-(DM1) and well-(DM2) controlled groups according to their glycemic control at examination. Seventy uncomplicated mothers were selected as controls. Parameters of fetal cardiac anatomy and function were measured and analyzed.

Results GDM fetuses' cardiac ventricular walls were thicker than controls', and the differences between DM1 and DM2 were not significant except for end-diastolic left ventricular walls. In both GDM groups, the aortic flow velocities increased earlier than pulmonary artery and DM1 fetuses changed earlier than DM2 ones. GDM fetuses' left atrial shortening fraction was smaller than the controls' in the period of ⩾34 weeks and negatively correlated with thicknesses of left ventricular walls and interventricular septum in DM1 fetuses (r=-0.438 and -0.506). The right ventricular diastolic function in DM1 and DM2 fetuses decreased after the period of 28-34 weeks and in the period of ⩾34 weeks respectively. Tei index of both left and right ventricles increased in DM1 group after the period of <28 weeks and in DM2 group only in the period of ⩾34 weeks, with no significant differences between DM1 and DM2 groups in this period.

Conclusion Fetuses of GDM mothers showed cardiac function impairments. Good maternal glycemic control may delay the impairments, but cannot reduce the degree. Some cardiac changes in GDM fetuses were similar to those in pregestational diabetic pregnancies except for several parameters and their changing time.

Key words: Gestational diabetes mellitus (GDM); Fetal; Hearts; Tei Index; Glycemic control

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INTRODUCTION

During gestation, maternal intrauterine environments exert significant influences on fetal developments. Maternal hyperglycemia is a common gestational risk factor for fetuses. In all the diabetic pregnancies, gestational diabetes mellitus (GDM) accounts for more than 80% and the rests are pregestational diabetes or gestational impaired glucose tolerance (GIGT). In China, the prevalence of GDM is reported as 2%-5% and has been increasing in recent years[1].

In pregestational diabetes, high maternal glucose levels usually appear before pregnancies and

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can cause fetal abortion or malformation in early pregnancies. In contrast with pregestational diabetes, abnormal maternal glucose levels in GDM usually start from the middle (12-28 weeks) and third (⩾28 weeks) trimesters, therefore seldom causing fetal abortion or malformation. However, maternal hyperglycemia in GDM can accelerate the fetal growth and cause fetal macrosomia and fetal distress in the third trimester[1]. Many previous studies showed that maternal hyperglycemia can cause fetal myocardial hypertrophy and impairments of cardiac diastolic function[2-5]. However, most of these studies were conducted in Western countries and focused on pregestational diabetic women. The impacts of GDM have been seldom reported. With an increasing prevalence of GDM in China, it is important to study the effects of GDM, especially under different maternal glycemic controls, on fetal cardiac function.

The aim of this study is to evaluate the fetal cardiac anatomy and function in GDM pregnancies under different maternal glycemic controls.

MATERIALS AND METHODS

Data Objects of Study

Between January 1, 2007 and August 31, 2008, 44 GDM women and 70 normal pregnant women were recruited from two obstetric clinical centers in Shanghai. Between gestational 24-28 weeks, an oral 50 gram glucose screening test was given. Women with 1 h glucose level ⩾7.8 mmol/L were regarded as abnormal and further received an oral glucose tolerance test (OGTT)[6]. GDM was determined when two or more of the glucose levels met the following thresholds after a 75-g oral glucose tolerance test: fasting blood glucose ⩾5.3 mmol/L, 1 h blood glucose ⩾10.3 mmol/L, 2 h blood glucose ⩾8.6 mmol/L, and 3 h blood glucose ⩾6.7 mmol/L[7]. Women with pregestational diabetes, gestational impaired glucose tolerance (GIGT) or any other kinds of abnormal pregnancies were excluded.

After being diagnosed, all the GDM women received the treatments of diet control and their blood glucose levels were monitored continuously. If their blood glucose was controlled unsatisfactorily, insulin therapy was applied. Fetal echocardiography was performed in these GDM women at three gestational periods: <28 weeks, 28-34 weeks and ⩾34 weeks. The 44 GDM women underwent 78 examinations and each woman was examined no more than once at each gestational period. According to the treatment targets recommended in the guidelines of American Diabetes Association, we used fasting/2 h postprandial blood glucose <5.0-5.5/6.7-7.1 mmol/L as the normal criteria[8]. When glucose levels met the criteria each time, GDM was regarded as well controlled. According to the glucose levels during one month before sonographic examinations, the GDM women were divided into poorly-controlled group (DM1) and well-controlled group (DM2). Seventy gestational ages-matched healthy pregnant women were recruited as controls.

All the women were followed up until delivery. To compare their babies' characteristics at birth, we also divided the GDM women into DM1' (poorly-controlled) and DM2' (well-controlled) groups according to their glycemic control at delivery.

Sonographic Examinations

Fetal echocardiograms were performed by using GE V730 or Philip HP4500 ultrasound systems with 3.5- and 5-MHz transducer. Gestational ages were determined by both last menstrual periods and fetal sizes. Intimate fetal echocardiographic scans were performed to exclude congenital heart structural abnormalities. The cardiac sizes and function were then measured as follows.

Cardiac sizes: end-systolic and end-diastolic thicknesses of interventricular septum (IVS), left ventricular (LV), and right ventricular (RV) walls were measured in the lateral four-chamber view.

Systolic function parameters: peak flow velocities and velocity-time integrals of aorta and pulmonary artery in pulsed Doppler pattern, left and right cardiac outputs were calculated by the formula "velocity time integral×valve area×heart rate", and right to left cardiac outputs ratios, LV fractional shortening and ejection fraction were calculated by LV dimension measured in the lateral four-chamber view.

Diastolic function parameters: E and A wave velocities in atrioventricular pulsed Doppler pattern, E/A ratios, left atrial shortening fraction(LASF) were calculated by the formula "(diastolic–systolic)/ diastolic left atrial dimension", and the "S", "D", and "a" wave velocities were measured in pulsed Doppler pattern of inferior vena cave flow and the preload index was defined by "a/S"[9].

Global function parameters: LV and RV Tei index (myocardial performance index) was measured and calculated by the standard pulsed Doppler methods[10].

In all the Doppler pattern, the angles between

the transducer beam and the blood flow direction were kept <20°. All the parameters were recorded by 3 to 5 cardiac cycles for offline analyses and measured by one single investigator (C.C.).

The study protocol was approved by the Clinical Pharmacology Base and Pediatric Ethics Committee of Fudan University. Informed consents were obtained from all participants.

Statistical Analysis

The data were analyzed by the statistical program SPSS Version 11.5. The results were expressed as $\bar{X} \pm s$. One-way analysis of variance (ANOVA) was used to compare the measurements between the DM1, DM2, and control groups. In multiple comparisons, Least Significant Difference (LSD) and Tamhane's T2 were used respectively in homogeneity and heterogeneity of variance. Correlations between two variables were calculated by using Pearson coefficients. *P*-value <0.05 was considered statistically significant.

RESULTS

Patients' Characteristics

Forty nine GDM women were identified in the DM1 group, and 29 in the DM2 group. Among the DM1, DM2, and control groups, the women's gestational weeks at examination were comparable at the three gestational periods (*P*=0.400 for <28 weeks, 0.492 for 28-34 weeks, and 0.166 for ⩾34 weeks)(Table 1).

Table 1. The Women's Gestational Ages at Examination

Subgroup (weeks)	DM1 (*n*=49)			DM2 (*n*=29)			Control (*n*=70)		
	<28	28-34	⩾34	<28	28-34	⩾34	<28	28-34	⩾34
n	14	21	14	2	8	19	25	25	20
Gestational Ages (weeks)	25.3±2.0	30.3±1.5	35.5±1.3	26.3±0.2	31.2±1.5	36.5±1.3	25.3±1.4	30.5±1.5	36.2±1.6

All the women had single births. According to the women's glycemic control at delivery, 23 women were identified in the DM1' group and 21 women were in the DM2' group. The women' ages were significantly different among the DM1' (33.2±4.7 years), DM2' (30.1±4.0 years), and control groups (28.3±2.7 years) (DM1' > DM2' > control, *P*<0.001). In both DM1' and DM2' groups, the fetuses' gestational ages at birth were smaller compared with the control group (37.5±1.3, 38.1±1.1, and 39.2±1.2 weeks, *P*=0.000 and 0.004 respectively), and the difference between DM1' and DM2' groups was not significant (*P*=0.168). The fetuses' birth weights were not significantly different in the DM1' (3291.8±483.6 grams), DM2' (3390.8±329.9 grams) and control groups (3352.3±398.7 grams, *P*=0.722).

Thicknesses of Ventricular Walls

Compared with the control group, the end-systolic and end-diastolic IVS in both DM1 and DM2 groups showed more progressive thickening during the three gestational periods. In the period of ⩾34 weeks, the IVS of DM1 fetuses were thicker than the IVS of DM2 fetuses, although the differences were not significant.

In the period of 28-34 weeks, the end-systolic and end-diastolic LV and RV walls in DM1 and DM2 groups were significantly thicker than in the control group (*P*<0.05) with no differences between DM1 and DM2 groups (*P*=0.489-1.000). In the period of ⩾34 weeks, the thicknesses of LV and RV walls increased progressively in the DM1 group and were thicker than in the control group (*P*=0.002-0.025). The differences between the DM2 and control groups were significant in the thicknesses of end-systolic LV and RV walls (*P*=0.028 and 0.033). In the period of ⩾34 week, no significant differences were found between the DM1 and DM2 groups except for the thicknesses of end-diastolic LV walls (*P*=0.027)(Table 2).

Ventricular Systolic Parameters

In each gestational period, the aortic flow velocities and left cardiac outputs in the DM1 group were higher than in the control group (*P*=0.000-0.047). In the DM2 group, they increased only in the periods of 28-34 and ⩾34 weeks (*P*= 0.000-0.031) and had no significant differences with the DM1 group in these two periods.

Compared with the control group, the pulmonary arterial flow velocities in the DM1 group increased from the period of 28-34 weeks (*P*=0.002-0.000), and in the DM2 group the increase occurred only in the period of ⩾34 weeks (*P*=0.013). The right cardiac outputs did not differ in the DM1, DM2, and control groups (*P*>0.05).

Table 2. Thicknesses of Ventricular Walls in the Three Groups

Gestational Ages \ Group		DM1	DM2	Control	*F* Value	*P* Value
<28 weeks	S-IVS	4.0±1.1	3.5±0.5	2.7±0.5	13.683	0.000
	S-LVW	3.0±0.7	3.3±0.5	2.6±0.6	2.097	0.137
	S-RVW	2.9±0.6	3.0±0.1	2.5±0.3	4.275	0.021
	D-IVS	2.5±0.6	2.5±0.1	1.9±0.2	4.694	0.034
	D-LVW	2.0±0.4	2.4±0.3	1.8±0.2	4.318	0.041
	D-RVW	2.0±0.4	2.4±0.2	1.8±0.2	3.535	0.065
28-34 weeks	S-IVS	4.6±1.0	5.0±1.0	3.1±0.7	23.067	0.000
	S-LVW	3.5±0.6	3.6±0.5	2.9±0.7	6.383	0.003
	S-RVW	3.4±0.6	3.5±0.6	2.7±0.4	11.436	0.000
	D-IVS	3.3±0.8	3.3±1.1	2.5±0.5	4.295	0.026
	D-LVW	2.6±0.4	2.7±0.5	2.2±0.3	4.845	0.018
	D-RVW	2.6±0.4	2.6±0.5	2.2±0.3	3.022	0.068
⩾34 weeks	S-IVS	5.8±2.0	5.2±0.8	3.6±0.6	14.387	0.000
	S-LVW	4.1±0.8	3.8±0.5	3.4±0.6	5.647	0.006
	S-RVW	4.1±0.8	3.8±0.7	3.3±0.4	5.730	0.006
	D-IVS	4.0±1.0	3.9±0.6	3.0±0.5	5.458	0.011
	D-LVW	3.3±0.6	2.9±0.3	2.7±0.4	5.029	0.015
	D-RVW	3.1±0.4	2.9±0.4	2.6±0.4	2.888	0.045

Note. Unit: mm; S-, systolic; D-, diastolic; IVS, interventricular septum; LVW, left ventricular wall; RVW, right ventricular wall.

During pregnancies, the ratios of right to left cardiac outputs increased in the control group but had no significant changes in the DM1 or DM2 group. In the period of ⩾34 weeks, the ratios in both DM1 and DM2 groups were smaller than in the control group (P=0.027 and 0.001, respectively). During all the three gestational periods, fetal LV fractional shortening and ejection fraction did not differ among the DM1, DM2, and control groups (P>0.05) (Table 3).

Table 3. Ventricular Systolic Function in the Three Groups

Gestational Ages \ Group		DM1	DM2	Control	*F*	*P*
<28 weeks	Vmax of AO* (cm/s)	85.6±9.9	83.3±10.0	74.3±10.2	5.641	0.007
	Left Cardiac Outputs (mL)	229.6±74.3	223.1±70.8	176.2±52.5	3.593	0.037
	Vmax of PA▲ (cm/s)	70.5±10.5	72.9±1.58	66.8±11.3	0.731	0.488
	Right Cardiac Outputs (mL)	268.1±71.3	285.1±25.7	224.2±77.0	1.929	0.159
	Right / Left Cardiac Outputs Ratio	1.21±0.25	1.36±0.55	1.30±0.32	0.451	0.640
	LV Fractional Shortening	0.315±0.052	0.327±0.014	0.312±0.068	1.756	0.186
	LV Ejection Fraction	0.673±0.071	0.701±0.015	0.665±0.088	1.851	0.171
28-34 weeks	Vmax of AO* (cm/s)	92.2±12.7	90.9±10.7	77.1±8.1	13.330	0.000
	Left Cardiac Outputs (mL)	342.1±104.9	370.2±128.0	269.6±69.4	5.154	0.009
	Vmax of PA▲ (cm/s)	75.0±10.4	71.8±9.4	66.9±6.2	5.153	0.009
	Right Cardiac Outputs (mL)	442.1±157.0	433.2±136.0	350.4±79.4	3.539	0.036
	Right / Left Cardiac Outputs Ratio	1.31±0.29	1.23±0.44	1.34±0.28	0.375	0.689
	LV Fractional Shortening	0.284±0.058	0.312±0.077	0.307±0.044	1.212	0.306
	LV Ejection Fraction	0.627±0.089	0.663±0.126	0.663±0.066	1.162	0.321

(Continued)

Gestational Ages \ Group		DM1	DM2	Control	*F*	*P*
⩾34 weeks	Vmax of AO* (cm/s)	100.1±14.9	97.0±6.9	81.4±1.6	14.580	0.000
	Left Cardiac Outputs (mL)	544.8±168.5	544.7±170.6	435.8±113.4	3.115	0.043
	Vmax of PA▲ (cm/s)	81.0±8.8	75.6±5.4	69.8±6.9	10.867	0.000
	Right Cardiac Outputs (mL)	691.1±187.4	630.3±148.0	627.0±100.4	0.949	0.394
	Right / Left Cardiac Outputs Ratio	1.30±0.20	1.19±0.21	1.50±0.32	7.100	0.002
	LV Fractional Shortening	0.277±0.053	0.277±0.058	0.297±0.041	3.016	0.058
	LV Ejection Fraction	0.616±0.080	0.613±0.098	0.650±0.062	3.264	0.057

Note. *Vmax of AO, peak velocities of the aortic flow; ▲Vmax of PA, peak velocities of pulmonary arterial flow.

Ventricular Diastolic Parameters

During the three gestational periods, the mitral E, A velocities, and E/A ratios did not differ significantly among the DM1, DM2, and control groups (P>0.05), except for mitral A velocities between the DM1 and control groups in the periods of <28 weeks and 28-34 weeks (P=0.022 and 0.012). In the period of ⩾34 weeks, LASF in both DM1 and DM2 groups were significantly smaller than in the control group (P=0.002 and 0.047), and had no significant difference between the DM1 and DM2 groups (P=0.173). In the DM1 group, LASF was negatively correlated with end-diastolic thicknesses of LV walls and IVS (Pearson r=-0.438 and -0.506, P=0.002 and 0.008 respectively).

In the periods of 28-34 weeks and ⩾34 weeks, tricuspid E velocities and E/A ratios in both DM1 and DM2 groups were smaller than in the control group (P=0.000-0.046). Tricuspid A velocities had no differences among the three groups. The preload index of inferior vena cave in both DM1 and DM2 groups were higher than in the control group (P=0.004-0.024), but had no significant differences between the DM1 and DM2 groups (Table 4).

Table 4. Ventricular Diastolic Function in the Three Groups

Gestational Ages \ Group		DM1	DM2	Control	*F*	*P*
<28 weeks	Mitral E (cm/s)	34.4±4.1	35.1±2.5	32.6±5.4	0.668	0.519
	Mitral A (cm/s)	55.4±6.2	51.5±4.0	49.9±7.4	2.858	0.070
	Mitral E/A Ratio	0.624±0.071	0.684±0.101	0.658±0.087	1.004	0.376
	LASF	0.252±0.084	0.190±0.038	0.278±0.067	1.662	0.203
	Tricuspid E (cm/s)	37.2±7.9	37.9±13.7	37.9±5.3	0.050	0.952
	Tricuspid A (cm/s)	58.7±10.0	53.3±11.9	55.3±8.4	0.739	0.484
	Tricuspid E/A Ratio	0.638±0.104	0.700±0.101	0.692±0.080	1.725	0.192
	Preload Index of Inferior Vena Cave	0.465±0.102	0.458±0.059	0.391±0.115	1.849	0.172
28-34 weeks	Mitral E (cm/s)	37.2±5.0	36.9±4.5	35.5±5.9	0.652	0.526
	Mitral A (cm/s)	53.0±5.7	50.8±7.0	48.2±6.3	3.437	0.040
	Mitral E/A Ratio	0.704±0.078	0.732±0.082	0.738±0.065	1.269	0.290
	LASF	0.281±0.061	0.234±0.035	0.259±0.071	1.739	0.186
	Tricuspid E (cm/s)	37.0±5.9	33.9±4.5	42.6±10.0	4.716	0.014
	Tricuspid A (cm/s)	55.7±7.3	58.2±9.9	52.9±7.6	1.523	0.229
	Tricuspid E/A Ratio	0.670±0.113	0.589±0.070	0.763±0.096	9.199	0.000
	Preload Index of Inferior Vena Cave	0.456±0.108	0.459±0.119	0.353±0.111	5.452	0.007
⩾34 weeks	Mitral E (cm/s)	38.1±6.8	34.3±5.1	37.0±5.5	2.024	0.143
	Mitral A (cm/s)	51.0±13.1	45.9±8.4	48.2±6.8	1.185	0.314
	Mitral E/A Ratio	0.766±0.115	0.756±0.089	0.770±0.064	0.121	0.886
	LASF	0.216±0.058	0.241±0.037	0.275±0.058	5.497	0.007
	Tricuspid E (cm/s)	41.1±9.7	40.3±8.7	47.4±7.6	3.525	0.037
	Tricuspid A (cm/s)	59.7±13.0	58.9±10.3	60.3±7.4	0.092	0.912
	Tricuspid E/A Ratio	0.689±0.096	0.685±0.102	0.781±0.074	5.632	0.006
	Preload Index of Inferior Vena Cave	0.472±0.115	0.481±0.153	0.343±0.125	5.253	0.009

Parameters of Global Ventricular Function (Tei Index)

In the control group, both LV and RV Tei index remained similar during the pregnancies. In both DM1 and DM2 groups, LV Tei index increased with the gestational weeks (r=0.379 and 0.499, P=0.05 and 0.007 respectively), and RV Tei index had no changes during the pregnancies.

During the three gestational periods, LV and RV Tei index in the DM1 group were significantly larger than in the control group (P=0.001-0.046). In the period of ⩾34 weeks, LV and RV Tei index in the DM2 group showed significant differences from the control group (P=0.000 and 0.003 respectively), and had no significant differences with the DM1 group in this period (Table 5).

Table 5. Global Ventricular Function in the Three Groups

Gestational Ages \ Group		DM1	DM2	Control	*F*	*P*
<28 weeks	LV -Tei Index	0.381±0.168	0.236±0.108	0.309±0.067	4.484	0.018
	RV –Tei Index	0.367±0.117	0.387±0.112	0.294±0.067	3.625	0.036
28-34 weeks	LV -Tei Index	0.401±0.140	0.354±0.084	0.316±0.054	4.039	0.024
	RV –Tei Index	0.390±0.096	0.344±0.065	0.295±0.063	8.414	0.001
⩾34 weeks	LV -Tei Index	0.479±0.161	0.484±0.148	0.303±0.073	11.896	0.000
	RV –Tei Index	0.390±0.086	0.372±0.109	0.281±0.068	7.768	0.001

DISCUSSION

To our knowledge, this is the first study in China to comprehensively evaluate the fetal cardiac anatomy and function under different maternal glucose levels in the GDM women. The results showed substantial changes in fetal hearts under maternal hyperglycemia. The maternal glycemic control may be able to delay the time of changes, but can not reduce the degree.

In this study, the glycemic poorly-controlled women were older than the well-controlled and normal women. Although these women were less than 35 years, our results suggested that more GDM and GDM with poor glycemic control took place in older pregnant women, which was consistent with previous reports that older pregnant women had a higher risk of GDM[11-12]. Although the GDM fetuses' birth ages were smaller than the control group', the birth weights did not differ, indicating that the maternal hyperglycemia stimulated the accelerated fetal growths.

Thickness of Ventricular Walls

Previous studies have reported the thickened IVS in the fetuses of pregestational diabetic mothers[2-3,13]. Similarly, the GDM fetuses in our study showed the thickened LV, RV walls and IVS, suggesting that the maternal hyperglycemia caused the fetal hypertrophic myocardium regardless of the time of onset. The poorly-controlled groups showed no differences in IVS and more thickened end-diastolic LV walls compared with the well-controlled group. Gardiner et al. reported similar findings on pregestational diabetes[4]. However, Wong et al. did not find the differences of IVS between poorly and well controlled groups[14]. Our results suggested that satisfactory maternal glycemic control could lessen the changes in thickening of ventricular walls, but had no obvious influences on the IVS in both GDM and pregestational diabetic pregnancies.

Ventricular Systolic Function

In this study, the increased systolic parameters in the GDM fetuses reflected the increased fetal circulation under hypermetabolism in GDM pregnancy. Jaeggi[5] and Lisowski[15] have reported similar findings in type-1 diabetic pregnancy. Meanwhile, the LV and RV systolic parameters increased after the middle and late pregnancies respectively, and the poorly-controlled groups changed earlier than the well-controlled groups, suggesting that fetal left hearts accommodated earlier than right hearts and fetuses under poor glycemic control had earlier changes.

In the normal fetuses, the ratios of right to left cardiac outputs increase during pregnancies, which is consistent with the right heart dominance in the fetal period. However, in GDM pregnancies, hyperglycemia causes fetal hypermetabolism, increased oxygen need, and relative lack of oxygen, leading to the redistribution of fetal circulation. More blood is thereafter distributed to the left heart to supply brain and then the ratios of right to left outputs decrease, as was described in our study.

Lisowski et al. have reported similar changes in type-1 diabetic pregnant fetuses[15]. Compensations of fetal circulation exist in these two types of diabetic pregnancies.

Ventricular Diastolic Function

The decreased ventricular diastolic parameters, including the mitral and tricuspid E velocities and E/A ratios in the late pregnancies, have been well documented in previous studies on pregestational diabetes[16-18]. In our study, similar changes in RV diastolic parameters were observed in GDM fetuses. However, the LV diastolic parameters, including mitral E velocities and E/A ratios, did not show significant changes. As pointed out by Briguori et al.[19], LASF correlates to LV compliance and provides a more reliable noninvasive assessment of the diastolic function than the Doppler indexes in hypertrophic cardiomyopathy. Therefore, we used LASF to evaluate the fetal LV diastolic function since GDM has similar pathologic manifestations with hypertrophic cardiomyopathy. The decreased LASF in GDM fetuses reflected the decreased LV compliance in late pregnancies. The correlations between LASF and thicknesses of LV walls suggested the close relationship between myocardial hypertrophy, impaired LV compliance and reduced left atrial activity. Zielinsky et al. reported similar findings on pregestational diabetic fetuses[20]. Moreover, our analysis showed that RV was affected earlier than LV, and maternal glycemic control had little influences on the changes of fetal ventricular diastolic function.

Global Ventricular Function

Tei index has been widely used as a sonographic parameter quantitatively measuring the global ventricular function in both adults and children[21-23]; however, its application in diabetic pregnant fetuses is seldom reported. Tsutsumi et al. reported the increased LV and RV Tei index in fetuses of diabetic mothers after 27 weeks[24]. Ichizuka et al. found that only fetuses of a larger gestational age showed increased Tei index[25]. In contrast, some studies reported opposite findings. For example, Eidem et al. did not find the differences in Tei index between diabetic and normal pregnant fetuses[26]. Another study on GIGT women found that fetal LV and RV Tei index decreased after 34weeks, which might be due to the potential increase in ventricular contractility[27].

In our study, the increased LV and RV index in the GDM fetuses indicated the impairments of the global ventricular function. In contrast with Wong et al.'s study[27], we speculated that the increased systolic parameters and compensation mechanism implied the potential impairments of ventricular contractility. Meanwhile, the diastolic function decreased; therefore, Tei index combined systolic and diastolic function showed earlier and more sensitive changes. Comparison of the gestational periods showed that RV changed earlier than LV, which was consistent with the changing time of single systolic or diastolic parameters. Moreover, comparisons between the poorly and well controlled groups indicated that good maternal glycemic control might delay the time of fetal cardiac function impairments to some extent.

There are some limitations in this study. First, glycosylated hemoglobin was not measured in the GDM women. Alternatively, we used glucose monitoring to determine the poorly- and well-controlled groups. Second, the limited sample size may decrease the statistical power and affect the results. Further studies with more participants are needed.

In conclusion, GDM fetuses showed changes of cardiac anatomy and function, including thickened ventricular walls, increased fetal circulation, decreased diastolic function and global ventricular function. Tei index changed earlier than other parameters. RV was affected earlier than LV. Some changes of fetal hearts in GDM were similar to those in pregestational diabetes. Good maternal glycemic control delayed the impairments of fetal hearts but could not reduce the degree. Our results may help to interpret why some GDM fetuses in good glycemic control still have unsatisfactory outcomes. We hope to supply more clinical information for gestational monitoring of GDM women in obstetric clinic.

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