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ORIGINAL ARTICLE

Prevalence of Gestational Diabetes Mellitus in Obstetric patients using Macrosomia and Increased Amniotic Fluid on Ultrasound, as Diagnostic Markers

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ABSTRACT

Aim: To determine the prevalence of gestational diabetes in obstetric patients with the help of ultrasonography and diagnostic biomarkers such as increased amniotic fluid and macrosomia.
Methods: In 110 women who failed the glucose tolerance test, longitudinal ultrasound measurements of foetal growth were taken during the first, second and third trimesters with the informed consent of every pregnant women. 524 ultrasound examinations were performed in total, and uncomplicated pregnancies were included as controls for the comparison of our results. Head circumference (HC), abdominal circumference (AC), femur length (FL), and head circumference to abdominal circumference ratio (HC/AC) was evaluated at 17th and 37th weeks of gestation while amniotic fluid was measured and recorded at 13th, 27th and 37th weeks of gestation respectively.
Results: The mean HbA1c (%) of the pregnant women in 1st semester recorded was 5.2±0.27SD which increased to 5.31 ±0.24SD in 2nd trimester and later changed to 5.54±0.17SD in 3rd semester. The measured amniotic fluid in ultrasound was 23.23cm ±3.18 SD at 13th weeks of gestation, 15.97cm±2.62SD at 27th weeks and 11.95cm±1.99SD at 37th week. The mean abdominal circumference at 37th week was 347.01mm ±7.28SD, mean head circumference was 1477.50mm±2.88SD, AC/HC ratio at 37th week estimated 0.89 ±0.08SD and femur length was 73.44mm ±2.28SD respectively.
Conclusion: The finding suggests that increased amniotic fluid and macrosomia are important biomarkers of gestational diabetes and can be assessed through ultrasonography.
Keywords: Gestational diabetes mellitus, obstetrics, macrosomiqa, amniotic fluid

INTRODUCTION

Previous researches say that in diabetic pregnancies, foetal hyperinsulinemia is a common final pathway to adverse outcomes. Amniotic fluid insulin levels predict diabetes-related morbidity[2], which reflects foetal insulin output directly; however, maternal parameters such as blood glucose and glycosylated haemoglobin values cannot reliably identify fetopathy[3]. It makes sense to use insulin levels in amniotic fluid as a guide when starting insulin therapy to avoid such neonatal morbidity. It's been shown in a new study that diet-treated glucose-intolerant women have better pregnancy outcomes than women who received insulin therapy if their amniotic fluid insulin levels were higher than the 97th percentile.

There has been an increase in the number of cases of foetal macrosomia as average birth weight has risen, a condition associated with increased neonatal and maternal morbidity[1]. Morbidity is most commonly increased by pregnancy-related conditions like gestational diabetes and maternal obesity. Some doctors may use ultrasound-estimated foetal weight as justification for inducing labour or performing an elective Cesarean section if a woman has foetal macrosomia.

It is common for macrosomia (defined as a birth weight above the 90th percentile) to cause short- and long-term complications in diabetic pregnancies, such as prolonged labour, shoulder dystocia, and neonatal asphyxia. Macrosomia is more common in pregnancies complicated by diabetes, whether type 1 (DM1), type 2 (DM2), or gestational diabetes mellitus (GDM)[5,6]. There is an increased risk of foetal macrosomia in diabetic pregnancies when the mother's body fat percentage is higher and the foetus has more fat mass. neonates have a higher total fat index (17%) and are larger than expected for their gestational age in comparison to other pregnancies[7,8].

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Non-macrosomic and proportionate macrosomia infants have lower rates of hyperbilirubinemia, hypoglycemia and perinatal acidosis than disproportionate macrosomia children. Understanding the foetal growth profiles that lead to macrosomia in diabetic pregnancies may help with the development of preventive strategies. For the time being, there is a dearth of information on the growth profiles of diabetic pregnancies as a whole. According to previous research in these pregnancies[21–23], foetal growth accelerates between weeks 18 and 24 of pregnancy. According to previous research, growth picks up speed around the 32-week mark. Mousavi et al[26] examined pregnancies in women with DM1 and discovered a rapid increase in AC at 24 weeks of pregnancy in these women. A new study by Souza et al[27] found a link between accelerated AC growth in the second trimester and LGA births in DM1 pregnancies. There was no difference between the subtypes treated with insulin after a single analysis of growth rates in different types of diabetes in foetal AC after 28 weeks. According to our findings, no studies have examined whether or not a larger-than-normal HC/AC ratio occurs in diabetic pregnancies. Stratified analyses can include women with DM1, DM2, or GDM, as well as normal or increased birth weight.

The aim of the current study is to evaluate the prevalence of gestational diabetes mellitus in the pregnant women while fetal macrosomia and high amniotic fluid are used as diagnostic biomarkers.

METHODOLOGY

With the approval of the ethical review committee and informed written consent from every patient, the study had started and the pregnancies were included which occurred between the years 2018 and 2020. A local control group of women was considered with normal pregnancies that had undergone research-related serial ultrasound examinations were used to compare the prenatal growth profiles. All pregnancies that didn't include prenatal dating scans were discarded from the analysis.

The newborns were found to be normal and free of any inherited conditions. Clubfoot, hypospadias, and palatoschisis were discovered in one child with an atrial septal defect Type II (which did not require surgery). An undiagnosed child with scafocephaly at the age of five months was also found to have the condition. Except for one of these pregnancies, women who became pregnant more than once during the study were excluded from the analysis.

Preterm birth is defined as occurring before 37 weeks of gestation. If you've been diagnosed with diabetes mellitus type 1 (DM1), you have a low or undetectable C-peptide and/or GAD positivity, as well as a ketoacidosis episode. Insulin treatment should be started within three months of your diagnosis. Oral glucose-lowering medication before conception, insulin treatment before conception, or long-term insulin treatment without ketoacidotic episodes and being GAD-negative with a normal or elevated C-peptide level were all considered risk factors for the development of type 2 diabetes mellitus (DM2).

According to American Diabetes Association recommendations[21], a 100-gram oral glucose tolerance test was used to identify GDM. Data on both the mother and the unborn child as well as the newborn were entered into a database for every diabetic pregnancy that had taken place. All three trimesters included glycosylated haemoglobin in the maternal glycemic profile (HbA1c). GDM diagnosis was delayed until the second trimester because haemoglobin A1c values from the first trimester were mostly in range. A comprehensive oral glucose tolerance test was only performed on women who were at high risk of developing GDM or who had hyperglycemia-related symptoms.

The software, SPSS version 25, was used to retroactively collect ultrasound data from a different database. The foetus' hip, ankle, and femur circumferences and lengths were all measured (FL). HC/AC was also calculated to determine whether or not it indicated growth issues. A birth weight greater than the 90th percentile, corrected for gestational age and gender, was considered macrosomia, while a birth weight greater than the 97.7th percentile, corrected for gender and ethnicity, was considered severe macrosomia. It was decided whether or not a baby had macrosomia based on whether or not it had it as an infant. The foetal growth in diabetic pregnancies was monitored with four or five ultrasounds between 17 and 37 weeks of pregnancy. Amniotic fulid was measured and recorded at 13th, 27th and 37Th weeks of gestation through ultrasound. It's worth noting that the non-experimental group was subjected to 5–9 ultrasounds between 20 and 36 weeks of pregnancy in order to measure HC, AC, and FL.

The statistical analysis is done using SPSS. Ordinal and nominal data was presented as frequencies and percentages while quantitative data was evaluated as mean and standard deviation. With the available reference ranges for amniotic fluid and fetal circumferences, one sample t-test has applied to analyze the significance statistically. P value> 0.05 with 95% confidence interval was considered significant.

RESULTS

Overall, 110 women who met the inclusion criteria were considered for the study. The demographic characteristics including their age, weight, previous histories of miscarriage, macrosomia, high amniotic fluid, family history of diabetes, delivery time and other co morbidities were recorded as described in table 1.

Table 1

Demographic characteristics	
Age (years)	29.14 ±6.73SD
Pre-pregnancy weight (kg)	69.80 ±10.01SD
Previous Pregnancies	2.46 ±1.25SD
Family History of Diabetes	33 (30%)
Previous Miscarriage History	28 (25.5%)
Previous Fetal Macrosomia History	28 (25.5%)
Previous Fetal High Amniotic Fluid History	22 (20%)
Preterm Delivery	19 (17.3%)
Delivery Time in weeks	37.8 ±1.38SD
Pre-eclampsia	18 (16.4%)
C- Section	64 (58.2%)

The mean HbA1c (%) of the pregnant women in 1st semester recorded was 5.2 ±0.27SD which increased to 5.31±0.24SD in 2nd trimester and later changed to 5.54 ±0.17SD in 3rd semester. The measured amniotic fluid in ultrasound was 23.23cm±3.18 SD at 13th weeks of gestation, 15.97cm ±2.62SD at 27th weeks and 11.95cm ±1.99SD at 37th week. The mean abdominal circumference at 37th week was 347.01mm ±7.28SD, mean head circumference was 1477.50mm ±2.88SD, AC/HC ratio at 37th week estimated 0.89 ±0.08SD and femur length was 73.44mm ±2.28SD respectively.

The mean birth weight of the babies was 8.06oz ±0.66SD, 44 born were boys and 66 were girls. 26.4% showed positive results of macrosomia (>90th percentile) and 10.9% positive results of severe macrosomia (>97%)

Table 2: Descriptive statistics

	N	Minimum	Maximum	Mean	Std. Deviation	P value
HbA1c (%) 1st Trimester	110	4.80	5.70	5.2445	.27912	<0.05
HbA1c (%) 2nd Trimester	110	4.90	5.70	5.3191	.24401	<0.05
HbA1c (%) 3rd Trimester	110	5.30	5.80	5.5482	.17066	<0.05
Amniotic fluid at 13 weeks (cm)	110	18.00	29.00	23.2364	3.18231	<0.05
Amniotic fluid at 27 weeks (cm)	110	12.00	21.00	15.9727	2.62122	<0.05
Amniotic fluid at 37 weeks (cm)	110	9.00	15.00	11.9545	1.99718	<0.05
Abdominal Circumference at 17 weeks (mm)	110	108.00	115.00	111.1000	2.35380	<0.05
Head Circumference at 17 weeks	110	143.00	152.00	147.5000	2.88543	<0.05
AC/HC ratio at 17 weeks	110	1.00	1.30	1.1482	.11230	<0.05
Femur Length at 17 weeks (mm)	110	20.00	31.00	25.4091	3.50176	<0.05
Abdominal Circumference at 37 weeks (mm)	110	335.00	360.00	347.0182	7.28701	<0.05
Head Circumference at 37 weeks (mm)	110	335.00	360.00	346.0364	7.28191	<0.05
AC/HC ratio at 37 weeks	110	.80	1.00	.8991	.08183	<0.05
Femur Length at 37 weeks (mm)	110	70.00	77.00	73.4455	2.28512	<0.05

Table 3:

Neonatal characteristics	
Birth Weight (pounds)	8.06 ±0.66SD
Macrosomia (>90th percentile)	29 (26.4%)
Severe Macrosomia (>97th percentile)	12 (10.9%)
Gender (Boy/Girls)	44/66

DISCUSSION

These findings show disproportionate growth in unborn children, particularly macrosomic children, but also in children of normal birth weight in women with diabetes mellitus types 1 and 2, as well as gestational diabetes mellitus (GDM). When a woman has diabetes, her babies do not grow in two cohorts, one with normal growth and the other with accelerated growth[19]. Most babies will be affected by an abnormal intrauterine environment, based on this data. Shoulder dystocia, which has been linked to abnormally large foetal growth in diabetic women, is more common during labour. Neonatal studies are rare and incomplete because of the rarity of disproportionate pregnancy growth being studied in such a systematic manner.

According to another study, the HC and AC length of diabetic infants were the same as those of control infants, but the

diabetic infants were heavier at birth[19]. Other evidence suggests that low birth weight is linked to early indicators of poor placentation, and normal placentation have higher birth weights in women with DM type 1, DM type 2 and GDM[16]. There are several types of diabetes in women, and it's important to look at the foetal growth patterns for each type individually. Only women with DM1 were studied by Dong et al[17] and Vitner et al[18], who discovered that early growth was accelerated in DM type 1. The results were the same for both of us.

To our surprise, a study by Wang et al[19] found no differences between the intrauterine growth trajectories of AC and FL in the foetuses of women with type 1 or type 2 diabetes. In contrast our population had the highest prevalence of macrosomia at birth, as compared to the one described by Wang et al[19], where birth-weight centiles did not differ significantly. These authors don't give an HC/AC ratio. Researchers have discovered a link between HbA1C levels in the first trimester and early placental markers (like pregnancy-associated plasma protein-A)[27]. DM type 1 pregnancies have shorter crown-rump lengths and a faster rate of biparietal diameter growth in the second trimester, according to the results[23]. Premature growth retardation has been linked to a number of factors, including insufficient yolk sac maturation and postponed conception. This is understandable because there are no differences between the GDM group and controls in early pregnancy. The glucose metabolism of GDM women is still normal during early pregnancy[22].

Women with type 1 diabetes had the greatest growth deviation. For the purposes of this research, diabetics with HbA1c levels greater than or equal to 7.0 were considered to have poor control (significant for the second and third trimesters). When it comes to glycemic control, "almost good is not good enough," but HbA1C values were better than most in the literature[24]. In spite of their importance, there are few links between HbA1c levels in the foetus and overall growth. These findings suggest that foetal growth may be influenced by other factors or that HbA1c is not a reliable indicator of glucose control, particularly in the slightly abnormal range (2–4 SD). The greatest impact on foetal growth rate may be found at low glucose levels, according to other researchers[22]. Even though HbA1c values weren't different between macrosomic and non-macrosomic foetuses in women with GDM, we'd previously seen higher second-trimester glucose profiles in macrosomic cases[21].

The insulin concentration in amniotic fluid can be used to diagnose biochemical fetopathy. As demonstrated in this series, physical diabetic fetopathy manifestations in the neonate should be avoided if maternal insulin therapy is initiated early enough during the course of such a biochemical fetopathy[22]. These variables can also be used to assess the success of therapeutic intervention biochemically (higher blood insulin levels in the umbilical cord) and clinically (birth weight, prematurity, and the presence of diabetic fetopathy). Intrauterine foetal death as a result of diabetes fetopathy occurs more frequently in obese (and thus hyperinsulinemic) pregnancies[23]. Macrosomia can also result in a traumatic or operative delivery.

Due to the fact that increased foetal insulin production inhibits pulmonary surfactant synthesis, it is possible that two neonates in the non-insulin-treated comparison group died of respiratory distress syndrome (4050 and 3050 g in the 38th and 36th weeks of gestation, respectively). Obesity in childhood is more prevalent in children who have a family history of diabetic fetopathy[24] and during adolescence[26]. According to one study[25], 25% of participants demonstrated an enhanced insulin response to a glucose load, while 18% had a pathologic glucose tolerance test. Diabetes type 2 is defined by a delayed insulin response in response to a glucose challenge[26]. Exposure to foetal beta cells during pregnancy may increase the risk of developing type 2 diabetes later in life[26,27]. After birth, the ability of affected cells to replicate is severely impaired. According to Souza AS[27], "fuel-mediated teratogenesis" may have long-term consequences depending on the gestational stage at which metabolism is disrupted and the cells "at risk" during that stage. This is why it is critical to detect and treat foetal hyperinsulinism as soon as possible.

CONCLUSION

This is concluded that increased amniotic fluid and macrosomia are important biomarkers of gestational diabetes and can be assessed through ultrasonography.

Conflict of interest: Nil

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