



P-ISSN: 2664-3685

E-ISSN: 2664-3693

IJPG 2020; 3(2): 13-15

Received: 15-10-2019

Accepted: 17-11-2019

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## Correlation of glycemic control in newborn at birth in relation to maternal glycemic control: A prospective clinical study

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**DOI:** <https://doi.org/10.33545/26643685.2020.v3.i2a.82>

### Abstract

**Aim:** Correlation of glycemic control in newborn at birth in relation to maternal glycemic control.

**Materials and Methods:** The present prospective clinical study was conducted in the Department of Pediatrics and Obstetrics and Gynecology, SVS Medical College and Hospital, MBNR. 35 cases were found to be eligible for inclusion in the study.

**Results:** Mean maternal age was 27.14 years. 40.0% patients were primigravida and 60.0% were multigravida. Significant correlation of neonatal hypoglycemia at maternal HbA1c >6.5 was observed.

**Conclusion:** The transmission of mother's glycemic control to newborn, which acts as a screening test and thereby helps in preventing asymptomatic hypoglycemia and long-term neurologic dysfunction.

**Keywords:** HbA1c, GDM, Hypoglycemia, Glycemic Control

### Introduction

Gestational diabetes is defined as carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy<sup>[1]</sup>. Glucose homeostasis is maintained by the balance between insulin, which reduces glucose levels by increasing cellular uptake and other hormones such as glucagon and cortisol, which both increase glucose production. During pregnancy the human placenta produces additional cortisol as well as other insulin antagonists such as human placental lactogen, progesterone and human chorionic gonadotropin, all of which tend to increase glucose level. Human placental lactogen blocks insulin receptors and increases in direct linear relation to the length of pregnancy. If the pancreatic  $\beta$  islet cells are unable to produce sufficient insulin to balance this increase, or if there is a maternal resistance, the mother may develop gestational diabetes<sup>[2]</sup>.

Excess insulin due to maternal hyperglycemia affects the fetus in two ways. Firstly, insulin promotes fat deposition due to the state of nutrient excess<sup>[3]</sup>.

Secondly, insulin acts as a growth factor, stimulating further growth of the infant in utero. Therefore, fetal hyperinsulinemia results in excessive growth of the fetus, causing one of the major perinatal concerns in GDM i.e. Macrosomia (birth weight greater than 4000 g). Macrosomia may lead to birth trauma including shoulder dystocia, nerve palsies and fractures<sup>[4]</sup>.

GDM is associated with respiratory distress syndrome, neonatal hypoglycemia, hyperbilirubinemia, polycythemia, and hypocalcaemia<sup>[5]</sup>. In utero, the exposure to hyperglycemia has long lasting effects on the infant, increasing their risk of future obesity and type II diabetes mellitus<sup>[6]</sup>.

Studies are needed to explore the possibility of GDM conferring future metabolic risk for mother and child postpartum. We therefore conducted a prospective study among pregnant women to evaluate the correlation of glycemic control in newborn at birth in relation to maternal glycemic control.

### Materials and Methods

The present prospective clinical study was conducted in the Department of Department of Pediatrics and Obstetrics and Gynecology, SVS Medical College and Hospital, MBNR.

**Inclusion Criteria**

1. Patients showed no evidence of;
  - Chronic hypertension,
  - Intrauterine growth restriction,
2. Patients who have provided the informed consent

**Exclusion Criteria**

1. Mothers and neonates, whose HbA1c was not tested
2. Patients who have not signed the informed consent

**Ethical approval and Informed consent**

The study protocol was reviewed by the Ethical Committee of the Hospital and granted ethical clearance. After explaining the purpose and details of the study, a written informed consent was obtained.

**Sample selection**

The sample size was calculated using a prior type of power analysis by G\* Power Software Version 3.0.1.0 (Franz Faul, Universitat Kiel, Germany). The minimum sample size was calculated, following these input conditions: power of 0.80 and  $P \leq 0.05$  and sample size arrived were 35 participants.

**Methodology**

A complete history was taken to know the duration and type of DM in the mother. Detailed anthropometry of the infants was taken and they were classified as appropriate, small, or large for gestational age (AGA, SGA, and LGA) as per Lubchenco’s chart. Maternal HbA1c was done before delivery by high-performance liquid chromatography (HPLC). HbA1c of the newborn was done at 24 h after birth by HPLC. Random blood sugar was determined as per protocol for IDM at 0, 1, 2, 3, 6, 12, 24, and 48 h of life by glucometer. 2D ECHO was performed for all infants to detect any structural or functional abnormality of the heart.

**Statistical analysis**

The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2010) and then exported to data editor page of SPSS version 19 (SPSS Inc., Chicago, Illinois, USA). Descriptive statistics included computation of percentages and means. Pearson correlation coefficient was applied to analyze the quantitative data.

**Results**

**Table 1:** Maternal demographic and anthropometric profile

<b>Mean Age (Years)</b>	27.14±2.51
Mean BMI	27.68±2.11
Gravidity	
Primigravida	21 (60.0%)
Mutigravida	14 (40.0%)
Residence	
Rural	19 (54.3%)
Urban	9 (25.7%)
Peri-urban	7 (20.0%)

**Table 2:** Neonatal anthropometric profile

<b>Gender (M/F)</b>	<b>22 (62.9%) /13 (37.1%)</b>
Mean Gestational Age (weeks)	37.04±1.21
Mean Birth Weight (Kgs.)	3.11±0.61
Mean Height (cm)	47.96±1.78

**Table 3:** Correlation between Maternal and Neonatal HbA1c (%) levels

<b>HbA1c Levels</b>	<b>Mean±SD</b>
Maternal	5.76±0.61
Neonate	5.69±0.79
r-value	-0.211
p-value	0.416 (NS)

Test applied: Pearson Correlation Coefficient

**Table 4:** Correlation between maternal HbA1c (%) and Neonatal Blood Sugar

<b>Maternal HbA1c (%) Levels</b>	<b>Neonatal Blood Sugar (mg/dl) (Mean±SD) at 48 Hr</b>
<5.5	74.03±1.91
5.5-6	73.17±1.09
6-6.5	74.21±1.21
>6.5	67.41±1.77*
p-value	0.007 (Sig.)

Test applied: Oneway ANOVA and Post Hoc (Bonferoni)

**Discussion**

In our study, neonatal hyperglycemia was not witnessed, as mothers were well controlled for their glycemic state. In another a case–control study done by González-Quintero *et al* [7]. Compared fetal outcome in well-controlled GDM versus suboptimal controlled and found higher incidence of macrosomic hypoglycemia, jaundice, and stillbirth in the suboptimally controlled GDM. In the present study macrosomia was the only defect encountered among 2.3% of the neonates.

We observed a significant correlation of neonatal hypoglycemia at maternal HbA1c >6.5. These results were in accordance with the study done by Mahapatra and Raj [8]. Mimouni *et al.* reviewed the pathophysiology and management of neonatal complication of diabetes in pregnancy and concluded that adequate maternal glycemic control before and during pregnancy was the best prevention of many potential problems of IDM [9]. This was similar to our study.

The rate of hypoglycemia was 8% in the study conducted by Qadir *et al.* which did not match with our study. Steninger *et al.* found that even asymptomatic hypoglycemia may be a risk factor for impaired neurodevelopment and must, therefore, be identified, prevented, and treated [11].

**Conclusion**

Our study concluded that the transmission of mother’s glycemic control to newborn which acts as a screening test and thereby helps in preventing asymptomatic hypoglycemia and long-term neurologic dysfunction. It was a single-center study and the sample size was small. Hence the studies with large sample needs to be carried to check the generalize the results.

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