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Maternal Age and BMI as Risk Factors of GDM

Authors
Dr Cinthya C Das[1], Dr Sreekala K N[2], Dr Chaithanya C Das[3]
[1,2]Assistant Professor, Department of Physiology, SUT AMS, TVM
[3]Clinical Associate, SUT Hospital Pattom, TVM

Introduction

Gestational diabetes mellitus (GDM) is carbohydrate intolerance of varied severity with first onset or first recognition during pregnancy [1]. GDM is one of the most prevalent medical conditions in pregnant population. Global prevalence of GDM is on the rise. A study conducted among pregnant population in south Kerala had shown a prevalence as high as11.2%.[2] Gestational diabetes seems to stem from the placenta and several placental hormones that help the development of fetus during pregnancy. During the second and third trimesters, these "insulin-antagonist" hormone levels increase and can cause insulin resistance. Insulin resistance makes it difficult for the mother's body to properly utilize insulin, the hormone that manages glucose or blood sugar levels. This causes a higher-than-normal blood sugar level or hyperglycemia. Hence GDM usually develops between the 24th and 28th week of pregnancy. Normally, after delivery, these hormone levels, as well as glucose levels, return to normal[3]. According to American Diabetes Association (ADA), GDM is pathophysiologically similar to Type 2 diabetes in that the abnormality is in islets cell function or peripheral insulin resistance at receptor level. Studies suggest that the peripheral insulin resistance that characterize GDM likely results from several integrated mechanisms including a decrease in insulin receptor number, a post-receptor defect in insulin action and alterations in glucose transport systems[4,5].

GDM if left untreated results in hyperglycemia in the mother. This can have dire consequences for both mother and baby; moreover these effects may last lifetime. GDM makes the mother more prone to infections and Pregnancy Induced Hypertension (PIH). The big baby of a diabetic mother increases the risk of instrumental delivery, Lower Segment Caesarean Section (LSCS), etc. Thus there is increased birth trauma and perinatal mortality. Moreover GDM increases the chance of developing Type-2 diabetes later in life[6].

Effects of GDM are not limited to the mother. Glucose is the primary fuel source used by foetus. It crosses the placenta by facilitated diffusion. The foetus is constantly exposed to higher levels of maternal circulating glucose and so will experience an increase in foetal insulin production (maternal insulin does not cross the placental barrier). Insulin acts as fetal growth hormone leading to macrosomia. The increased available nutrition also contributes to macrosomia. . As significant brain growth and development takes place during second trimester abnormal fuel

metabolism can result in low IQ, altered behavioural, intellectual and psychological pattern. Unexplained intra-uterine death can occur near term in GDM[7]. Although neonates are large, organs are not necessarily mature. Insulin response to continued excess glucose leads to ß cell hyperplasia. So, newborns are susceptible to hypoglycemia. In addition there is risk of hypocalcemia, hyperbilirubinemia, hypermag-nesemia and respiratory distress syndrome in the neonatal period. Babies born with excess insulin run a higher risk of obesity in adulthood, thereby putting them at higher risk of Type 2 diabetes later in life[8].

Studies have identified several risk factors for the development of GDM such as higher maternal age ,higher BMI, higher parity, higher haemoglobin, higher serum ferritin, family history of type 2 diabetes etc.The objective of our study was to correlate maternal age and BMI to the risk of development of GDM.Identification of risk factors may hopefully aid in lifestyle changes which will help in bringing down the prevalence of GDM in future.

Materials and Methods

A case control study was conducted in the obstetric department at a tertiary care centre in South India. The study was conducted after getting clearance from the Institutional research committee and ethical committee.

The case control study included 85 cases and 85 controls. Cases were women with gestational diabetes mellitus at 24-28 weeks gestation who attended the obstetric OPDof the Hospital during the study period. GDM was diagnosed as per guidelines of National Diabetes data group and American Diabetes Association.

Controls were pregnant women at 24-28 weeks gestation who attended the obstetric OP at the hospital during the study period. GDM was excluded as per guidelines of National diabetes data group and American Diabetes Association.

Those pregnant women with pre-existing diabetes, poly cystic ovarian disease, Acute or Chronic liver disease and acute infections were excluded from the study population.

Cases

Glucose challenge test > 200 mg% or
Glucose tolerance test: 2 or more values abnormal

Controls

Glucose challenge test < 130 mg%

This is as per guidelines of National diabetes data group and American Diabetes Association.

Mode of Data Collection

Baseline characteristics of the study group were recorded using a pretested questionnaire.

Height

Pre-Pregnant height was obtained by recall.

Weight

Weight in kg at entry to antenatal care was obtained from records.

BMI

BMI was calculated asWeight in kg / Height in m^2.

Statistical Analysis

The data collected were entered into a personal computer using Microsoft Excel. Statistical analysis was done using the software SPSS.Age and BMI were expressed as mean +/- standard deviation.Chi square test was applied to find an association between maternal age and GDM. Similarly, chi square test was applied to find an association between BMI and GDM. A p value ≤ .05 was taken as statistically significant.

Results

Age

Table 1. Distribution of age (in years) in cases and controls

AGE (in years)			
Group	Mean	Std. Deviation	p-value
Case	27.8	4.4	<0.001
Control	23.7	3.6	<0.001

Cases belonged to a higher age group (mean 27.8 years) than controls (mean = 23.8 years) Chi-square analysis indicated a significant difference in (p value <0.001) age distribution of cases and controls.

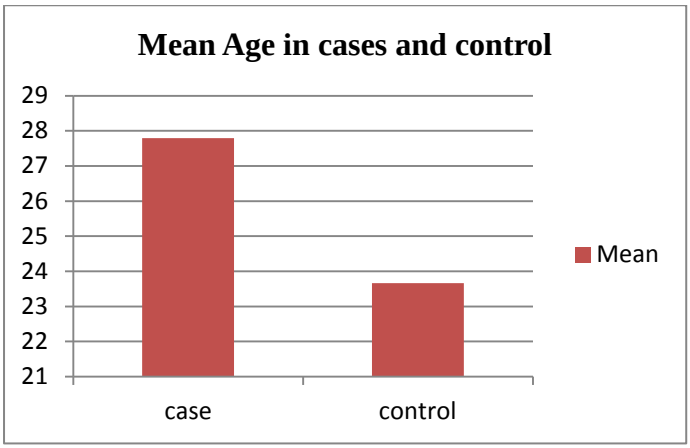

Fig. 1. Mean age in cases and control

BMI

Table 2 Association between BMI and GDM

BMI	Cases	Controls	Number
High	45	13	58
Normal	40	72	112
Total	85	85	170

Chi square 24.64 p value is 0.00

Mean BMI in the study population was 26.8 with a standard deviation of 4.2.

According to BMI the study population was divided into 2 groups: BMI<25 as normal BMI and BMI ≥ 25 as high BMI

Chi square analysis was done and p value was significant (p value is 0.00). This shows that there is a significant association between maternal BMI and the risk of development of GDM. So as the BMI increases there is more chance of developing GDM

Discussion

Role of maternal age

Our study showed a significant difference in maternal age between cases (27.8 years) and controls (23.7 years).The positive correlation in the prevalence of GDM with increasing maternal age was statistically significant (p value <0.001) (Table 1). Maternal age is an established risk factor for GDM[9]; but there is no consensus on the age above which there is significantly increased risk of GDM. In the literature, the lowest cutoff is ≥ 25 years, as recommended by the American Diabetes Association[10], but there is little data to support this. For each year gained after 25 years, the relative risk of GDM is increased by about 4%[11].

Why age is such a strong risk factor is unknown, aside from the general supposition that ß – cell function declines with age[12]. As we age, our body processes become slow or diminished. Carbohydrate intolerance can develop as a part of the ageing process; this may be the consequence of insulin resistance caused by a post-receptor defect resulting in reduced rate of peripheral glucose disposal[12]. Glucose tolerance is determined by a balance between insulin secretion and insulin action and this deteriorates with age. Insulin resistance that develops with age may also be a consequence of increased adiposity.

Increasing maternal age at delivery in the current society may be the reason for the increasing incidence of GDM. The present study reinforces the findings of previous studies and it would be wise for mothers to complete their family before 25-30 years of age.

Role of BMI

In our study, subjects with higher BMI showed a greater risk of developing GDM(table2). This positive association was found to be statistically significant (p value is 0.00).

Body Mass Index (BMI) tends to be used worldwide to define obesity.

$$\text{BMI} = \frac{\text{Weight in kg}}{\text{Height in m}^2}$$

BMI of 25 to 29.9 kg/m^2 is defined overweight and ≥ 30 kg/m^2 obese.[13]

Pregravid obesity is the most well – documented modifiable risk factor for GDM. Obesity is thought to increase the risk of GDM through its association with insulin resistance. The incidence of GDM is increased three-fold in those with BMI ≥ 30 kg/m^2 versus those with a BMI ≤ 20 kg/m^2[9].

In obese individuals, adipose tissue releases increased amounts of non-esterified fatty acids (NEFA), glycerol, hormones, pro-inflammatory cytokines and other factors that are involved in the

development of insulin resistance[14]. Insulin resistance leads to increased activity of hormone sensitive lipase which is probably sufficient to explain the increase in circulating non-esterified fatty acids. The higher circulating non-esterified fatty acids also contribute to insulin resistance. There is now clearevidencethat obesity associated with or without type 2 diabetes is an inflammatory state, consistent with the production of cytokines by the adipose tissue. This also contributes to insulin resistance associated with pregnancy[15].

When insulin resistance is accompanied by dysfunction of pancreatic islet ß-cells – the cells that release insulin – failure to control blood glucose level results. Women with history of GDM are metabolically vulnerable with insufficient ß-cell reserve and are insulin resistant[16]. Abnormalities in ß-cell function are therefore critical in defining the risk and development of Type 2 diabetes. This knowledge is fostering exploration of the molecular and genetic basis of the disease and new approaches to its treatment and prevention.

Thus, gestational diabetes and obesity are the common metabolic abnormalities occurring during pregnancy. Decreased maternal pregravid insulin sensitivity (insulin resistance) coupled with an inadequate insulin response are the pathophysiologic mechanisms underlying, the development of GDM. Insulin regulated carbohydrate, lipid and protein metabolism are all affected to a variable degree. Decreased maternal insulin sensitivity in women with GDM may increase nutrient availability to fetus, possibly accounting for an increased risk of fetal overgrowth and adiposity.

Maintaining healthy body weight will protect mothers from development of such metabolic diseases. Our study recommends mothers to have a long term healthy life style with adequate diet and exercise so that they can have a healthy and uneventful pregnancy.

Conclusions

Our study has shown that increasing maternal age and BMI increase the risk for development of GDM.There was a statistically significant relation between increased maternal age and the risk of development of GDM. Similarly, the relation between increased maternal BMI and risk of development of GDM was also statistically significant.

Extensive studies may be needed to elucidate the maternal age above which risk of development of GDM increases significantly.

A change in lifestyle aiming at a healthy prepregnant BMI may significantly lower the incidence of GDM.

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