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# **Gestational Diabetes Mellitus in Primigravida: An Observational Study.**

# K Saraswathi\*, and S Nirupa.

Department Of Obstetrics and Gynaecology, Sree Balaji Medical College and Hospital, Bharath University, Chennai, Tamil Nadu, India.

# ABSTRACT

GDM is defined as carbohydrate intolerance of varying degrees of severity with first onset or identification during pregnancy. GDM is a widely prevalent disease in our population. Prompt diagnosis of GDM in pregnant women and education about the disease process and complications may delay possible complications. Obesity, ethnicity and history of previous GDM are important and significant risk factors. The current study was done to analyse the prevalence of GDM in primigravidae alone using universal screening approach& influence of obesity and positive family history. 200 primigravida less than 30 yrs of age, who were screen negative for GDM at booking were taken into the study. Risk factors analysed were obesity ( pre pregnancy BMI >=25 kg per metre square) and a positive family history of GDM upto grandparents. 98 women had risk factors and 102 women had no risk factors. All were subjected to universal screening with 75grams ogct between 24 to 28 weeks and the results were noted. Positive predictive value was 70% for family history as a risk factor and it also showed a significant p value(0.05). Obesity as a risk factor did not show a significant p value. 19.6 % of women with no risk factor had GDM in our study. Hence ethnicity plays an important role in GDM in our population. Hence our study comcludes the imporytance of universal screening to detect GDM in non-risk population.

Keywords: Gestational diabetes (GDM), Primigravida, Ethnicity, Universal screening

\*Corresponding author



#### INTRODUCTION

There has been a marked increase in the prevalence of diabetes in Asia, in particular GDM over recent years. Multi-ethnic studies have highlighted the increased risk of GDM among the different Asian populations. Prevalence of GDM in Asian countries varies substantially according to the screening strategy and diagnostic criteria applied, and ranges from 1% to 20%, with evidence of an increasing trend over recent years.

"Gestational diabetes mellitus" (GDM) is defined as carbohydrate intolerance with onset or first recognition during pregnancy [20,21].

GDM mothers are at increased risk of future type 2 diabetes mellitus (DM) as are their children. Thus, its important to screen, diagnose and treat GDM. Timely action taken now in screening all pregnant women for glucose intolerance, achieving euglycemia in them and ensuring adequate nutrition may prevent in all probability, the vicious cycle of transmitting glucose intolerance from one generation to another [1].

# Epidemiology

Hyperglycaemia complicating pregnancy is estimated to affect approximately 16.9% of pregnancies globally, with the highest prevalence in South-East Asia, where an estimated 25% of pregnancies are affected [3].

Among ethnic groups in south Asian countries, Indian women have highest frequency of GDM [27].

The prevalence of GDM in India varied from 3.8 to 21% in different parts of the country, depending on the geographical location and diagnostic methods used [2].

Known risk factors for gestational diabetes include previous history of gestational diabetes, advanced maternal age, obesity, family history of diabetes mellitus and certain ethnicities, including Asians [9-12].

#### **Screening And Diagnosis**

| Organisation   | Fasting<br>Plasma<br>Glucose<br>Mmol/dl | Glucose<br>Challenge | 1Hour<br>Plasma glucose | 2Hours<br>Plasma glucose | 3Hours<br>Plasma glucose |                           |
|--|---|----------------------|-------------------------|--------------------------|--------------------------|---------------------------|
| WHO( 1999)   | ≥ 7.0<br>(126mg)                        | 75g OGTT             | Not<br>required         | ≥ 7.8<br>(140mg)         | Not<br>Required          | Any 1 value               |
| American Congress of<br>Obstetricians and<br>Gynecologists<br>(2001) | >5.3<br>(95mg)                          | 100g<br>OGTT         | >10<br>(180mg)          | >8.5<br>(155mg)          | ≥ 7.8<br>(140mg)         | Any 2 values              |
| IADPSG<br>(2010)   | >5.1<br>(92mg)                          | 75g OGTT             | >10<br>(180mg)          | >8.5<br>(153mg)          | Not required             | Any 1 value<br>except FBS |
| DIPSI(2009)  | -                                       | 75g OGCT             | Not required            | 140mg                    | Not required             | -                         |
| ADA(2010)  | >5.3<br>95mg                            | 75g OGTT             | >10<br>180mg            | 140mg                    | Not required             | Any 2 values              |

#### TABLE 1

#### World Health Organization Procedure

WHO procedure is feasible, sustainable, cost-effective and high impact best buy for low resource settings.

#### The International Association of the Diabetes and Pregnancy Study Groups [7]

Based on the Hyperglycemia and Adverse Pregnancy Outcome (HAPO) study, IADPSG found following disadvantages like-drop outs due to FBS sampling, inconvenience and time consuming.

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# A Single Test Procedure to Diagnose GDM in the Community (Diabetes in Pregnancy Study Group India) [19]

Advantages of the DIPSI procedure are

- Pregnant women need not in fasting state(18)
- Causes least disturbance in a pregnant woman's routine activities
- Less time consuming
- Serves as both screening and diagnostic procedure. This single-step procedure has been approved by Ministry of Health, Government of India(24) and also recommended by WHO

| TABLE 2             |       |            |            |  |  |
|---------------------|-------|------------|------------|--|--|
| TRIAL(75GM GLUCOSE) | FBS() | 1HR PPBS() | 2HR PPBS() |  |  |
| WHO                 | 126   |            | 140        |  |  |
| IADPSG              | 92    | 180        | 153        |  |  |
| DIPSI               | -     |            | 140        |  |  |

VALUES OF WHO,IADPSG AND DIPSI In mg/dl)

#### Aim of Present Study

- To study and compare OGCT in risk and no risk primigravida,<30yrs of age.
- To assess the predictive value of the risk factors- family history and obesity.
- To substantiate the need of universal screening for GDM in our population.

#### MATERIALS AND METHODS

The current study was done in primigravidae, attending antenatal clinic at SBMCH to analyse the outcome of 75grams OGCT at 24-28weeks, among women with and without risk factors.

**Risk factors studied** 

- Family History upto grandparents
- Pre- pregnancy BMI-25kg/m<sup>2</sup> and above

Study period

June 2012 to May 2013

Type of study

Prospective

Study population

200

#### Study pattern

200 primigravidae with 98 women at risk and 102 women without risk. All 200 were screen negative for GDM at booking.

#### **Inclusion Criteria**

- Primi<30yrs
- Spontaneous conception
- Singleton pregnancy
- Family history of DM upto grandparents

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- Screen negative at booking visit
- Booking BMI(obese and non-obese patients)

# **Exclusion Criteria**

- Known pregestational DM
- Multigravidae
- Multiple pregnancies
- First visit at and after 24weeks of gestation.

#### Procedure

All women included in study were given 75 g oral glucose load, irrespective of her fed state, a venous blood sample was collected at 2 hours for estimating plasma glucose by the GOD-POD method. GDM diagnosed if 2-hour post glucose is  $\geq$  140 mg/dL.

# Statistical Analysis: (epi info software)

#### TABLE 3: OGCT STATUS AMONG RISK AND NON RISK GROUPS

|               | OGCT STATUS |            |       |
|---------------|-------------|------------|-------|
|               | OGCT +VE    | OGCT -VE   | TOTAL |
| RISK GROUP    | 50(51.02%)  | 48(48.98%) | 98    |
| NO-RISK GROUP | 20(19.61%)  | 82(80.39%) | 102   |
| TOTAL         | 70(35%)     | 130(65%)   | 200   |

X<sup>2</sup> - 41.96293902 p-0.000000040854

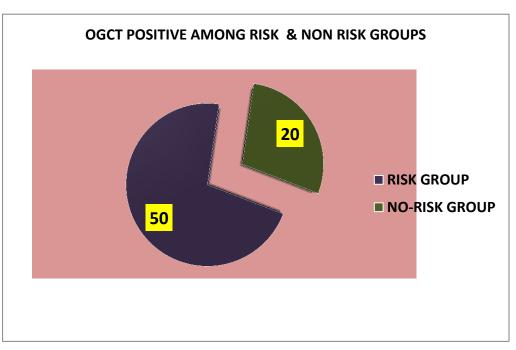
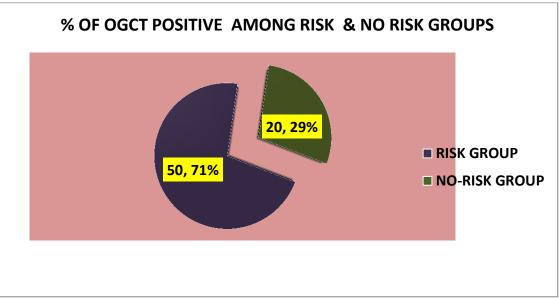


FIGURE 1

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FIGURE 2



# TABLE 4- PREDICTIVE VALUE OF RISK FACTORS

|                    |                        |                     | OGCT STATUS |          |       |
|--------------------|------------------------|---------------------|-------------|----------|-------|
|                    |                        |                     | OGCT +VE    | OGCT -VE | TOTAL |
| RISK GROUP         | FAMILY HISTORY         |                     | 35          | 15       | 50    |
| KISK GROUP         | BMI                    |                     | 10          | 30       | 40    |
|                    | FAMILY HISTORY -       | FAMILY HISTORY +BMI |             | 3        | 8     |
| NO-RISK GROUP      |                        |                     | 20          | 82       | 102   |
| TOTAL              |                        |                     | 70          | 130      | 200   |
| Risk Factors       |                        |                     | ·           |          |       |
| Family History     | +ve Predictive Value = | 70.00%              |             |          |       |
| BMI                | +ve Predictive Value = | 25.00%              |             |          |       |
| Family History+BMI | +ve Predictive Value   | 62.50%              |             |          |       |
| No Risk            | +ve Predictive Value = | 19.61%              |             |          |       |

FIGURE 3

| CLUSTERED BAR CHART SHOWING THE RISK & NON RISK GROUPS VERSUS<br>OGCT STATUS |          |          |         |  |  |
|--|----------|----------|---------|--|--|
|  |          |          |         |  |  |
| 0.0070   | OGCT +VE | OGCT -VE |         |  |  |
|  | OG DM    | STATUS   | TOTAL   |  |  |
| RISK GROUP FAMILY HISTORY  | 70.00%   | 30.00%   | 100.00% |  |  |
| RISK GROUP BMI   | 25.00%   | 75.00%   | 100.00% |  |  |
| RISK GROUP FAMILY HISTORY<br>+BMI  | 62.50%   | 37.50%   | 100.00% |  |  |
| NO-RISK GROUP  | 19.61%   | 80.39%   | 100.00% |  |  |
| TOTAL  | 35.00%   | 65.00%   | 100.00% |  |  |



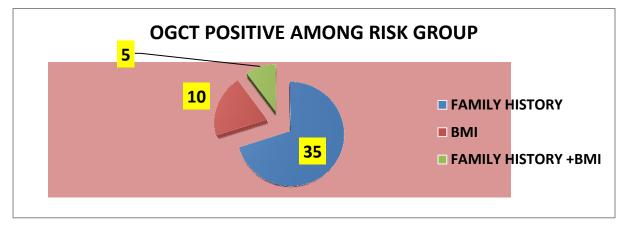
# PREDICTIVE VALUE OF FAMILY HISTORY AS RISK FACTOR FOR GDM

#### TABLE 5 - PREDICTIVE VALUE OF FAMILY HISTORY AND BMI

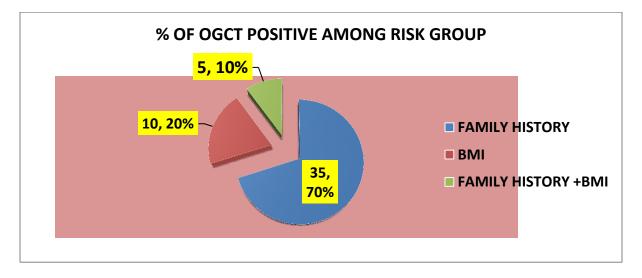
|                | OG DM    | TOTAL    |       |  |
|----------------|----------|----------|-------|--|
|                | OGCT +VE | OGCT -VE | TOTAL |  |
| FAMILY HISTORY | 35       | 15       | 50    |  |
| NO-RISK GROUP  | 20       | 82       | 102   |  |
| TOTAL          | 55       | 97       | 152   |  |

By Fisher's exact test 2 Tailed P-value:0.000000024

FIGURE 4









#### RESULTS

Out of the 200 primigravidae(<30 yrs) No risk with risk 102 98 Oget +ve oget -ve oget +ve oget -ve 20 82 50 48

Out of the 70 OGCT positive cases, 50(71.4%) had risks and

# 20(28.6%) did not have risk.

Thus, apart from positive family history and obesity, **ethnicity** plays a major role in GDM in our population.

#### DISCUSSION

Increasing maternal hyperglycemia is associated with increasing pregnancy morbidity and increased likelihood of subsequent diabetes in the mother. In addition, maternal hyperglycemia has a direct effect on the development of fetal pancreas and is associated with increased susceptibility to future diabetes in the infant, an effect which is independent of genetic factors [13,14]. Over the next two to three decades there will be 80 million reproductive age group women with diabetes in the world. Of these 20 million will live in India alone creatinga potential for extremely high rates of maternal and infant morbidity. With a huge population in the reproductive age in India, a significant segment developing abnormal glucose tolerance is a major concern.

Ethnically Indian women have high prevalence of diabetes and the relative risk of developing GDM in Indian women is 11.3 times compared to White ] [16], necessitating universal screening for glucose intolerance during pregnancy in India.

In Indian context screening is essential in all pregnant women as Indian women have a 11 fold increased risk of developing GDM compared to Caucasian women [26].

GDM diagnosis is overlooked in about 1/3rd of the women where selective rather than universal screening is performed [17] and when this is applied to the 20 million reproductive age women in India, we are missing a lot of women likely to have glucose intolerance.

The selective screening recommended by ADA is not suitable for our country and we need universal screening.

In our study 19.6% of no risk population had positive OGCT, which suggests the role of ethnicity and also stresses the need for universal screening [26]

In our study PPV for family history as risk factor was 70% and P value was 0.0000000024 which is statistically significant. It correlates with another study conducted in Iranian population by Khooshideh M et al in 2008.

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PPV of obesity as risk factor in our study was 30% and p value was 0.4 which is not statistically significant. This finding correlates with the above mentioned Iranian study. However further studies with screening of larger population are needed.

# CONCLUSION

- In our comparative study, family history is a significant risk factor for GDM which has a PPV of 70%
- Obesity as a predictive risk factor for GDM needs to be studied in a larger population group as it is not statistically significant in our study.
- Among 102 primigravidae without risk factors, 19.6% had OGCT positive. If universal screening is not done at 24-28 weeks, all these patients would have been missed.
- The other major etiology could be the ethnicity.

Hence universal screening for GDM is needed in all Indian women irrespective of risk factors.IADPSG also recommends the same. Local guidelines among Asian countries are increasingly moving to adopt universal screening.

Compared to selective screening, universal screening for GDM detects more cases and improves maternal and neonatal prognosis [4].

Hence universal screening for GDM plays a vital role for achieving the targets of MDG-4 & 5 and CSSM.

# REFERENCES

- [1] Seshiah V, Balaji V, Balaji MS. J Assoc Physicians India 2008;56:109-13.
- [2] Seshiah V, Balaji V, Balaji MS, et al. Int J Gynaecol Obstet 2009;104(Suppl 1):S35- 8.
- [3] www.idf.org/diabetesatlas
- [4] Cosson E. Screening and insulin sensitivity in gestational diabetes. Abstract volume of the 40th Annual Meeting of the EASD, September 2004;A 350.
- [5] Dornhorst A, Paterson CM, Nicholls JS, et al. Diabet Med 1992;9(9):820-5.
- [6] Alberti KG, Zimmet PZ. Diabet Med 1998;15(7):539-53.
- [7] Metzger BE, Gabbe SG, Persson B, et al. Diabetes Care 2010;33(3):676-82.
- [8] King H. Diabetes Care 1998; 21:B9–B13.
- [9] Obstet Gynecol 2013; 122: 406–416.
- [10] Teh WT, Teede HJ, Paul E, Harrison CL, Wallace EM, Allan C. Aust N Z J Obstet Gynaecol 2011; 51: 26– 30.
- [11] Yang H, Wei Y, Gao X, Xu X, Fan L, He J et al. Diabet Med 2009; 26: 1099–1104.
- [12] Morikawa M, Yamada T, Yamada T, Sato S, Cho K, Minakami H. Int J Gynaecol Obstet 2012; 118: 198– 201.
- [13] Dornhorst A, Beard RW. Diabet Med 1993;10:897-905.
- [14] Jarrett RJ. Diabet Med 1994;11:992-3.
- [15] Ramachandran A, Snehalatha C, Kapur A et al. Diabetologia 2001;44:1094-1101.
- [16] Dornhorst A, Paterson CM, Nicholls JS et al. Diabet Med 1992; 9: 820-5.
- [17] Cosson E, Benthimol M, Carbilon L et al. Universal screening for gestational diabetes mellitus improves maternal and fetal outcomes compared with selective screening. I:Mateclinsky FM (ed). Abstractbook of the 64th Scientific Sessions of the American Diabetes Association. Florida. American Diabetes Association 2004; A61.
- [18] Franks PW, Looker HC, Kobes S, et al. Diabetes 2006;55(2):460-5.
- [19] Anjalakshi C, Balaji V, Balaji MS, et al. Acta Diabetol 2009;46(1):51-4.
- [20] American Diabetes Association. Diabetes Care 2001;24(suppl 1):S77-S79.
- [21] Metzger BE, Coustan DR. Diabetes Care 1998;21(suppl 2):B161-B167.
- [22] Government of India, Ministry of Health and Family Welfare, NirmanBhavan, New Delhi (DO No. M-12015/93/2011-MCH/2011).
- [23] Ben-Haroush A, Yogev Y, Hod M. Diabet Med 2004;21(2):103-13.
- [24] Dornhorst A et al. 1992;9:820-5
- [25] BEISHER na et al. Diabetes 1991;40 suppl 2 :35-8
- [26] Khooshiedeh M, et al.SEM J 2008;9:1

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