Fasting C-peptide and Insulin Resistance/ Sensitivity in Gestational Diabetes Mellitus

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P206: Fasting C-peptide and Insulin Resistance/ Sensitivity in Gestational Diabetes Mellitus

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Abstract

Context:

Defective insulin secretion and/or insulin action are two characteristics of gestational diabetes mellitus (GDM) that are not settled and remains matter of controversy yet.

Objectives:

To see C-peptide and insulin indices in homeostatic model assessment (HOMA) in GDM.

Methods:

Glucose was measured by glucose oxidase, insulin by ELISA and C-peptide by immunochemiluminescent methods whereas insulin indices were calculated by using HOMA-IR, HOMA-B and HOMA-%S.

Patients:

This study encompassed 120 pregnant women irrespective of age of gestation divided into GDM (n=64, age: 27.02±4.26 years, BMI: 26.38±4.75 kg/m2; mean±SD) and normal glucose tolerance (NGT: n= 56, age: 26.11±4.13 years, BMI: 24.38±3.59 kg/m2; mean±SD) based on WHO 2013 criteria for diagnosis of GDM.

Intervention: Cross-sectional observational study

Main outcome measures: C-peptide and insulin indices

Results:

Out of 120, C-peptide was below the detection limit (<0.1 ng/ml) in 58 mothers. C-peptide insignificantly whereas fasting insulin significantly (10.57±1.09 vs. 7.68±0.56; p 0.039) were higher in GDM than those of NGT. Mothers having undetectable C-peptide had lower fasting plasma glucose than in mothers having detectable C-peptide (4.7±0.57 vs. 5.05±0.74 mmol/L; p= 0.005). HOMA-IR (GDM vs. NGT: 2.24±1.36 vs. 1.49±1.03; p 0.001) was higher whereas HOMA-B (GDM vs. NGT: 120.56±83.40 vs. 180.64±169.19; p 0.013) and HOMA-%S (GDM vs. NGT: 61.78±38.57 vs. 122.69±186.13; p= 0.012) lower in GDM than those of NGT. Neither C-peptide nor insulin differs significantly among trimesters within each group or between GDM and NGT (p ns for all comparisons). Circulating concentration of C-peptide (body mass index, BMI- <23 vs. ≥23 kg/m2: 0.185±0.06 vs. 0.331±0.44 ng/ml; p= .028) was significantly lower in GDM with low BMI. BMI showed positive correlation with fasting insulin in both GDM (r= 0.369; p= 0.019) and NGT (r= 0.412, p= 0.008) groups as well as with HOMA-IR (r 0.324; p= 0.041 and r = 0.415; p= 0.009 respectively). Analyses with covariates revealed BMI (p =0.007) and HOMA-B (p 0.06) as independent predictors over the glycemic abnormality.

Conclusions:

Fasting C-peptide did not differ between GDM and pregnancy NGT. However, insulin resistance was higher in GDM that was also true even when there was minimally undetectable secretion of C-peptide by pancreas in the face of abnormally higher glucose in GDM.

Introduction:

Insulin resistance is an essential component in the pathogenesis of gestational diabetes mellitus (GDM) but not enough alone without β-cells secretory defect. In comparison with the information on insulin resistance, concept on insulin secretion in GDM remains contradictory [1, 2].The aim of this study was to determine C-Peptide levels as a marker of insulin secretory capacity in GDM.

Materials and methods:

Study participants: In this observational cross-sectional pilot study, 120 pregnant women attending the 'GDM Clinic' of department of Endocrinology BSMMU, who fulfilled the inclusion and exclusion criteria, were screened and enrolled consecutively after having informed written consent during the period of March to July, 2018.

Operational definition: GDM was diagnosed according to WHO, 2013 criteria

Analytic methods: Glucose was measured by glucose oxidase method, insulin by ELISA and C-peptide by immunochemiluminescent methods whereas insulin indices were calculated by using HOMA-IR, HOMA-B and HOMA-%S.

Results:

Table 1: Baseline characteristics of the study population (N= 120)

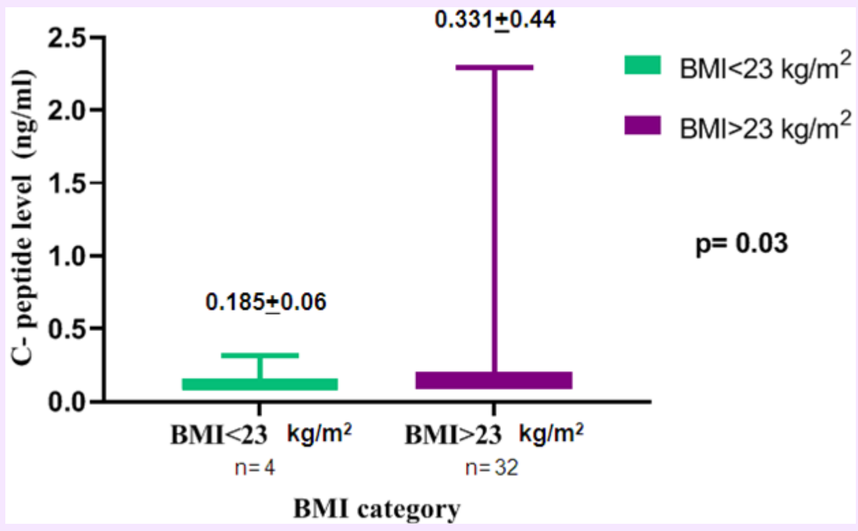
Variables	GDM	NGT	p
N	64	56	
Age (years, mean±SD)	27.02 ± 4.26	26.11 ±4.13	0.190*
BMI (kg/m^2, mean±SD)	26.38±4.75	24.38±3.59	**0.011***
Gravida, no. (%)			
Primigravida	23 (35.94)	34 (60.71)	0.160**
Multigravida	41 (64.06)	22 (39.29)	
SBP (mm Hg, mean±SD)	103.98±13.16	99.91±10.89	0.070*
DBP (mm Hg, mean±SD)	66.56±7.81	65.09±6.91	0.279*
History of abortion, no. (%)	16 (25.0)	7 (12.5)	0.083**
History of previous macrosomia, no. (%)	1 (1.56)	0 (0)	0.348**
Family history of DM in 1st degree relatives, no. (%)	18 (28.13)	20 (35.71)	0.373**

(Within parenthesis are percentages over column total)
*p-values were calculated using independent sample t-test
**p- values were calculated using Chi-square test
GDM: gestational diabetes mellitus; NGT: normal glucose tolerance
BMI: body mass index; DM: diabetes mellitus
SBP: systolic blood pressure; DBP: diastolic blood pressure

Table 2: Fasting C-peptide (N= 62*) and HOMA-IR (120) in GDM and NGT

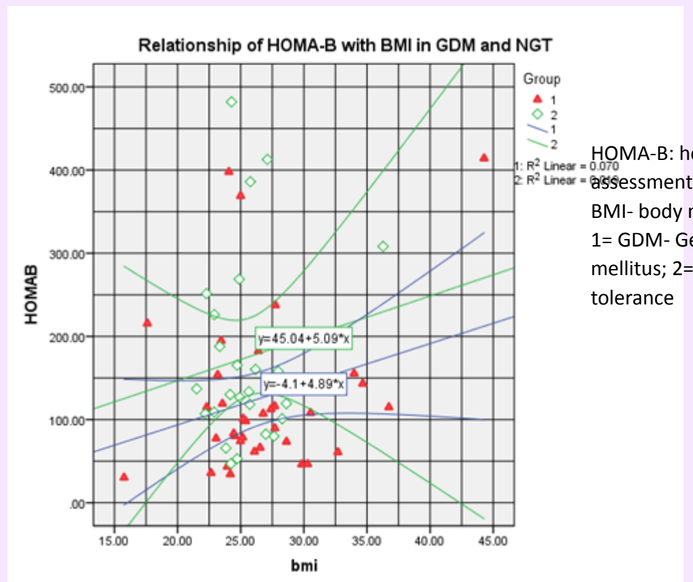
Variables	GDM	NGT	p
	Mean±SD		
C-peptide (ng/ml)	0.34±0.08 (n=36)	0.26±0.33 (n=26)	0.465
HOMA-IR	2.24±1.36 (n=64)	1.49±1.03 (n=56)	0.001

p-values were calculated using independent sample t-test
*C-peptide was undetectable (<0.1 ng/mi) in 58 (GDM- 28, NGT- 30) participants
GDM: gestational diabetes mellitus; NGT: normal glucose tolerance



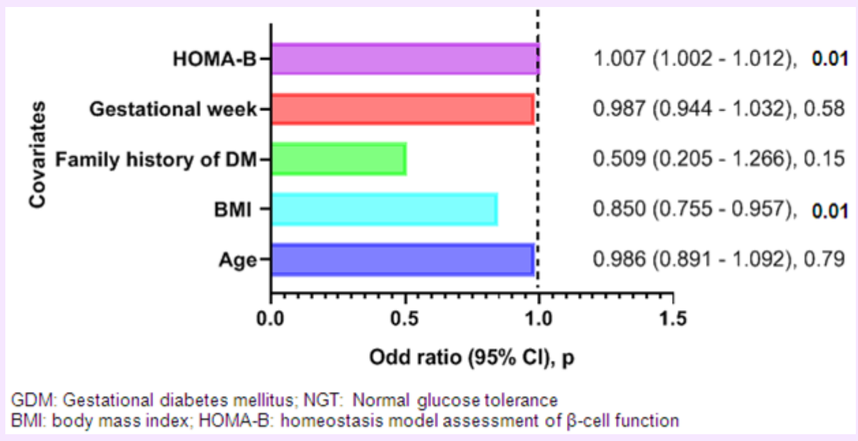
Comparison between groups done by Student's t test, GDM: gestational diabetes mellitus

Figure 1: C-peptide in GDM according to BMI categories (cut-off of 23 kg/m2) (N= 36)



HOMA-B: homeostasis model assessment of β-cell function; BMI- body mass index
1= GDM- Gestational diabetes mellitus; 2= NGT- Normal glucose tolerance

Figure 2: Relationship of HOMA-B with BMI in GDM and NGT



GDM: Gestational diabetes mellitus; NGT: Normal glucose tolerance
BMI: body mass index; HOMA-B: homeostasis model assessment of β-cell function

Figure 3: Multivariate binary logistic regression of GDM as dependent variable

Discussions and Conclusions:

Fasting C-peptide level did not differ significantly between women with GDM and pregnancy without glucose aberration despite a significantly higher HOMA-IR in GDM. Although both insulin resistance and insulin secretory defect were common in overweight women of GDM, decreased insulin secretory capacity seemed to be the primary defect causing GDM in women with low BMI. The β-cell response relative to the pregnancy induced insulin resistance was therefore unbalanced compared with the Western Europeans [3].

References:

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