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The Levels of Glucagon Like Peptide-1 in Gestational Diabetes Mellitus and Normal Pregnancy

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ABSTRACT

Objectives: Gestational diabetes mellitus (GDM) is a chronic hyperglycaemic condition occurring in pregnancy. The increased blood glucose level is due to dysfunction of beta cells of the pancreas associated with chronic insulin resistance. Glucagon-like peptide-1 (GLP-1), an incretin hormone, is involved in the process of insulin secretion from beta cells. It is proposed that defects in GLP-1 are related to both Type 2 diabetes mellitus and probably GDM. Thus, a study was planned to measure and compare fasting blood GLP-1 levels between GDM and normal pregnant females along with other anthropometric and blood parameters.

Method: Eighty-four pregnant females were divided into 3 groups (n=28 in each group), pregnant females with normal glucose tolerance (NGT), GDM for the first time (GDM-1), and GDM for the 2nd time (GDM-2). Anthropometric parameters, fasting blood glucose level, GLP-1, serum insulin level and HOMA-IR were assessed in all study subjects. Analysis was done by SPSS-20. One-way ANOVA was applied for comparison of variables among study groups.

Results: There was a significant difference ($p < 0.05$) regarding waist-to-hip ratio, fasting blood glucose level and HOMA-IR among groups, with an increase in both GDM-1 and GDM-2. There was a non-significant difference in fasting GLP-1 levels among the study groups ($p = 0.688$).

Conclusion: GLP-1 levels are not altered with deranged fasting blood glucose levels and HOMA-IR in GDM patients.

Keywords: Gestational diabetes mellitus, Glucagon Like Peptide-1, insulin resistance (HOMA-IR).

Authors' Contribution:
[1,2]Conception; Literature research; manuscript design and drafting; [3,4]Critical analysis and manuscript review; [5,6]Data analysis; Manuscript Editing.

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Introduction

Gestational diabetes mellitus (GDM) is glucose intolerance detected first time in pregnancy and that subsides after delivery. It is currently the most common medical complication of pregnancy.[1] Almost 5-30% of pregnancies are complicated by GDM depending on the criteria used to diagnose it.[2] Major risk factors for GDM includes, advanced maternal age, family history of type 2 Diabetes mellitus, maternal pre-pregnancy obesity, previous history of GDM etc[3]. Prevalence in Pakistan has been reported by a study conducted in Aga Khan Hospital to be continuously rising from 6.3% in 2005, to 9.86% in 2011 and in year 2018, it was 19%.[4] Pathophysiology of GDM is not completely understood but generally it involves either insufficient insulin release or increased peripheral insulin resistance.[5]

GDM is usually diagnosed on oral glucose tolerance test (OGTT). Usually, it is managed by life style modifications, dietary restrictions and insulin therapy.[6] Its management is quite important as GDM poses risks not only to mother but to neonate as well. It results in large for gestational age fetus and thus increased chances of caesarian delivery. Moreover, it increases the risk of future development of type 2 DM and other cardiovascular diseases in mother and in neonates, it increases the risk of postpartum hypoglycaemia and multiple birth defects.[7]

Glucagon like peptide (GLP-1), an incretin hormone, secreted from entero endocrine cells of intestinal mucosa in response to glucose intake. It acts on Beta cells of pancreas to increase the insulin secretion by activating a receptor (GLP-1R) and decrease the release of glucagon, the function that is impaired in patients of type 2 DM.[8] Role of GLP-1 in type 2 DM has been well studied and even GLP-1R agonists are now in practice as a treatment modality in Type 2DM.[9] While its role in GDM patients is not well established and few studies that have been done show controversial results. Impaired levels of GLP-1 may results in post prandial impaired glucose tolerance, seen in some GDM phenotypes.[10] Literature also suggests that GLP-1 secretion in GDM patients is not sufficient for their blood glucose levels during pregnancy and even in postpartum period.[11] Another study reports that postprandial GLP-1 responses are reduced in GDM as compared to normal pregnancy.[12] However, research data showing association of GLP-1 with deranged blood glucose profile in current and subsequent pregnancies is lacking around the globe in general and Pakistan in particular. Thus, the present study was planned to clarify the picture in our local GDM patients in order to improve overall health and quality of life in both neonates and mothers.

Methodology

A cross-sectional comparative study was conducted in the Physiology department of the University of Health Sciences, Lahore. The sample size was calculated by given formula:

$$n_1 = \frac{(Z_{1-\beta} + Z_{1-\alpha/2})^2(\sigma_1^2 + \sigma_2^2)}{(\mu_1 - \mu_2)^2}$$

A total of 84 pregnant females were recruited from the Gynaecology Departments of Jinnah Hospital, Lahore by using purposive sampling technique and divided into three groups NGT (Normal glucose tolerant pregnant females), GDM-1 (pregnant females diagnosed as GDM in current pregnancy) and GDM-2 (females who developed GDM in current as well as previous pregnancy). A sample size was 28 subjects in each group. All pregnant females were age (20-35 years) and BMI (18.5-24.9kg/m^2) matched, at 24-32 weeks of gestation. GDM was diagnosed by 100 g oral glucose tolerance test (OGTT), with cut-off values for glucose defined as 105, 190, 164 and 145 mg/dl for fasting and after 1, 2 and 3 hours after glucose intake respectively. If two or more serum glucose values are more than cut off values, then GDM was diagnosed. Cut off values were taken from National Diabetes Data Group Conversion, ACOG Practice Bulletin 30.[13]

Pregnant females with Diagnosed type 1 and 2 diabetes and other endocrinopathies such as Polycystic Ovarian syndrome, Cushing syndrome, obesity, those using corticosteroids or drugs affecting thyroid and pancreatic hormones and conceived after ovulation induction were excluded from study. Written informed consent was taken from all selected subjects. History was taken along with relevant clinical information. Anthropometric parameters like weight, height, BMI, waist hip ratio, sub scapular and triceps skin fold thickness, mid arm circumference. All measurements were made according to the protocols mentioned in National Health and Nutrition Examination Survey (NHANES) Anthropometry procedures manual 2009.[14] All instruments were standardized before examination and calibrated as zero.

5 ml of fasting venous blood was drawn by using aseptic technique. 2ml was secured in non-coated

gel vacutainer for serum insulin measurement, remaining 3 ml was put in pre chilled approtinin and ETDA coated vacutainer for GLP-1 measurement. Blood was centrifuged at 2000 rpm for 15 min, serum was stored at -80 ◦C. GLP-1 was measured using commercially available ELISA kits manufactured by Glory Science Co. Ltd. 2400 Veterans Blvd. Suite 16 -101, Del Rio, TX 78840, USA. Insulin levels were measured using commercially available ELISA kit manufactured by Perfemed Group, Inc.385 Oyster Point Blvd Suite5-6., South San Francisco, USA. Normal reference range for GLP-1 concentrations was taken between 5 and 15 pmol /L in the basal state and insulin was 9.68 ± 2.76 (µIU/ml). Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated by the following formula.[15] HOMA-IR=Fasting insulin (µIU/ml) × Fasting glucose (mmol/L)/22.5. Cut off value of HOMA-IR for GDM was 2.60.[1]

Data was entered and analyzed using IBM SPSS version 20.0. Normality of the data was checked by Shapiro-Wilk's test. Mean ± SD was given for normally distributed quantitative variables and Median with IQR was given for non-normally distributed quantitative variables. One-Way ANOVA was used to compare the means between more than two groups and Post-hoc Tukey's test was applied to compare every group mean with every other group mean. Pearson correlation (r) was used to observe correlation between quantitative variables. A p-value of ≤ 0.05 was considered statistically significant.

Ethical approval was taken from the institutional ethical review board of University of Health Sciences, Lahore.

Results

Study population consisted of 28 NTG, 28 GDM-1 and 28 GDM-2 pregnant females. General characteristics are given in table 1. There was a statistically significant difference (p=0.001) in gestational age among groups, with higher age in GDM-2 and GDM-1.

Table I: Comparison of general characteristics of study groups (ANOVA) (n=28)

Parameter	NGT	GDM-1	GDM-2	p-value
	Mean ± SD	Mean ± SD	Mean ± SD	
	Median (IQR)	Median (IQR)	Median (IQR)	
Age (years)	26.76 ± 4.54	27.87 ± 4.40	30.80 ± 3.54	0.002*[b]
	25.50 (25.01–30.01)	26.50 (24.01–30.01)	30.00 (30.01–32.76)	
Gest. Age (wks)	28.62 ± 2.75	31.12 ± 3.61	31.58 ± 2.75	0.001*[b]
	28.00 (27.01–29.76)	32.00 (28.26–34.01)	32.00 (30.01–34.01)	
Pre-Pregnancy BMI (kg/m²)	23.85 ± 2.88	24.04 ± 3.69	24.19 ± 3.35	0.774[b]
	24.20 (21.53–25.61)	22.70 (21.31–25.98)	24.40 (22.01–27.16)	

erum insulin levels showed no significant difference between NGT and GDM-1 and GDM-2 (p = 0.210). When HOMA-IR was compared among the groups, the results showed a statistically significant difference (p=0.001) among groups, with higher HOMA-IR of GDM-1 and GDM-2 as compared to NGT, but no significant difference seen in GDM-1 and GDM-2 (p=1.000). GLP-1 of NGT group was 7.66±2.34 pmol/L, in GLP-1 of group GDM-1 was 8.02(5.98-9.12) and median IQR of GLP-2 group was 7.55 (5.95-10.33) pmol/L. There was no significant difference (p = 0.688) in GLP-1 among groups. Comparison of parameters among NGT and GDM-1 showed significant difference of WHR, blood glucose levels and HOMA IR, with high values of these parameters seen in GDM-1 as compared to NGT (table 4), similarly theses three parameters also show significant difference in NGT and GDM-2 groups (table 5).

Table II: Comparison of Anthropometric variables among study groups (ANOVA) (n=28)				
Parameter	**NGT**	**GDM-1**	**GDM-2**	***p*-value**
	Mean ± SD	**Mean ± SD**	**Mean ± SD**	
	Median (IQR)	**Median (IQR)**	**Median (IQR)**	
Height (cm)	158.86 ± 7.06	155.86 ± 9.60	160.97 ± 7.04	0.023*[b]
	159.00 (155.01-163.01)	156.50 (153.01-161.51)	162.50 (158.01-165.01)	
Weight (kg)	70.15 ± 8.29	69.04 ± 9.40	74.40 ± 9.51	0.072[a]
	71.00 (68.01-74.76)	69.00 (61.26-74.01)	73.50 (67.26-81.26)	
Current BMI (kg/m²)	27.86 ± 3.47	28.57 ± 4.51	28.54 ± 3.52	0.745[b]
	27.95 (25.08-29.41)	27.40 (25.33-30.73)	28.30 (26.03-31.48)	
WHR	0.98 ± 0.05	1.036 ± 0.08	1.04 ± 0.07	<0.001*[b]
	0.97 (0.96-1.01)	1.05 (0.98-1.11)	1.02 (1.01-1.09)	
Mid arm circumference (cm)	29.58 ± 3.42	29.58 ± 3.77	30.80 ± 3.94	0.373[a]
	29.00 (27.01-32.01)	29.00 (27.26-32.76)	31.50 (27.26-33.01)	
Sub scapular skinfold Thickness (mm)	21.62 ± 4.83	21.90 ± 5.21	23.76 ± 6.16	0.585[b]
	22.00 (19.26-24.01)	22.50 (18.01-25.76)	21.50 (20.01-28.01)	
Triceps skin fold thickness (mm)	17.01 ± 4.074	16.76 ± 3.76	18.01 ± 4.70	0.808[b]
	17.00 (13.51-19.01)	17.00 (15.01-19.01)	17.00 (15.01-20.01)	

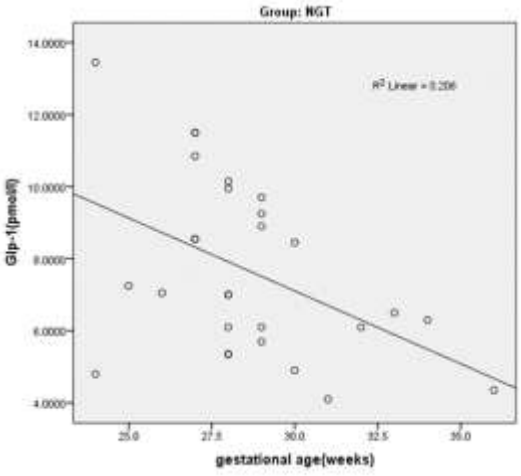

Figure 1: Scatter plot showing significant correlation of GLP-1 with gestational age in NGT group (p =0.015).

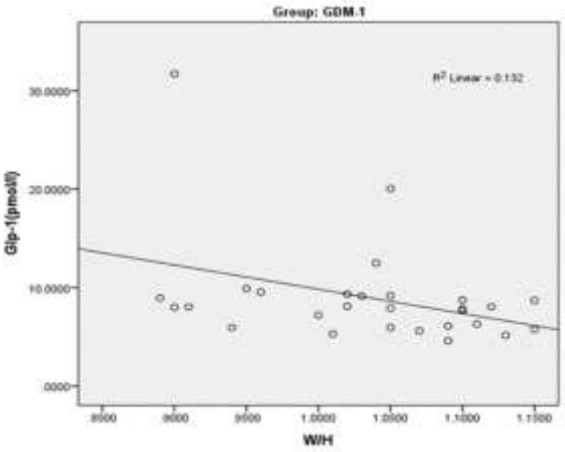

Figure 2: Scatter plot showing significant correlation of GLP-1 with WHR in GDM-1 group.

Table III: Comparison of biochemical parameters in study groups (ANOVA) (n=28)				
Parameters	**NGT**	**GDM-1**	**GDM-2**	**p-value**
	Mean ± SD Median(IQR)	Mean ± SD Median(IQR)	Mean ± SD Median(IQR)	
FBG (mg/dl)	86.12 ± 14.17 86.00 (80.01-92.01)	117.72 ± 37.21 109.50 (98.26-122.76)	129.65 ± 41.20 122.00 (96.51-162.51)	<0.001*[b]
Insulin (µIU/ml)	13.99 ± 4.31 13.49 (10.72-17.32)	18.62 ± 10.49 16.66 (10.96-23.75)	15.88 ± 9.77 14.98 (10.67-18.01)	0.210[b]
GLP-1 (pmol/L)	7.67 ± 2.45 7.02 (5.81-9.59)	8.98 ± 5.33 8.02 (5.99-9.19)	10.47 ± 9.48 7.55 (5.96-10.34)	0.688[b]
HOMA-IR	2.99 ± 1.12 2.66 (2.20-3.93)	5.64 ± 4.21 5.01 (3.25-6.02)	5.10 ± 3.31 3.97 (2.75-7.09)	0.001*[b]

Table IV: Comparison of parameters between NGT and GDM-1 by Tukey test. (n=28)			
Parameters	**NGT**	**GDM-1**	***p*-value**
	Mean ± SD **Median(IQR)**	**Mean ± SD** **Median(IQR)**	
WHR	0.98 ± 0.05 0.97 (0.96-1.01)	1.036 ± 0.08 1.05 (0.98-1.11)	0.002*[b]
FBG (mg/dl)	86.12 ± 14.17 86.00 (80.01-92.01)	117.72 ± 37.21 109.50 (98.26-122.76)	<0.001*[b]
HOMA-IR	2.99 ± 1.12 2.66 (2.20-3.93)	5.64 ± 4.21 5.01 (3.25-6.02)	0.002*[b]

Table V: Comparison of parameters between NGT and GDM-2 by Tukey test. (n=28)			
Parameters	**NGT**	**GDM-2**	***p*-value**
	Mean ± SD **Median (IQR)**	**Mean ± SD** **Median (IQR)**	
WHR	0.98 ± 0.05 0.97 (0.96-1.01)	1.04 ± 0.07 1.02 (1.01-1.09)	0.001*[b]
FBG (mg/dl)	86.12 ± 14.17 86.00 (80.01-92.01)	129.65 ± 41.20 122.00 (96.51-162.51)	<0.001*[b]
HOMA-IR	2.99 ± 1.12 2.66 (2.20-3.93)	5.10 ± 3.31 3.97 (2.75-7.09)	0.015*[b]

No difference was seen in GDM-1 and GDM-2. Correlation was assessed among GLP-1 and different anthropometric parameters among groups by Spearman correlation. The result showed a significant negative correlation of GLP-1 with gestational age in NGT group (Spearman's rho = -0.454, p = 0.015) as shown in figure 1 and significant negative correlation of GLP-1 also observed with waist to hip ratio in GDM-1(Spearman's rho = -0.390, p = 0.040) as shown in figure 2. No correlation of GLP-1 was seen with any anthropometric parameter in GDM-2.

Discussion

The current study was conducted on 84 pregnant women, 28 normal pregnant, 28 with GDM for first time and 28 those suffering second time with GDM. Anthropometric parameters and blood parameters that include, fasting blood glucose level, serum insulin, GLP-1 and HOMA- IR were measured in all studied subjects. The general characteristics of study subjects showed a significant difference of maternal age in groups, with higher maternal age observed in GDM-1 and GDM-2. Literature has shown that GDM was associated with higher maternal age and the risk of GDM was significantly and progressively increased from 30 years of age onwards and maternal age ≥35 years are predictive factor of GDM.[16]

Regarding anthropometric parameters, results of current study showed a significant difference in waist to hip ratio, with more WHR in GDM-1 and GDM-2 as compared to NGT females. No significant difference was observed in other studied parameters such as weight, BMI, triceps skin fold thickness, subscapular skin fold thickness and mid arm circumference. The study conducted by Xingtong et. al. in 2022 showed a significant difference of WHR in normal pregnant females and those of GDM and concluded that higher WHR is a risk factor for development of GDM as WHR has been identified as a tool for detecting central obesity that is a key factor in developing GDM.[17,18] Regarding weight and BMI, the literature showed conflicting results with some studies suggest that there is no significant difference in weight gain or BMI between GDM and normal pregnant females.[19] However, other studies have found that normal weight women with GDM may have a higher risk of adverse outcomes compared to overweight counterparts.[20] One study found that BMI in early pregnancy was a risk factor for GDM, but BMI gain before GDM screening was not associated with the risk of GDM.[21]

When compared to normal pregnant females, the fasting blood glucose levels in GDM-1 and GDM-2 were considerably higher. These findings are consistent with the research done by Stewart and Murphy, who found that elevated blood glucose levels represent a separate risk factor for GDM.[22] GDM-2 had greater blood glucose levels than GDM-1, although the differences in the results were not statistically significant. The current study found no significant differences in insulin levels between NGT, GDM-1, and GDM-2 (p = 0.210). There are similarities between current work and those of Telejko et al.[23] This could be because normal pregnant women usually have hyperinsulinemia later in their pregnancy. Conversely, we found that GDM-1 exhibited higher levels of insulin than GDM-2. The decreased insulin levels in GDM-2 may be explained by the body starting to adapt for hyperinsulinemia in subsequent pregnancies, or by pregnant women learning to change their diet and lifestyle to avoid complications from GDM in their later pregnancies.

As far as, HOMA-IR is concerned, insulin resistance normally occurs in pregnancy but it may lead to hyperinsulinemia and development of GDM. Current study found higher insulin resistance in GDM-1 and GDM-2 as compared to NGT. However, in both diabetic groups, there was no significant difference in results for insulin resistance. Many studies have proved the development of insulin resistance in GDM.[24]

Regarding the levels of GLP-1, the current study found no significant difference in fasting GLP-1 levels in study groups. The GLP-1 levels of NGT, GDM-1 and GDM-2 were similar. Only a few studies investigated the levels of GLP-1 in GDM subjects and reported contrasting results. Some studies found fasting levels of GLP-1 while others found the levels after OGTT. One of the studies conducted by Lencioni et.al., found reduced GLP-1 secretion as compared to the controls during OGTT.[11] The population in their study was weight and BMI matched as in our study. Reduced GLP-1 response during OGTT has also been found by Sukumar et.al, these subjects also showed no significant difference in HOMA-IR as well.[25] Bonde et.al. measured post prandial response to GLP-1 in GDM subjects and showed no difference in GLP-1 response in GDM and normal pregnant females.[12] Mosavat et.al., found lower fasting GLP-1 levels in GDM subjects as compared to normal pregnant females and concluded that lower GLP-1 levels are independent risk factor for development of GDM.[26] Contrarily, Fristche et.al., found increased GLP-1 secretion during OGTT in BMI matched GDM subjects as compared to normal pregnant females.[27] The author explained this enhanced response of GLP-1 in GDM as a part of compensatory response to GLP-1 resistance in GDM patients. Most recent study conducted by krystynik et.al., in 2023,[28] show resemblance to our study with the findings that GLP-1 levels after OGTT (our levels were fasting) were unaltered in GDM (n= 24) and normal pregnant females (n=24) although GDM patient showed impaired fasting blood glucose levels in comparison to normal pregnant females and concluded that women with impaired fasting blood glucose levels did not show altered incretin (GLP-1) secretion in early pregnancy and thus impaired incretin production has no major role in GDM development as it has a major role in type 2 diabetes. Correlational analysis of GLP-1 levels with various anthropometric parameters in three studied groups showed a statistically significant negative correlation of GLP-1 with gestational age in normal pregnant females and significant negative correlation with waist to hip ratio in GDM-1 females. No correlation with any anthropometric parameters observed in GDM-2 females. This negative correlation of GLP-1 with waist to hip ratio suggests obesity as a risk factor for GDM development. Interestingly, no such correlation was found in available literature and these results are also novel in our own population demanding the need of more studies on large sample size.

Conclusion

GLP-1 levels are not significantly different between GDM and normal pregnant females. GDM patients were elder than normal pregnant females and showed more waist to hip ratio and increased insulin resistance i.e. increased HMOA-IR. Thus, fasting GLP-1 levels does not seem to play a major role in development of GDM.

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