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Original Article

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GLUT-2 and Plasma Insulin Concentrations in Women with and without GDM

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Abstract

Objective: To determine GLUT-2 and plasma insulin concentrations in women with and without gestational diabetes. ***Design:*** A prospective longitudinal cohort study of Mexicans. ***Subjects/Patients:*** included pregnant patients aged 18-45 years with normal pregnancy and gestational diabetes, with gestation>24 weeks. ***Methods:*** Sociodemographic characteristics, gestational risk factors, and baseline and final measurements of anthropometrics, glucose, insulin, lipid, and GLUT-2 were recorded. The Mann-Whitney U test was used, 95%CI and p≤0.05 were considered, SPSS software was used vs25, approved by the Ethics and Research Committee (code: 2022-08-799). ***Results:*** A sample of 33 participants was analysed, 76.5% comprised the control group and 23.5% the GDM group, baseline and final clinical characteristics of the participants increased in the gestational diabetes group: GLUT-2 (28. 9±13.1 ng/mL vs 37.6±9.2 ng/mL, p=0.035) and insulin (33.8±11.1 µIU/mL vs 37.6±5.1 µIU/mL, p=0.017), both groups had hypercholesterolemia, and insulin resistance in the gestational diabetes group. ***Conclusion:*** The gestational diabetes group had higher GLUT-2 and insulin concentrations, as well as risk factors associated with pregnancy, suggesting that GLUT-2 measurement represents an alternative for better metabolic control and prevention of the development of this pathology.

Keywords: ***Gestational Diabetes Mellitus, GLUT-2, insulin, metabolism, pregnancy.***

Introduction

Gestational diabetes mellitus (GDM) is an endocrine disorder of multifactorial origin, characterized by hyperglycemia that develops during pregnancy [1]. Current global prevalence estimates range from 1% to 30% of pregnancies, affecting more than 20 million live births worldwide [2]. In Mexico, the prevalence of GDM is 13.7%, which is particularly worrying because it is a potent risk factor for complications during pregnancy [3-9]. During gestation, glucose is the basic energy substrate for fetal development; thus, maternal-fetal glucose transport and, uteroplacental blood flow represent the most studied factors in gestational development, GDM metabolism and, intrauterine growth restriction [10].

The determinants of placental glucose transport capacity are the maternal-fetal glucose concentration gradient, blood flow and, the expression of specific glucose transporters (GLUT) [11]. GLUTs are known to be active in the pathogenesis of various pregnancy complications, in particular those associated with intrauterine metabolic disorders, and, exhibit different kinetic characteristics depending on organ expression, cellular localization and, substrate specificity [12].

GLUTs allow the facilitated diffusion of glucose to the plasma membrane of skeletal muscle cells, adipocytes, hepatocytes, placenta and, microvilli, producing a constant influx of glucose, thus providing a hypoglycaemic effect of insulin that allows the regulation of glucose levels [13].

GLUT-2 is expressed in pancreatic, hepatic, renal, intestinal and, astrocyte cell membranes and, mediates the transport of glucose, fructose, galactose and, glucosamine [14]. Because of its low affinity for glucose, it acts as a sensor of glucose levels, allowing glucose to enter when elevated in plasma and, then significantly releasing insulin into the bloodstream, physiologically controlling fluctuations in blood glucose [15].

Thus, glucose concentration is the key regulator of insulin secretion. To activate a glucose molecule, it must be transported by GLUT-2 into the β-cell, phosphorylated by the enzyme glucokinase and, metabolized [16]. The molecular mechanisms of insulin secretion are initiated by the detection of glucose in the blood and, entry into pancreatic cells by GLUT-2 [17].

After food intake, the liver can incorporate glucose through GLUT-2 to rapidly convert it into glycogen, conversely, during the late postprandial period (period between 6 - 8 hours of fasting), glycogen is degraded to form glucose molecules, which leave the liver cell into the systemic circulation, preserving physiological glucose values [14]. Therefore, GLUT-2s are bidirectional

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transporters that can conduct glucose from blood to tissue or from tissue to blood, as required [18,19].

The available data on the gestational development of GLUTs are relatively scarce, due to cost, tissue to be studied, and population it can be stated that GLUT-2 expression and GDM in humans is almost null. Given the above, the present study aimed to determine the concentrations of GLUT-2 and plasma insulin in women with and, without GDM, as a metabolic indicator of one of the most prevalent pathologies during gestation. The available data on the gestational development of GLUTs are relatively scarce, due to cost, tissue to be studied, and population it can be stated that GLUT-2 expression and GDM in humans is almost null. Given the above, the present study aimed to determine the concentrations of GLUT-2 and plasma insulin in women with and, without GDM, as a metabolic indicator of one of the most prevalent pathologies during gestation.

Methods

Prospective longitudinal cohort study, conducted at the Hospital Materno Perinatal 'Monica Pretelini Saenz' (HMPMPS), in Toluca Mexico. Pregnant patients aged 18 to 45 years were included. They were organized into groups according to the presence or absence of GDM (controls) and, both groups with pregnancy >24 SDG. Baseline and, final biochemical and anthropometric measurements were recorded, with a four-week interval between each measurement. Pregnant patients with diagnoses of DM1, DM2, carbohydrate intolerance, <18 years and <24 SDG were excluded. Patients were chosen at convenience by stratified sampling.

Anthropometric measures

Pregestational weight and, height was measured with a calibrated adult scale (SECA®, Germany). All patients had their pregestational Body Mass Index (BMIpreg) calculated, classified as follows: underweight <18.5, normal weight 18.5-24.9, overweight 25.0-29.9, obese >30 [20]. To determine fat mass, skinfold measurements were considered (bicipital, tricipital, subscapular and, suprailiac), the personnel involved in the measurements were ISAK level 1 certified, and a pharmacobiological chemist was available to take blood samples.

Sociodemographic data and risk factors associated with pregnancy were recorded:

personal and family pathological history: DM2, hypertension, obesity, cancer, dyslipidemias, [21,22] advanced maternal age ≥5 years, [23] number of pregnancies: multiparous, [24] miscarriages, [21] macrosomic children >4000g, [22] resolution of current pregnancy cesarean section, [22] smoking, alcoholism, [25] physical activity <150 min per week, [26] marital status: single or "unmarried", [27] and age at menarche [28] using a Google Forms questionnaire.

Measurement of biochemical parameters

After eight hours of fasting, blood samples were taken. Glucose analyses (mg/dL) were performed (Atellica CH Glucose Hexokinase_3 (GluH_3), ref. 11097592), the parameters to be considered were normal fasting blood glucose ≤ 126 mg/dL; diabetes ≥ 126 mg/dL (7 mmol/L) [29].

Cholesterol (mg/dL) (Atellica CH Cholesterol_2 (Chol_2), ref. 11097609), triglycerides (mg/dL) (Atellica CH Triglycerides (concentrated) (Trig), ref. 11097591), (Dimension R × L Max, Dade Behring, USA), blood cytometry (Advia 120, Bayer Health) and HDL-c; LDL-c analysis (Atellica Direct HDL Cholesterol (D-HDL), ref. 11097630). Dyslipidaemia was defined as elevated cholesterol (>180 mg/dL) or triglycerides (>170 mg/dL) [30,31].

Quantification of insulin and GLUT-2

Human Glucose Transporter-2, (GLUT2) Cat. EH3146 (Fine Test) (China) and Insulin Cat. ME E-0900, (LDN) (China) was measured by ELISA on the ELx800™ PrimeQ® kit at the Ciprés Grupo Médico S.C. (CGM) Research Laboratory. GLUT-2 concentrations are not reported; normal insulin values 5-25 U/ml, and insulin resistance >30 U/ml [32].

Statistical analysis

Data was processed using the statistical package SPSS version 25 (Armonk, New York, USA). Descriptive statistics were used: quantitative variables were represented by measures of central tendency. The Kolmogorov test was used to determine the normality of variables, and the Mann-Whitney U test was used to compare quantitative variables between the two groups. Spearman's test with a 95% confidence interval (95%CI) was used to assess correlations. Values of p <0.05 were considered significant.

Results

The sample consisted of 33 participants, of which 72.7% corresponded to the control group and 23.5% to the GDM group. The mean age for the first group was 27±8.4 years and for the second group 38.75±5.8 years, with a similar gestational age (28.4±3.4 vs 27±4.1). Table 1 shows the general characteristics of the sample studied, the predominant marital status in both groups was single, and personal diseases in the control group prevailed overweight and/or obesity (11.5%). However, when obtaining the BMIpreg, the data were higher in the GDM group (34.6% overweight and/or obese in the control group vs. 75% in the GDM group), and 37.5% in the control group reported having more than one comorbidity. The most frequent hereditary disease was DM2 for both groups (19.2% vs. 37.5%).

In table 2, it can be observed that the baseline and final clinical characteristics of the participants increased, being significant in the control group the concentrations of total cholesterol (188.5±12.6 mg/dL vs 199.5±57.2 mg/dL, p=0. 017) and triglycerides (269.1±55.8 mg/dL vs 289.2±106.1 mg/dL), in contrast to the GDM group clinical conditions are higher with significant data GLUT-2 (28.9±13.1 ng/mL vs 37.6±9.2 ng/mL, p=0.035), insulin (33.8±11.1 µIU/mL vs 37.6±5.1 µIU/mL, p=0.017). It should be noted that both groups had hypercholesterolemia (>180 mg/dL), hypertriglyceridemia (>170 mg/dL), and insulin resistance (>30 U/ml) in the GDM group.

Spearman's test showed that insulin was positively correlated with triglyceride (r2=0.571, p<0.05), GLUT-2 (r2=0.409, p<0.05), in the control group, while basal GLUT-2 was positively correlated with HDL-c (r2=0.790, p<0.05) in the GDM group.

Table 1: General characteristics of participants

Variable	CONTROL 76.5%	GDM 23.5%	Value p
Age (mean±DS)	27±8.4	38.75±5.1	0.500
Gestional age (GA)	28.4±3.4	27±4.1	0.270
Age at first pregnancy (years)	22.19±5.9	24.13±6.8	0.550
Marital status (%)			
Married or in union	38.5	37.5	0.961

Single	61.5	62.5	1.000
Number of pregnancies	2	3	0.990
Parity (%)			
Primigestation	53.8	25	0.154
Multigestation	46.2	75	0.233
History of miscarriage (%)			
Yes	7.7	75	0.229
No	92.3	25	0.219
Resolution of previous pregnancies			
Natural childbirth	6	3	0.500
Caesarean section	7	1	0.500
History of offspring with fetal macrosomia			
Yes	-	8	0.980
No	26	-	0.970
Personal diseases (%)			0.000
Overweight / obesity	11.5	-	
Diabetes Mellitus	-	50	
Hypertension	3.8	-	
Preeclampsia	3.8	-	
Dyslipidemia	3.8	-	
More than 1	3.8	37.5	
None	73.1	12.5	
Family diseases (%)			
Overweight / obesity	7.7	12.5	
Diabetes	19.2	37.5	
HBP	7.7	12.5	0.719
Preeclampsia	-	-	
Dyslipidemia	-	-	
More than 1	15.4	12.5	
None	50	25	
Smoking (%)			
Ex-smoker	11.5	25	0.570
Never smoked	88.5	75	0.334
Sitting time in a day			
Less than 25 minutes a day	30.8	-	
30 minutes a day	26.9	12.5	
Between 30-60 minutes	26.9	25	0.879
More than one hour a day	15.4	62.5	
BMIpreg (kg/m^2)			
Normal	65.4	25	0.047*
Overweight	26.9	75	0.070
Obese	7.7	-	0.260

*Abbreviation: WG: weeks of gestation, HT: Hypertension, BMIpreg: Pregestational Body Mass Index. Data are expressed in %, BMI mean ± SD. *P < 0.05 statistically significant.*

Table 2: Baseline and final clinical characteristics of participants by study group

Variable	**CONTROL**			**GDM**		
(media±DS)	**BASELIN**	**FINAL**	***Valor p***	**BASELINE**	**FINAL**	***Valor p***
Glucose mg/dL	74.5±11.8	75.2±10.6	0.051	87.0±15.4	87.1±18.3	0.085
TC mg/dL	188.5±12.6	199.5±57.2	0.017*	230.8±37.4	230.3±44.9	0.130
c-HDL mg/dL	67.5±15.6	67±14.4	0.035*	54.2±12.6	54.8±7.4	0.031*
c-LDL mg/dL	124.2±35.8	146.2±69.3	0.084	154.1±37.4	158.1±46.6	0.327
TG mg/dL	269.1±55.8	289.2±106.1	0.858	277.3±97.1	353.2±179.4	0.413
Lipid-related risk factor	2.4±0.6	2.4±0.5	0.827	2.3±0.6	2.4±0.6	0.858
GLUT-2 (ng/mL)	24.4±11.7	28.2±11.7	0.618	28.9±13.1	37.6±9.2	0.035*
Insuline (µUI/mL)	27.3±24.7	28.4±14.3	0.347	33.8±11.1	37.6±5.1	0.017*
Body fat mass (kg)	34±7.6	43±7.2	0.771	23.9±6.8	38±9.2	0.048*
Lean body mass (kg)	29.5±7.9	25.7±4.8	0.879	48.4±10.3	27.7±8.4	0.030*

*Abbreviation: TC: total cholesterol, c-HDL: high-density lipoproteins, c-LDL: low-density lipoproteins, TG: triglycerides, GLUT-2: glucose transporter family 2, member 2. Data are expressed as Mean ± SD. *P < 0.05 statistically significant.*

Discussion

GDM is an endocrine disruption that develops during pregnancy, [1] worldwide affects up to 30% of pregnancies, increases maternal and fetal morbi-mortality [7] due to hyperglycemia is the cause of micro- and macrovascular complications, [8,9] during gestation GLUTs are involved in glucose transport in the placenta [11] when overexpression is present, they show activity in the pathogenesis of intrauterine metabolic disorders. [12].

GLUT-2 act as sensors of blood glucose levels, allowing the entry of glucose when it is elevated in plasma and then releasing insulin into the bloodstream, physiologically controlling fluctuations in blood glucose [15]. In the context of GDM, GLUT-2 expression in pancreatic β-cells contributes to impaired insulin secretion and glucose intolerance [33]. While GLUT-2 has not been studied as extensively in the context of GDM compared to other GLUT isoforms (GLUT-1 and GLUT-4), making it imperative to understand its role in glucose metabolism, during pregnancy, changes in hepatic glucose metabolism, influenced by factors such as insulin resistance and hormonal fluctuations, may affect GLUT-2 expression and activity, potentially contributing to glucose dysregulation in GDM [34].

In this study, we explored GLUT-2 and plasma insulin concentrations in women with and without GDM as a metabolic marker during gestation. The GDM group showed increased GLUT-2 concentration, recently, studies by Kuan Jiang Y et al. revealed that GLUT2 expression was higher in the GDM group ($p=<0.05$) demonstrating the potential role of GLUT2 in the pathogenesis of gestational diabetes [35].

When pancreatic ß-cells dysfunctional and fail to meet the body's insulin needs, it results in hyperglycemia with inflammation in organs [36].

In the case of impaired GLUT2 function, it fails to transfer insulin from the intracellular cytoplasm to the membrane [37] may promote hyperglycemia. In animal studies, a lack of GLUT2 showed high blood glucose levels [38].

Similarly, placental adipokine-induced insulin resistance and hormone secretion contribute to the development of GDM [39] At the renal level, the physiological decrease in the threshold for glucose in a recent study suggested that it depends on the renal glucose reabsorption capacity associated with GLUT2 expression, which was shown to be increased in GDM with hyperglycemia, [40] which may lead to increased renal glucose reabsorption in patients with diabetes.

Differently, one study showed that the threshold for renal glucose in pregnancy with normal glucose tolerance was significantly lower than in GDM, [41] In cases with GDM it may confer the up-regulation of GLUT2, therefore, our findings, in patients with GDM suggest that GLUT2 expression is significantly increased which may contribute to elevated blood glucose level and aggravate insulin resistance and eventually metabolic risk in women with GDM [35,43].

Furthermore, highlighting hypercholesterolemia, hypertriglyceridemia, and insulin resistance in the GDM group and a correlation between GLUT-2 and HDL-c expression, like that reported by other authors, showed an increase in total cholesterol, triglycerides and LDL-cholesterol and a decrease in HDL ($p<0.001$) in the GDM group compared to the control group [42]. Parameters proposed by Garduño Alanis in pregnant Mexican women for dyslipidemia (cholesterol >180 mg/dL, triglycerides >170 mg/dL) as gestational age progresses. [30].

Among the strengths of our study was the determination of plasma GLUT-2 on insulin behavior in patients with GDM. Among the limitations was that some of the patients did not comply with the measurement phases due to pregnancy complications associated with the pathology, so they were eliminated from the study analysis complicating the matching of the number of participants. Further research is needed to elucidate the specific involvement of GLUT-2 in the development and progression of GDM which may provide valuable information on its involvement in GDM and possible therapeutic targets.

Conclusions

The present study points to a probable involvement of GLUT2 in the pathogenesis of GDM and insulin resistance, this work contributes to being one of the pioneers in establishing GLUT-2 parameters in human patients with GDM since most of the studies are performed in murine models, as well as to continue exploring the effects on the involvement of glucose metabolism and insulin in the pathogenesis of GDM to open new horizons and provide new evidence on this disease.

Declarations

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Conflict of interest

The authors declare that they have no conflicts of interest.

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Ethical statements

The study was approved by the Research and Research Ethics Committee of the 'HMPMPS' (code: 2022-08-799), and all participants signed the informed consent.

Trial details

This original research article titled "GLUT-2 and Plasma Insulin Concentrations in Women with and without GDM" has the objective of determining GLUT-2 and plasma insulin concentrations in women with and without gestational diabetes, the design is a prospective longitudinal cohort study of Mexicans. The gestational diabetes group had higher GLUT-2 and insulin concentrations, as well as risk factors associated with pregnancy, suggesting that GLUT-2 measurement represents an alternative for better metabolic control and prevention of the development of this pathology.

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