https://doi.org/10.15218/zjms.2024.043

Correlation of blood sugar and lipid profile with first, second and third trimester of pregnancy

Received: 08/11/2022 Accepted: 05/02/2023

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Abstract

Background and objective: Many physiological, hematological, biochemical and metabolic changes can be associated with pregnancy. Changes in carbohydrate and lipid metabolism occur during pregnancy to ensure a continuous supply of nutrients to the growing fetus. The present study was undertaken to evaluate the influence of duration of pregnancy in term of trimester on glucose and lipid metabolism.

Methods: This study was cross – sectional study comprised 62 women. The study population was classified into four groups, non-pregnant (14), first trimester (16), second trimester (16) and third trimester (16). The resulting Sera were analyzed at the same day of sampling for the determination of FBS and serum lipid profile in DNA medical lab.

All data are expressed as mean ± SD. Differences between mean levels of fasting blood sugar and lipid profile of the four groups were evaluated statistically using students' t-test. A value of P <0.05 was considered statistically significant.

Results: Mean of BMI, FBS, TG, TC and LDL in the 3rd trimester pregnant group were significantly higher than non-pregnant group (P = 0.002), (P = 0.025), (P <0.001), (P = 0.021) and (P = 0.034) respectively. Mean of TG in the 3rd trimester group was significantly higher than that in 1st and 2nd trimester groups (P <0.001) respectively.

Conclusion: Lipid profile and blood glucose is variable during each trimester of a normal pregnancy. Total cholesterol, triglycerides and LDL-cholesterol increased in both second and third trimester. The estimation of lipid profile is highly recommended during pregnancy.

Keywords: Maternal lipid profile; Gestational diabetes mellitus.

Introduction

A pregnancy starts with fertilization, when a woman's egg joins with a man's sperm.[1] Many physiological, hematological, biochemical and metabolic changes can be associated with pregnancy, these changes are reversible after delivery if there is no complication.[2]

The hormonal and physiological changes that come with pregnancy are unique, resistance to insulin action on glucose uptake and utilization can be associated with Healthy pregnant women.[3] Insulin resistance is defined as decreasing the ability of target tissues to respond to normal circulating concentrations of insulin.[4]

Sudden and dramatic increases in estrogen and progesterone can be seen during pregnancy. Estrogen levels increase steadily during pregnancy and reach their peak in the third trimester.[5]

Estrogen causes significant alterations in the mother's body metabolism in regards to her salt and water balance, insulin secretion, and ability to break down sugars and carbohydrates.[6]

There are substantial changes in carbohydrate metabolism during pregnancy, such as impaired glucose tolerance (IGT) and gestational diabetes (GDM), is a relatively frequent disease affecting 2–5% of all pregnancies.[7–9]

Changes in carbohydrate and lipid

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https://doi.org/10.15218/zjms.2024.043

metabolism occur during pregnancy to ensure a continuous supply of nutrients to the growing fetus despite intermittent maternal food intake. These metabolic changes are progressive and may be accentuated in women who develop gestational diabetes mellitus (GDM).[10]

During early pregnancy, glucose tolerance is normal or slightly improved and peripheral (muscle) sensitivity to insulin and hepatic basal glucose production is normal.[11] Basal glucose and insulin concentrations do not differ significantly from nongravid values.[12]

Pregnancy produces a 50–60% decrease in insulin sensitivity by late gestation.[13,14] Glucose tolerance during early gestation shows a progressive increase in nutrient-stimulated insulin responses despite an only minor deterioration in glucose tolerance, consistent with progressive insulin resistance in the second and third trimester.[15]

A progressive increase in basal and postprandial insulin concentrations is seen with advancing pregnancy. The first and second phases of insulin release are 3- to 3.5-fold greater in late pregnancy.[16]

By the third trimester, basal and 24-h mean insulin concentrations may double.[17] Skajaa et al state that obese pregnant women develop peripheral and hepatic insulin resistance during the third trimester of pregnancy.[9] Although the precise mechanism is uncertain, alterations in the hormonal milieu during pregnancy are probably responsible for the reduced insulin sensitivity.[18] Increased estrogen, progesterone, and insulin favor lipid deposition and inhibit lipolysis promoting the accumulation of maternal fat stores in early and mid-pregnancy and enhance fat mobilization in late pregnancy.[17]

The higher concentration of estrogen and insulin resistance are thought to be responsible for the hypertriglyceridemia of pregnancy.[19] Gestational diabetes mellitus induces a state of dyslipidemia consistent with insulin resistance. higher serum triacylglycerol concentrations can be seen in women with GDM than do normal pregnant women.[20]

As glucose and lipid metabolism altered during pregnancy.[21] So, the present study was undertaken to evaluate the influence of duration of pregnancy in term of trimester on glucose and lipid metabolism.

Methods

Study design:

This study was cross – sectional study comprised (62) women, 14 non-pregnant women taken as a control group, 16 pregnant women in first trimester, 16 pregnant women in second trimester and 16 pregnant women in third trimester consulted to Awatydwarosh medical Center between July to August 2022 and subjected for a questionnaire.

Exclusion criteria:

Pregnant women with preeclampsia, a history of dyslipidemia or type II diabetes, smokers, hypertensive women, history of abortion and protein urea were excluded.

Ethical consideration:

The study approved by Ethic Committee of College of Pharmacy/ Hawler Medical University and the consent was given from study participants in verbal form (HMU-EC-Ph 02062022-652).

Data collection:

Blood samples were collected from each woman after fasting for 12 hours. 5ml of blood were obtained from each woman by vein puncture. The blood samples were allowed to clot at room temperature, and then centrifuged at 3000 rpm for 10 minutes. The resulting Sera were analyzed at the same day of sampling for the determination of FBS and serum lipid profile in DNA medical lab.

A person's height and weight are used to calculate their Body Mass Index. BMI is calculated by taking weight in kilograms divided by height in meters squared (kg/ m2). A BMI of 25.0 or more is overweight, while the healthy range is 18.5 to 24.9. BMI applies to most adults 18-65 years.[22]

Statistical analysis:

Statistical analyses were done using

https://doi.org/10.15218/zjms.2024.043

the Statistical Package for Social Sciences (SPSS version 23) IBM-computer software in association with Microsoft Excel. All data are expressed as mean ± SD. Differences between mean levels of fasting blood sugar and lipid profile of the four groups were evaluated statistically using ANOVA test. A *P*-value of <0.05 was considered statistically significant.

Results

As shown in Table 1, a statistically significant differences in mean age (*P* = 0.005) between the non-pregnant group and the entire pregnancy group. Mean of age and BMI in the 3rd trimester pregnant group were significantly higher than non-pregnant group (*P* = 0.005) and (*P* = 0.002) respectively.

Table 2 shows a clearly demonstrated statistically significant differences in mean TG (*P* <0.001) between the non-pregnant group and the entire pregnancy group. Mean of FBS, TG, TC and LDL in the 3rd trimester pregnant group were significantly higher than non-pregnant group (*P* = 0.025), (*P* <0.001), (*P* = 0.021) and (*P* = 0.034) respectively. Mean of TG in the 3rd trimester group was significantly higher than that in 1st trimester and 2nd trimester groups (*P* <0.001) respectively. Even though the mean FBS of the 3rd trimester group (141 ± 53.33)were higher than that of the 1st trimester (133.8 ± 22.5) and 2nd trimester group (125.3 ± 16.4), these differences were not significant (*P* >0.05).

Table 1 Difference in Mean ± SD of age and BMI by study Groups

Study groups and variables	NO.	Mean ± SE	*P* (ANOVA)	LSD (Groups)	*P* (LSD)
Age (years)					
A) Non pregnant	14	(33.4 ± 7.9)	0.005	A X B	0.29
B) 1st trimester	16	(28.8 ± 6.1)		A X C	0.008
C) 2nd trimester	16	(27.8 ± 3.2)		A X D	0.005
D) 3rd trimester	16	(26.0 ± 4.0)		B X C	0.613
				B X D	0.148
				C X D	0.344
BMI					
A) Non pregnant	14	(33.4 ± 5.6)	0.430	A X B	0.094
B) 1st trimester	16	(36.2 ± 4.5)		A X C	0.908
C) 2nd trimester	16	(33.2 ± 4.8)		A X D	0.002
D) 3rd trimester	16	(37.0 ± 2.8)		B X C	0.064
				B X D	0.613
				C X D	0.02

https://doi.org/10.15218/zjms.2024.043

Table 2 Difference in Mean ± SD of FBS and lipid profile by study Groups

Study groups and variables	NO.	Mean ± SE	*P* (ANOVA)	LSD (Groups)	*P* (LSD)
FBS					
A) Non pregnant	14	(122.6 ± 13.9)	0.342	A X B	0.333
B) 1st trimester	16	(133.8± 22.5)		A X C	0.819
C) 2nd trimester	16	(125.3 ± 16.4)		A X D	0.025
D) 3rd trimester	16	(141 ± 53.33)		B X C	0.442
				B X D	0.501
				C X D	0.152
TG					
A) Non pregnant	14	(186.0 ± 18.3)	< 0.001	A X B	0.958
B) 1st trimester	16	(178± 18.9)		A X C	0.870
C) 2nd trimester	16	(188.9 ± 46.4)		A X D	< 0.001
D) 3rd trimester	16	(251.3 ± 77.3)		B X C	0.909
				B X D	< 0.001
				C X D	< 0.001
HDL					
A) Non pregnant	14	(46.6 ± 15.1)	0.909	A X B	0.971
B) 1st trimester	16	(46.8± 20.0)		A X C	0.584
C) 2nd trimester	16	(45.1 ± 19.1)		A X D	0.765
D) 3rd trimester	16	(44.0 ± 14.4)		B X C	0.545
				B X D	0.634
				C X D	0.888
TC					
A) Non pregnant	14	(184.3 ± 24.7)	0.343	A X B	0.289
B) 1st trimester	16	(186.1± 38.4)		A X C	0.502
C) 2nd trimester	16	(175.6 ± 16.7)		A X D	0.707
D) 3rd trimester	16	(189.2 ± 50.3)		B X C	0.021
				B X D	0.477
				C X D	0.280
LDL					
A) Non pregnant	14	(94.97 ± 48.1)	0.577	A X B	0.410
B) 1st trimester	16	(95.9± 56.0)		A X C	0.721
C) 2nd trimester	16	(94.7 ± 36.0)		A X D	0.034
D) 3rd trimester	16	(100.5 ± 30.0)		B X C	0.224
				B X D	0.230
				C X D	0.987

https://doi.org/10.15218/zjms.2024.043

Discussion

In pregnancy profound anatomic and physiologic occur in almost every organ system.[23] These changes start occurring just after conception have taken place and keeps on evolving throughout the pregnancy including the delivery period.[24] These changes occur in order to facilitate the needs of mother and fetus.[25]

Maternal physiology is highly influenced by the placental hormones especially in last trimester of the pregnancy. The variation in hormonal levels generally affects the glucose and lipid metabolism and such variations take place in order to make sure that the fetus receives an ample supply of nutrients for its development.[26]

Throughout gestation, blood levels of total cholesterol, triglycerides, and glucose significantly elevate.[27] These results are consistent with our findings. There was a significant difference in total cholesterol, TG and FBS between non pregnant and pregnant women in 3rd trimester.

The large rise in triglycerides is due to two factors, increased hepatic lipase activity, leading to enhanced hepatic triglyceride synthesis and reduced lipoprotein lipase activity, resulting in decreased catabolism of adipose tissue.[28] These changes in lipid metabolism help the mother and fetus to adapt. High triglyceride concentrations are thereafter used for maternal metabolic needs while sparing glucose for the fetus.[29]

HDL-Cholesterol levels in third trimester pregnant women were lower than non-pregnant women but the results were not significant. Our results differ than the results of Abdelhadi AT, Jamil et al., on women at different stages of pregnancy that found a significant decrease in high density lipoprotein cholesterol with increased low-density lipoprotein concentrations.[30] This difference may be due to the difference in ethnicity and low number of participants in our study.

Wang J et al. state that all the lipid parameters were significantly modified particularly in second and third trimester when compared to non-pregnant females as well as when compared to the values in first trimester.[31] Our findings support these conclusions.

Furthermore, in another study done by F. Okojie, Blessing et al. on 120 women., found that, the concentrations of total cholesterol, high density lipoprotein, triglycerides and FBG during the first, second and third trimesters were significantly high (P <0.05) as compared to that of the control subjects. However, the change in low density lipoprotein was not significantly high (P >0.05) during the first trimester but was significantly high (P <0.05) during the second and third trimester as compared to that of the control.[32] Our study's findings strongly support these conclusions. Our results also agreed by Zhu Y et al that stated that there is a significant difference between the means of TG of control (non-pregnant) group and (2nd, 3rd trimester) pregnant group (P <0.05).[15]

In short, lipid profile is variable during each trimester of a normal pregnancy.[28] There was a difference between lipid profile for the 1st, 2nd and 3rd trimester pregnant group, but it was not significant, this may be due to the small number of women that participate in this study as mentioned before.

Our investigation revealed a significant difference between FBS of the third trimester pregnant and non-pregnant control group, these outcomes are supported by the findings of Assel et al. They discovered that, in comparison to women who are not pregnant, their glucose production increases significantly during the duration of pregnancy.[33]

Conclusion

Pregnancy associated with an “atherogenic” lipid profile which could act as a potential risk factor for insulin resistance which is the main reason of gestational diabetes.

Total cholesterol and triglycerides significantly increased in both second and third trimester. The increase is more significant in third trimester, when

compared to second. HDL-Cholesterol is decreased in third trimester when compared to second trimester.

The estimation of lipid profile is highly recommended during pregnancy so as to institute prompt management strategies to prevent deleterious effect of hyperlipidemia associated with pregnancy.

Funding

Not applicable.

Competing interests

The author declares that he has no competing interests.

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