



## Correlation of Serum Adiponectin Levels with Gestational Diabetes Mellitus Indices

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### KEYWORDS:

Adiponectin, Dyslipidaemia, and Gestational Diabetes Mellitus

### ABSTRACT:

**Introduction:** Maternal and fetal complications may be avoided by early detection of GDM using biomarker measurement during the pregnancy. Adipose tissue secretes adiponectin, which lowers blood glucose levels and modifies insulin sensitivity. The aim of this research to assess the relationship between the adiponectin and development of GDM.

**Objective:** The aim of this research to assess the relationship between the adiponectin and development of GDM.

**Methods:** This cross-sectional study included 150 pregnant women age between 19-35 years, out of which 50 gestational diabetes mellitus weeks between 25-29, 50 gestational diabetes mellitus weeks between 25-29 and 50 were normal pregnant women considered as controls. Biochemical and adiponectin levels was measured.

**Results:** The mean adiponectin level was drastically decreased in both gestational diabetes mellitus cases than control groups, respectively, ( $P = 0.001^{**}$ ). These levels were significant negative correlation with blood sugars, glycated haemoglobin and dyslipidaemia ( $P=0.00^{**}$ ).

**Conclusion:** This study indicates insufficient levels of serum adiponectin in gestational diabetes mellitus when compared with age and body mass index matched normal pregnant women.

### 1. Introduction

Gestational diabetes mellitus (GDM), a common pregnancy complication, is diabetes mellitus that initially develops during pregnancy. Depending on the population and diagnostic standards applied, the prevalence of GDM may be as high as 18%. Maternal insulin sensitivity significantly decreases during the pregnancy (1-3). However, GDM may occur as a result of decreased  $\beta$  cell reserve or maladaptation to increased insulin demands. Pregnancy-related aberrant metabolic conditions may have a negative impact on the development of the fetus (4-5). Furthermore, epidemiological studies have shown that GDM is a strong predictor of a woman's propensity

to develop type 2 diabetes later in life. Furthermore, GDM is a major predictor of cardiovascular illness in later life. Women with a prior history of GDM had a 70% higher risk than those without (6-8).

Adipocytokines such as adiponectin and others are produced by adipose tissue, a very active endocrine organ. A physiologically active polypeptide hormone is called adiponectin. The primary regulator of its release from adipocytes is insulin (9-10). By attaching to its receptor and activating AMP activated protein kinase (AMPK) and peroxisome proliferator activated receptor a (PPAR-a) in the liver and skeletal muscle, it promotes the absorption of glucose and fatty acid oxidation (11).



By blocking gluconeogenesis, it lowers glucose levels. By stimulating the beta oxidation of fatty acids in skeletal muscle, it decreases the need of insulin. The Body Mass Index (BMI), intraabdominal fat, and instances of insulin resistance are all negatively linked with plasma adiponectin (12-13).

Adiponectin levels in pregnant GDM subjects have been found to be low. According to certain research, women with low adiponectin levels are five to six times more likely to develop GDM than those with normal or high levels (14-15). The placenta does not generate or express adiponectin. Maternal adipose tissue can be evaluated as an independent biomarker because it is the primary source of maternal plasma adiponectin (16-17). Serum adiponectin levels can be used as predictors of GDM in early pregnancy with later therapies to prevent problems if any correlation between these levels and the subsequent development of GDM can be assessed. This study sought to determine whether early pregnancy serum adiponectin levels were associated with the development of GDM.

## 2. Objectives

The aim of this research to assess the relationship between the adiponectin and development of GDM.

## 3. Methods

The present cross-sectional study was conducted in the Department of Obstetrics and Gynaecology, Akash Institute of Medical Sciences and Research Centre, Bangalore, India after obtaining the ethical clearance from the institutional ethical committee between March 2023 to January 2025. A total 150 pregnant women age between 19-35 years were included, out of which 50 gestational diabetes mellitus weeks between 25-29, 50 gestational diabetes mellitus weeks between 25-29 and 50 were normal pregnant women considered as controls.

### Criteria of the study

The pregnant women visiting to Obstetrics and Gynaecology outpatient department and diagnosed with gestational diabetes according to National Diabetes Data Group (NDDG) criteria were included. Women with chronic hypertension, renal disease, hypothyroidism, collagen vascular disease, diabetes mellitus and multiple pregnancy, other complications (PIH, anemia, preeclampsia) not willing for study, on steroids,

metformin, any drug that interferes with vitamin D metabolism like anti-epileptics and nifedipine were excluded from the study. Blood sample was collected at study place and sent to the laboratory for routine biochemical parameters measured and serum adiponectin determined by ELISA method.

## Statistical analysis

Continuous variables were expressed as mean  $\pm$  SD and Graphically the data was presented by box and scatter plots. Analysis of variance was employed for comparing the variables. Pearson's correlation used to correlate between the study variables. A p value of less than 0.05 was considered statistically significant.

## 4. Results

### Table 1: baseline measurements between controls and both groups of GDM

The age, weeks of gestation and BMI significant high in both the groups of GDM cases when compared to controls ( $P=0.001^{**}$ ). Along with that gestational diabetic women shown significantly high levels of FBS, PPBS, HbA1c, total cholesterol, triglycerides, VLDL, and LDL than controls ( $P=0.001^{**}$ ). Furthermore, HDL was very low in both groups of GDM cases than controls ( $P=0.001^{**}$ ). Additionally, serum adiponectin levels were significant and drastically decreased in both groups of GDM cases when compared to controls ( $P=0.001^{**}$ ) (Table 1).

### Table 2: Serum adiponectin and other study variables correlation

The serum adiponectin levels were significant and negatively correlated with weeks of gestation, BMI, blood sugars, glycated haemoglobin, total cholesterol, triacylglycerides, VLDL and LDL ( $P=0.001^{**}$ ) and positively correlated with HDL levels ( $P=0.001^{**}$ ) (Table 2).

### Figure 1: Graphical representation of FBS, HbA1c and adiponectin levels between study subjects

Figure 1 demonstrates the box plots for FBS, HbA1c and serum adiponectin levels. There were significant and drastically elevated levels of FBS, and HbA1c levels in both groups of GDM cases when compared to controls. Additionally, serum adiponectin levels drastically



reduced in both groups of GDM cases when compared to controls ( $P=0.001^{**}$ ).

## 5. Discussion

The hallmark of gestational diabetes mellitus (GDM) in pregnancy is reduced glucose tolerance, which is initially identified by low serum adiponectin levels during screening. Pregnancy causes increased IR, which cannot be compensated for by the  $\beta$ -cell reserve.  $\beta$ -cell mass changes in response to increased functional demands and physiological demands (18-20). Neogenesis, hyperplasia, and hypertrophy are possible methods for achieving these  $\beta$ -cell mass alterations. Placental lactogen, prolactin, and growth hormone all have comparable effects on  $\beta$ -cells and are all enhanced during pregnancy, which stimulates  $\beta$ -cell proliferation (21-23). Lowering serum adiponectin levels have a significant impact on placental nutrient transport and fetal development during pregnancy because they prevent insulin-stimulated amino acid transfer across the placenta (24).

In this study, we show that beta-cell activity in the middle of pregnancy is independently correlated with low serum adiponectin levels. Compared to normoglycemic controls, the mean adiponectin level was considerably lower in GDM cases.

Additionally, recent studies also found women with gestational diabetes mellitus had lower adiponectin concentrations than women with a normal pregnancy (25-27). Similarly, recent researchers observed the serum adiponectin levels and BMI have been found to be negatively correlated in both GDM and normoglycemic pregnant women (28-29). Based on study findings, the serum adiponectin, it emerges as an important factor potentially linking IR and beta cell dysfunction in the pathogenesis of gestational diabetes mellitus.

## 7. Conclusion

In comparison to age- and BMI-matched normoglycemic pregnant women, our study showed that women with GDM had lower serum adiponectin levels. According to the research, reduced adiponectin levels in GDM may be an early occurrence in the disease's natural history and could be a crucial preliminary assessment in GDM prenatal screening.

## References

1. Guariguata L, Linnenkamp U, Beagley J, Whiting DR, Cho NH. Global estimates of the prevalence of hyperglycaemia in pregnancy. *Diabetes Res Clin Pract* 2014;103:176-85.
2. Metzger BE, Coustan DR. Summary and recommendations of the Fourth International Workshop-Conference on Gestational Diabetes Mellitus. The Organizing Committee. *Diabetes Care* 1998;21 Suppl 2:B161-7.
3. Seshiah V, Balaji V, Balaji MS, Sanjeevi CB, Green A. Gestational diabetes mellitus in India. *J Assoc Physicians India* 2004;52:707-11.
4. Swami SR, Mehetre R, Shivane V, Bandgar TR, Menon PS, Shah NS. Prevalence of carbohydrate intolerance of varying degrees in pregnant females in Western India (Maharashtra) – A hospital-based study. *J Indian Med Assoc* 2008;106:712-4, 735. 5.
5. Catalano PM. Obesity, insulin resistance, and pregnancy outcome. *Reproduction* 2010;140:365-71.
6. Robinson DP, Klein SL. Pregnancy and pregnancy-associated hormones alter immune responses and disease pathogenesis. *Horm Behav* 2012;62:263-71.
7. Al-Noaemi MC, Shalayel MH. Pathophysiology of gestational diabetes mellitus: The past, the present and the future. *Gestational Diabetes* 2011;6:91-114.
8. Cortelazzi D, Corbetta S, Ronzoni S, Pelle F, Marconi A, Cozzi V, et al. Maternal and foetal resistin and adiponectin concentrations in normal and complicated pregnancies. *Clin Endocrinol (Oxf)* 2007;66:447-53.
9. American Diabetes Association. Standards of medical care in diabetes-2015 abridged for primary care providers. *Clin Diabetes* 2015;33:97-111.
10. Retnakaran R, Hanley AJ, Raif N, Connelly PW, Sermer M, Zinman B. Reduced adiponectin concentration in women with gestational diabetes: A potential factor in progression to type 2 diabetes. *Diabetes Care* 2004;27:799-800.



11. Al-Badri MR, Zantout MS, Azar ST. The role of adipokines in gestational diabetes mellitus. *Ther Adv Endocrinol Metab* 2015;6:103-8.
12. Doruk M, Ugur M, Oruç AS, Demirel N, Yildiz Y. Serum adiponectin in gestational diabetes and its relation to pregnancy outcome. *J Obstet Gynaecol* 2014;34:471-5.
13. Saini V, Kataria M, Yadav A, Jain A. Role of leptin and adiponectin in gestational diabetes mellitus: A study in a North Indian tertiary care hospital. *Internet J Med Update EJ* 2015;10:11-4.
14. Nimptsch, K., Konigorski, S. & Pischon, T. Diagnosis of obesity and use of obesity biomarkers in science and clinical medicine. *Metabolism* 2018;92:61–70.
15. Nielsen, M. B., Çolak, Y., Benn, M. & Nordestgaard, B. G. Low Plasma adiponectin in risk of type 2 diabetes: Observational analysis and one- and two-sample mendelian randomization analyses in 756,219 individuals. *Diabetes* 2021;70:2694–2705.
16. Fuglsang, J., Skjaerbaek, C., Frystyk, J., Flyvbjerg, A. & Ovesen, P. A longitudinal study of serum adiponectin during normal pregnancy. *BJOG* 2006;113:110–113.
17. Andersson-Hall, U. et al. Longitudinal changes in adipokines and free leptin index during and after pregnancy in women with obesity. *Int. J. Obes. (Lond.)* 2020; 44:675–683.
18. Lindberger, E. et al. Early mid-pregnancy blood-based proteins as possible biomarkers of increased infant birth size in sex-stratified analyses. *Reprod. Sci* 2023; 30:1165–1175.
19. Wang, J. et al. Relationship of adiponectin and resistin levels in umbilical serum, maternal serum and placenta with neonatal birth weight. *Aust. N. Z. J. Obstet. Gynaecol.* 2010;50:432–438.
20. Nanda, S., Akolekar, R., Sarquis, R., Mosconi, A. P. & Nicolaides, K. H. Maternal serum adiponectin at 11 to 13 weeks of gestation in the prediction of macrosomia. *Prenat. Diagn.* 2011; 31: 479–483.
21. Lekva, T. et al. Large reduction in adiponectin during pregnancy is associated with large-for-gestational-age newborns. *J. Clin. Endocrinol. Metab* 2017;102:2552–2559.
22. Noguez, P. et al. Maternal obesity influences expression and DNA methylation of the adiponectin and leptin systems in human third-trimester placenta. *Clin. Epigenet.* 2019: 11- 20.
23. Duval, F. et al. Adiponectin inhibits nutrient transporters and promotes apoptosis in human villous cytotrophoblasts: Involvement in the control of fetal growth. *Biol. Reprod* 2016;94:111.
24. Rosario, F. J. et al. Chronic maternal infusion of full-length adiponectin in pregnant mice down-regulates placental amino acid transporter activity and expression and decreases fetal growth. *J. Physiol* 2012; 590:1495–1509.
25. Aye, I. L., Rosario, F. J., Powell, T. L. & Jansson, T. Adiponectin supplementation in pregnant mice prevents the adverse effects of maternal obesity on placental function and fetal growth. *Proc. Natl. Acad. Sci.* 2015: 112:12858–12863.
26. Jansson, N. et al. Maternal hormones linking maternal body mass index and dietary intake to birth weight. *Am. J. Clin. Nutr.* 2008;87:1743–1749.
27. Haghiac, M. et al. Patterns of adiponectin expression in term pregnancy: Impact of obesity. *J. Clin. Endocrinol. Metab* 2014; 99:3427– 3434.
28. Xu J, Zhao YH, Chen YP, Yuan XL, Wang J, Zhu H, Lu CM. (2014) ‘Maternal circulating concentrations of tumor necrosis factor-alpha, leptin, and adiponectin in gestational diabetes mellitus: a systematic review and meta-analysis’, *Scientific World Journal*, 2014; 926-932.
29. Madhu SV, Bhardwaj S, Jhamb R, Srivastava H, Sharma S, Raizada N. ‘Prediction of Gestational Diabetes from First Trimester Serum Adiponectin Levels in Indian Women’, *Indian journal of endocrinology and metabolism*, 2019; 23(5):536–539.



**Table 1: baseline measurements between controls and both groups of GDM**

Parameters	Controls		GDM 1		GDM 2		P-value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD			
Age (Years)	28.0 ± 1.2	24.8 ± 2.0	27.1 ± 2.0	0.001*			
Weeks of Gestation	23.9 ± 1.5	26.8 ± 1.4	33.0 ± 1.6	0.001*			
BMI (kg/m <sup>2</sup> )	23.0 ± 1.5	22.7 ± 1.8	33.2 ± 3.1	0.001*			
FBS (mg/dL)	84.8 ± 8.2	136.1 ± 1.4	154.4 ± 1.7	0.001*			
PPBS (mg/dL)	278.7 ± 4.4	276.5 ± 4.1	283.0 ± 5.7	0.001*			
HbA1C (%)	5.08 ± 0.06	6.69 ± 0.05	8.02 ± 0.04	0.001*			
Total Cholesterol	162.35 ± 1.0	173.17 ± 1.6	273.85 ± 1.5	0.001*			

Triacylglycerides (mg/dL)	115.92 ± 1.7	138.08 ± 1.7	195.35 ± 2.3	0.001*
HDL (mg/dL)	47.75 ± 0.5	36.82 ± 0.4	30.79 ± 0.5	0.001*
VLDL (mg/dL)	24.18 ± 0.1	27.13 ± 0.1	41.82 ± 0.1	0.001*
LDL (mg/dL)	87.49 ± 0.2	109.47 ± 0.9	205.72 ± 0.6	0.001*
Serum Adiponectin (mg/L)	47.17 ± 0.1	28.68 ± 0.6	14.72 ± 0.2	0.001*



**Table 2: Serum adiponectin and other study variables correlation**

Serum Adiponectin (mg/L)		
Parameters	r- Value	P-Value
Weeks of Gestation	-0.857	0.001**
BMI (kg/m <sup>2</sup> )	-0.722	0.001**
FBS (mg/dL)	-0.886	0.001**
PPBS (mg/dL)	-0.042	0.001**
HbA1C (%)	-0.835	0.001**
Total Cholesterol (mg/dL)	-0.813	0.001**
Triacylglycerides (mg/dL)	-0.801	0.001**
HDL (mg/dL)	0.832	0.001**
VLDL (mg/dL)	-0.787	0.001**
LDL (mg/dL)	-0.861	0.001**

**Figure 1: Graphical representation of FBS, HbA1c and adiponectin levels between study subjects**

