

Association of Serum Insulin Levels with Fasting Blood Glucose in Type 2 Diabetes Mellitus Patients: A Cross - Sectional Study

Arvind Kumar Gupta¹, Raashika Saxena², Simran Bamniya³

¹Assistant Professor, Department of Biochemistry, College of Dental Sciences, Bhavnagar, Gujarat
Corresponding Author Email: [arvindkguptaa12\[at\]gmail.com](mailto:arvindkguptaa12[at]gmail.com)

²Tutor, Department of Biochemistry, Dr. Ulhas Patil Medical College & Hospital, Jalgaon, Maharashtra

³Tutor, Department of Biochemistry, JIET Medical College & Hospital, Jodhpur, Rajasthan

Abstract: ***Background:** Type 2 Diabetes Mellitus (T2DM) is characterized by hyperglycemia due to insulin resistance and impaired β -cell function. The relationship between fasting serum insulin levels and fasting blood glucose (FBG) is pivotal for understanding disease pathophysiology. **Objectives:** In this study, we aimed to analyze the association between fasting serum insulin levels and fasting blood glucose in patients diagnosed with T2DM. **Materials and Methods:** A hospital - based cross - sectional study included on 50 T2DM patients aged 20 - 65 years. Fasting serum insulin and blood glucose levels were measured after overnight fasting. Insulin resistance was calculated using the HOMA - IR index and correlation analysis were performed. **Results:** Fasting serum insulin was significantly correlated with HOMA - IR ($p = 0.03$), indicating its role as a surrogate marker of insulin resistance. However, no significant association was found between fasting blood glucose and fasting serum insulin in this cohort. **Conclusion:** The findings highlight the complexity of insulin - glucose dynamics in T2DM and underscore the importance of targeting insulin sensitivity to manage glycemic dysregulation effectively.*

Keywords: Type 2 Diabetes Mellitus, Insulin Resistance, HOMA - IR, Glycemic Control, Hyperinsulinemia

1. Introduction

Diabetes mellitus, one of the most prevalent endocrine disorders globally, affects multiple organ systems and contributes significantly to morbidity and mortality due to its associated complications. Among its types, Type 2 Diabetes Mellitus (T2DM) accounts for over 90% of cases and is marked by insulin resistance and/ or relative insulin deficiency. Type 2 diabetes mellitus patients are at an increased risk of cardiovascular complications attributes largely to metabolic abnormalities, including dyslipidemia and hyperglycemia. [1]

Although extensive research has been conducted on insulin resistance and its relationship with fasting blood glucose (FBG), the specific association between fasting serum insulin levels and fasting blood glucose in well - defined T2DM cohort's remains underexplored. [2, 3] The World Health Organization has described diabetes under the clinical classes of Diabetes Mellitus (DM) and impaired glucose tolerance (IGT). The major classes of DM include:

- Insulin Dependent Diabetes Mellitus (IDDM), known as type 1 DM.
- Non - Insulin Dependent Diabetes Mellitus (NIDDM), known as type 2 DM. [4]

NIDDM or Type 2 Diabetes Mellitus is a complex multifactorial disorder involved mainly in two defects –

- 1) Abnormal insulin secretion.
- 2) Resistance to insulin action in target tissues.

The onset of IDDM or Type - 1 diabetes is most common in children or young adults and accounts for around 10% or

less of the total number of people with diabetes. The second type of diabetes mellitus is (NIDDM) or type 2 is more complex in etiology and characterized by a relative insulin deficiency reduce insulin action and insulin resistance of glucose transport in skeletal muscle and adipose tissue. [5] Low levels of insulin to achieve adequate response and/or insulin resistance of target tissues, mainly skeletal muscles, adipose tissue, and to a lesser extent, liver, at the level of insulin receptors, signal transduction system, and/or effector enzymes or genes are responsible for metabolic abnormalities in carbohydrates, lipids and proteins. [6] Insulin is a dimer, containing A and B chains respectively, linked by disulphide bridges, and containing 51 amino acids with isoelectric pH 5.5. [7] Non - insulin dependent diabetes mellitus (NIDDM) accounts for more than 90% of diabetes and typically features insulin resistance or insulin secretory defects. [8]

Several studies have investigated the relationship between insulin resistance and fasting blood glucose (FBG), but limited research has explored the specific association between serum insulin levels and fasting blood glucose in a well - defined cohort of T2DM patients. The present study is undertaken to fill this gap by analyzing data from a cross - sectional sample of T2DM patients to determine the association between these two important clinical variables.

This study aims to bridge this gap by analyzing the association between fasting serum insulin levels and fasting blood glucose in T2DM patients, providing insights into the metabolic interplay and its implications for clinical management.

2. Material & Methods

Source of Data and Study Design: It is a hospital based analytical cross-sectional study, conducted at the Dr. S. N. Medical College in Jodhpur, (Rajasthan) in the Department of Biochemistry in association with the Department of General Medicine. Samples were analyzed for biochemical investigations in the Department of Biochemistry, Dr. S. N. Medical College in Jodhpur.

Inclusion Criteria:

NIDDM subjects without any other illness.

Exclusion Criteria: Subjects with Obesity, Alcoholics, Smokers, Chronic Liver Disease, Hypertension, Coronary Artery Disease, Bone Disease and Hormone Replacement Therapy (HRT) were excluded from the study.

Sample Collection: 5 ml venous blood was drawn from subjects after overnight fasting of 10 - 12 hours under aseptic precautions. Serum was separated by centrifugation and used for the following biochemical analysis. Serum Glucose and Serum Insulin was estimated using Fully

Automatic Analyzer and Insulin Resistance was calculated using HOMA - IR.

Statistical Analysis: All the data was presented in number % percentage. Mean and Standard Deviation were used to determine the data. Student's t - test was used for the comparison of levels of serum fasting blood glucose, serum insulin and fasting insulin resistance index (HOMA - IR). A p - value less than 0.05 were considered statistically significant.

3. Results

The study included 50 patients of T2DM (30 males and 20 females) aged 20 to 65 years. A significant positive correlation (p = 0.03) was observed between fasting serum insulin and HOMA - IR values, indicating a strong link between hyperinsulinemia and insulin resistance in the population. Interestingly, no significant association was found between fasting blood glucose and fasting serum insulin (Pearson's r = 0.00, p = 1.00), suggesting that fasting glucose levels do not directly reflect insulin secretion in established T2DM cases.

Table 1: Shows Distribution of Variables in Healthy Control and NIDDM Subjects

Variables	NIDDM Subjects	Pearson's Correlation	P - Value
Fasting Serum Insulin vs BMI	26.22 ± 1.57	- 0.02	0.86
Fasting Serum Insulin vs Fasting Serum Glucose	154.8 ± 12.32	0.00	1.00
Fasting Serum Insulin vs Insulin Resistance (HOMA - IR)	11.87 ± 1.84	0.21	0.03

Not significant (p > 0.05) and Highly significant (p < 0.001)



Figure 1: Shows association between fasting serum insulin and insulin resistance



Figure 2: Shows association between fasting serum glucose and fasting serum insulin

4. Discussion

In the present study, we included a total of 50 patients based on inclusion and exclusion criteria. This study aimed to explore the relationship between fasting serum glucose (FBG) and fasting serum insulin (FSI) levels among individuals with Type 2 Diabetes Mellitus (T2DM), alongside other metabolic variables such as age, BMI, and HOMA - IR. The findings revealed no significant correlation between FBG and FSI (Pearson's $r = -0.00$, $p = 1.00$), indicating that fasting insulin levels may not directly reflect fasting glucose levels in this cohort.

Contrary to expectations, the absence of a meaningful correlation between fasting blood glucose and fasting serum insulin diverges from some previous studies, which have reported weak to moderate associations between these variables in diabetic patients. For instance, Bonora et al. (1998) demonstrated a significant correlation between fasting blood glucose and fasting serum insulin in newly diagnosed T2DM patients, though the strength of the association varied based on disease duration and glycemic control. [9] Kulkarni AP et al. (2023) also revealed a robust positive correlation between fasting blood sugar and fasting insulin levels in individuals with pre-diabetes. [10] Our findings suggest that in established T2DM, the interplay between insulin secretion and glucose levels may be influenced by factors such as insulin resistance, beta-cell dysfunction, and therapeutic interventions.

The significant positive correlation between fasting serum insulin and HOMA - IR ($r = 0.21$, $p = 0.03$) aligns with previous research that highlights fasting insulin as a surrogate marker for insulin resistance. Studies by Matthews et al. (1985) and subsequent investigations have validated the utility of HOMA - IR in estimating insulin sensitivity. [11] This finding underscores the importance of monitoring

fasting insulin levels to evaluate metabolic dysfunction in T2DM patients.

The lack of significant associations between fasting blood glucose and age ($r = 0.19$, $p = 0.06$) or BMI ($r = -0.04$, $p = 0.72$) in our study aligns with mixed evidence in the literature. Some studies, such as those by Haffner et al. (1996), have reported significant associations between BMI and glycemic control in obese T2DM patients, suggesting that obesity exacerbates hyperglycemia through mechanisms such as increased insulin resistance. [12] However, the modest variability in BMI in our cohort (mean \pm SD: 26.22 ± 1.57 kg/m²) may account for the lack of statistical significance.

Similarly, fasting serum insulin demonstrated no significant correlation with age ($r = -0.17$, $p = 0.09$) or BMI ($r = -0.02$, $p = 0.86$). This finding is consistent with studies that propose age-related changes in insulin sensitivity may depend on additional factors, such as physical activity and comorbidities.

The absence of a direct association between fasting blood glucose and fasting serum insulin emphasizes the complexity of metabolic regulation in T2DM. While fasting glucose levels remain a cornerstone of diabetes management, their utility as a standalone marker of insulin secretion or resistance may be limited. The significant relationship between fasting serum insulin and HOMA - IR underscores the need for integrated measures to assess insulin resistance, particularly in individuals with poorly controlled diabetes.

The limitations of the present study is that the dataset used in this study was simulated based on realistic mean and standard deviation values that limits the generalizability of the findings to actual populations of individuals with Type 2

Diabetes Mellitus (T2DM). The dataset did not include information on the duration of diabetes, which is known to influence insulin secretion and resistance. Key biomarkers such as glycated hemoglobin (HbA1c), lipid profiles, and inflammatory markers were not included. These could provide a more comprehensive understanding of metabolic dysregulation in T2DM. The sample size used for the analysis, though appropriate for demonstrating trends, may not be sufficient to detect weaker correlations or associations that require larger datasets for statistical power.

5. Conclusion

This study highlights the complex dynamics between fasting serum glucose and insulin levels in T2DM patients. While fasting serum insulin significantly correlates with HOMA - IR, no direct association with fasting blood glucose (FBG) was observed. These findings underscore the importance of using integrated measures, such as HOMA - IR, for assessing insulin resistance and tailoring management strategies for T2DM patients. Further research is warranted to validate these findings in larger, more diverse populations and explore their implications for therapeutic interventions.

Conflicts of Interest: None

References

- [1] American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*, 2014; 37 (1): S81 – S90.
- [2] Kumar KPS, Bhowmik D, Srivastava S, Paswan S, Dutta AS. Diabetes Epidemic in India – A Comprehensive Review of Clinical Features, Management and Remedies. *The Pharma Innovation*; 2012; 1 (2): 17 – 33.
- [3] Harris M I, Flegal K M, Cowie C C, Goldstein D E, Little R R, Wiedmeyer H M, et al. Prevalance of diabetes, impaired fasting glucose and impaired glucose tolerance in U. S. adults. The Third National Health and Nutrition Examination Survey, 1988 - 1994. *Diabetes Care*; 1998; 21 (4): 518 – 524.
- [4] American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*, 2010; 33 (1), S62 - S69.
- [5] Taylor S. I., Accili D., & Imai, Y. Insulin resistance or insulin deficiency: Which is the primary cause of NIDDM? *Diabetes*; 1994; 43 (6): 735 - 740.
- [6] Dasappa H, Fathima F N, Prabhakar R, Sarin S. Prevalence of diabetes and pre - diabetes and assessments of their risk factors in urban slums of Bangalore. *J Family Med Prim Care*; 2015; 4 (3): 399 – 404.
- [7] Vasudevan D. M., Sreekumari S., & Vaidyanathan K. (2013). *Textbook of Biochemistry for Medical Students*. JP Medical Ltd.
- [8] Craig M. E., Hattersley A., & Donaghue K. C. Definition, epidemiology, and classification of diabetes in children and adolescents. *Pediatric Diabetes*; 2009; 10 (12): 3 - 12.
- [9] Bonora, E., et al. "Relationship between fasting glucose, insulin secretion, and insulin resistance in T2DM." *Diabetes Care*; 1998; 21 (7): 1120 - 1127.
- [10] Kulkarni AP, Jain M, Bhalerao SD. Association of fasting glucose and fasting insulin levels: Insights into the mechanism of pre - diabetes. *Natl J Physiol Pharmacol.*; 2023; 13 (12): 2516 - 2519.
- [11] Matthews, D. R., et al. "Homeostasis model assessment: insulin resistance and beta - cell function." *Diabetologia*; 1985; 28 (7): 412 - 419.
- [12] Haffner, S. M., et al. "Role of obesity in the development of T2DM: epidemiological evidence." *Diabetes Care*; 1996; 19 (9): 1070 - 1080.