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A Study of the Correlation Between Serum Adenosine Deaminase Level and Fasting Serum Insulin Levels in Type 2 Diabetes Mellitus

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Keywords:
Type 2 Diabetes mellitus, serum adenosine deaminase level, fasting serum insulin levels

Abstract

Introduction:

Diabetes mellitus (DM) represents a group of common metabolic diseases that results in inadequate insulin secretion and diminished sensitivity to insulin. Adenosine mimics insulin's effects on lipid and glucose metabolism and blocks those effects on total hepatic glucose output, which raises the possibility that adenosine induces localised insulin resistance in the liver. The purine metabolism enzyme adenosine deaminase (ADA) breaks down adenosine into inosine and ammonia, which lowers the levels of adenosine.

Aims and objectives:

This was a study of correlation between serum adenosine deaminase level and fasting serum insulin levels in patients with type 2 diabetes mellitus.

Materials and methods:

It is a cross sectional study of 70 patients with newly diagnosed or known type 2 diabetes mellitus from outpatient and inpatient department.

Result:

Mildly positive correlation was found between serum ADA with FBG, PPBG and HbA1C with r value of 0.085,0.193 and 0.157 respectively. Serum fasting insulin had a mild correlation with FBG and significant correlation with PPBS (p=0.027) and HbA1C (p = 0.022). Serum adenosine deaminase was positively correlated with QUICKI (r=0.126) and serum fasting insulin was strongly and significantly correlated with QUICKI (p=0.0001). Serum adenosine deaminase level and serum fasting insulin were mildly correlated with each other (r=0.201 and p=0.991).

Conclusion:

DM type-2 is an important health problem globally. Diabetes is becoming more common and prevalent at a concerning rate. We discovered that patients with poorly managed type-2 DM had the highest levels of ADA. FBS and PPBS had a favourable correlation with ADA. It was discovered that ADA levels and QUICKI had an inversely proportional association. Even if a positive correlation was discovered, more future studies are required to adequately evaluate the function of ADA in the beginning and development of type-2 DM.

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1. Introduction

Diabetes mellitus (DM) represents a group of common metabolic diseases caused by genetic and environmental factors that result in inadequate insulin secretion and diminished sensitivity to endogenous or exogenous insulin.[1] It results in undesirable glucose levels and alterations in the metabolism of carbohydrates, fats, and proteins. Symptoms of hyperglycemia includes frequent urination, increased thirst and increased hunger.

Type-2 diabetes mellitus, which was earlier known as "noninsulin-dependent diabetes" or "adult-onset diabetes," represents 90–95% of all cases. Various factors can culminate into onset of diabetes. Beta-cells are not destroyed by the immune system, despite the fact that the precise causes are unknown, and there are no additional known diabetes-causing variables in the patients. Even with normal or raised insulin levels in type 2 diabetes mellitus patients, the failure to normalise blood glucose is caused by a relative shortage of insulin. Because of this, these patients' insulin secretion is compromised and cannot overcome resistance.[2] The purine metabolism enzyme adenosine deaminase (ADA), also known as adenosine amidohydrolase, catalyses the hydrolytic break down of adenosine into inosine and ammonia, resulting in a decrease in the concentrations of adenosine. Adenosine mimics insulin's effects on the metabolism of lipids and glucose in adipose tissue and the heart, but it blocks insulin's effects on the total synthesis of glucose in the liver, suggesting that adenosine is quietly responsible for isolated insulin resistance in the liver.[3] Adenosine primarily inhibits cyclic AMP accumulation, whereas insulin inhibits lipolysis through a noncyclic AMP-dependent mechanism. According to Fain et al., unless adenosine keeps cyclic AMP accumulation at low levels, insulin cannot suppress lipolysis since there are significant quantities of lipolytic chemicals.[4] Because T-lymphocyte activity was shown to be the primary biological function of ADA, it was thought to be an excellent indicator of cell-mediated immunity and to be crucial for lymphocyte development and differentiation.[5]. Human diabetes is often associated with impaired lymphocyte activity and increased susceptibility to infections. [6] Studies on type 2 diabetes have found increased ADA activity[3,7] and concluded that the ADA measures oxidative stress and peroxidation of lipid in diabetes. In addition, proinflammatory mediators enhance the stimulation of cytokine signalling proteins, which in turn inhibits the beta cells in pancreatic islets from activating the insulin signalling receptor.[8]

The quantitative insulin sensitivity check index (QUICKI) is a discrete mathematic translation of plasma insulin concentrations and fasting blood sugar that was developed empirically. QUICKI is identical to the HOMA model's simple equation form in every way except that QUCKI is calculated using a log transform of the insulin and glucose product.[9]

2. Materials and Methods

It is a cross-sectional study of 70 entrants with newly diagnosed or known type 2 diabetes mellitus who matched the diagnostic criteria mentioned below which was done in a hospital providing tertiary care from outpatient and inpatient department from January 2021 to June 2022. Patients on insulin therapy, with active infections like tuberculosis, acute lymphadenitis, enteric fever, infectious mononucleosis, leprosy, hepatitis A and B, drug-induced lymphadenitis and hematopoietic malignancies like Hodgkin's lymphoma were excluded from the study to rule out confounding factors.

Diagnostic criteria of type-2 Diabetes Mellitus[10]:

Fasting blood sugar levels more than or equivalent to 126 mg/dl (7.0 mmol/l)

OR

Increased frequency of urination, extreme thirst, unanticipated weight loss, and other symptoms, along with a random blood sugar measurement of 200 mg/dl (11.1 mmol/l) or higher

OR

Blood glucose level ≥200 mg/dl (11.1 mmol/l), measured two hours after a 75-gram glucose load

OR

HbA1c ≥ 6.5%.

After participants had received a complete explanation of the study, informed consent was acquired.

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An anthropometric assessment, FBS, PPBS, HbA1c, fasting blood insulin levels, and calculation of serum ADA levels were performed after the physical examination. The In-Patient/Out Patient cards, blood reports, and other pertinent sources were used to collect all the additional information necessary to rule out confounding variables.

Measurement of insulin resistance:

Fasting plasma insulin (I) estimation in IU/ml and fasting plasma glucose (G) estimation in mg/dl were used to calculate the quantitative insulin sensitivity check index (QUICKI)[9].

$QUICKI = 1/ \log(I_0) + \log(G_0)$.

Statistical Analysis

Using a statistical software for the social sciences (Version 20), the acquired data were statistically analysed. Results were shown as Mean (Median) SD, counts and percentages, and graphs. For the comparison of categorical variables, the Chi-square test has been used. Correlation between variables were calculated by Person's/Spearman's correlation. $p<0.05$ was considered statistically significant. All statistical tests were performed two-tailed.

3. Results

Patient characteristics:

Out of 70 entrants, 30 were females and 40 were males with a highest number of entrants (28) were in the age group of 60-69 years (Table 1 and 3; Figure 1 and 2) with mean and median value of 58.93 and 60 years respectively with standard deviation of 11.994 (Table 2).

PATIENT'S DISTRIBUTION ACCORDING TO AGE

Age (Years)	No. of patients	Percentage
< 20	1	1.4
20 - 29	1	1.4
30 - 39	1	1.4
40 - 49	10	14.3
50 - 59	17	24.3
60 - 69	28	40.0
70 - 79	9	12.9
80+	3	4.3
Total	70	100.0

TABLE – 1

	AGE
Mean	58.93
Median	60
Std. Deviation	11.994

Table -2 PATIENT'S DISTRIBUTION ACCORDING TO SEX

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Gender	No. of patients	Percentage
Female	30	42.9
Male	40	57.1
Total	70	100.0

Table -3

	Age	**ADA**	**F.INSULIN**	**FBS**	**PPBS**	**HbA1C**
Mean	58.93	22.8109	13.7349	174.31	219.86	8.4586
Median	60.00	20.5050	11.8650	165.50	207	7.9500
Std. deviation	11.994	10.318	10.61600	51.903	59.384	2.18920

Table -4

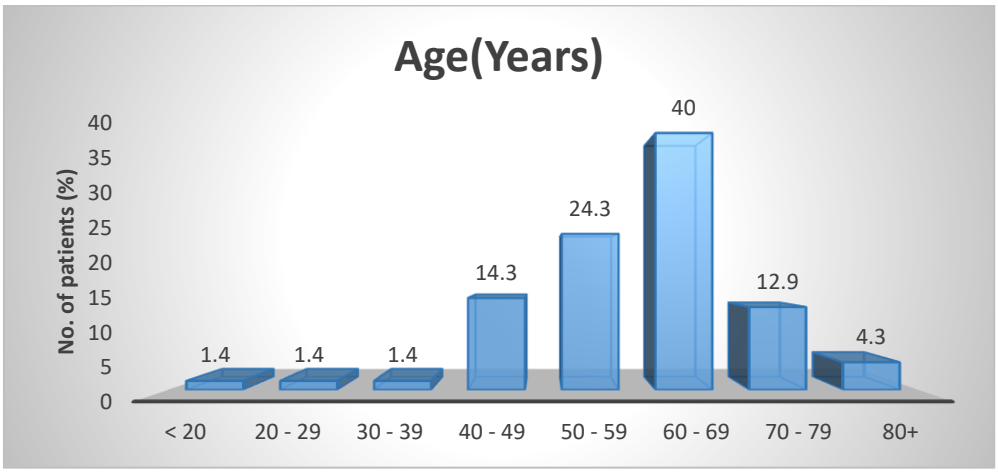

Figure- 1

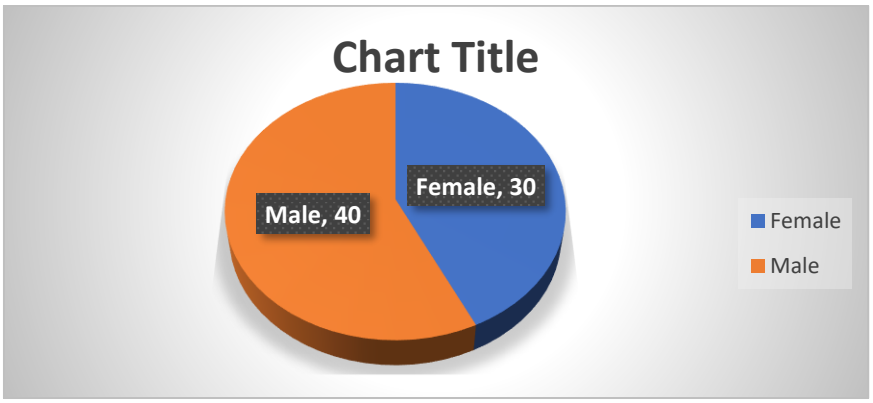

Figure-2

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The mean and median value of serum ADA was 22.8109 and 20.5050; serum fasting insulin was 13.7349 and 11.8650; FBS was 174.31 and 165.50; PPBS was 219.86 and 207; HbA1C was 8.45 and 7.95. (Table 4).

Mildly positive correlation was found between serum ADA with FBG, PPBG and HbA1C with r value of 0.085,0.193 and 0.157 respectively. Serum fasting insulin had a mild correlation with FBG and significant correlation with PPBS (p=0.027) and HbA1C (p = 0.022). QUICKI and serum adenosine deaminase showed a positive correlation (r=0.126) and serum fasting insulin was strongly and significantly correlated with QUICKI (p=0.0001). Fasting insulin and serum adenosine deaminase levels had a mild correlation. (r=0.201 and p=0.991).

Association between serum ADA and Fasting blood glucose levels: In this study we found mild positive correlation between serum ADA and serum fasting blood glucose level with p value = 0.483 and r= 0.085 **(Figure 3)** in contrast to studies done by Jae-GeunL et al.[11] ,Dharamveer et al.[12], Amandeep et al.[13] and Nisha et al.[14]

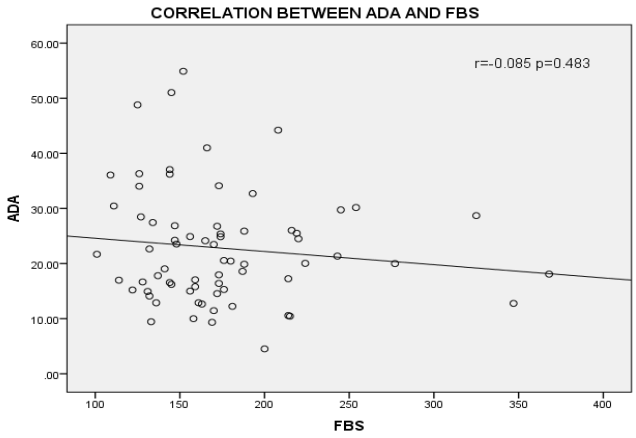

Figure – 3

Association between serum ADA and Post prandial blood glucose levels:

A mild positive correlation was found between serum ADA and PPBS level with with r value=0.193 and p value=0.385 **(Figure 4)**, in concordance with studies by Jae-GeunL et al.[11] ,Dharamveer et al.[12], Amandeep et al.[13] and Nisha et al.[14]

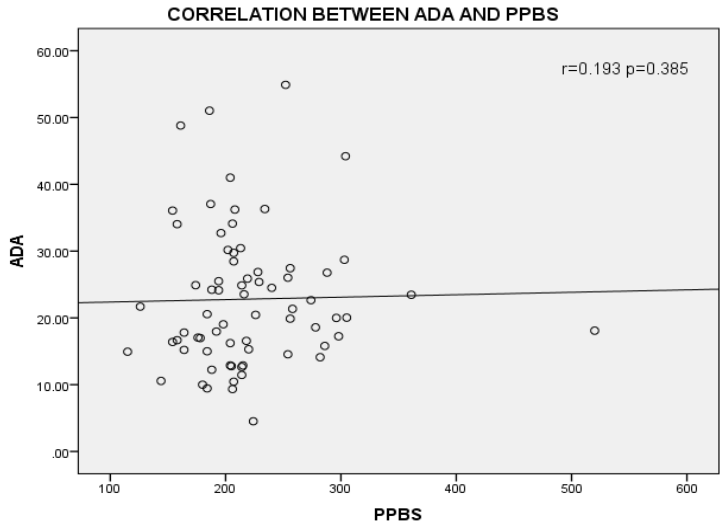

Figure – 4

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Association between serum ADA and HbA1C:

Correlation between serum ADA and HbA1C with r value= 0.157 and p value = 0.395 was mildly positive. **(Figure 5).** A higher HbA1C level was accompanied by higher serum ADA levels similar to that encountered in previous studies done Jae-GeunL et al.[11], Dharamveer et al.[12], Amandeep et al.[13] Nisha et al.[14] and also by Muthiah et al. [15]

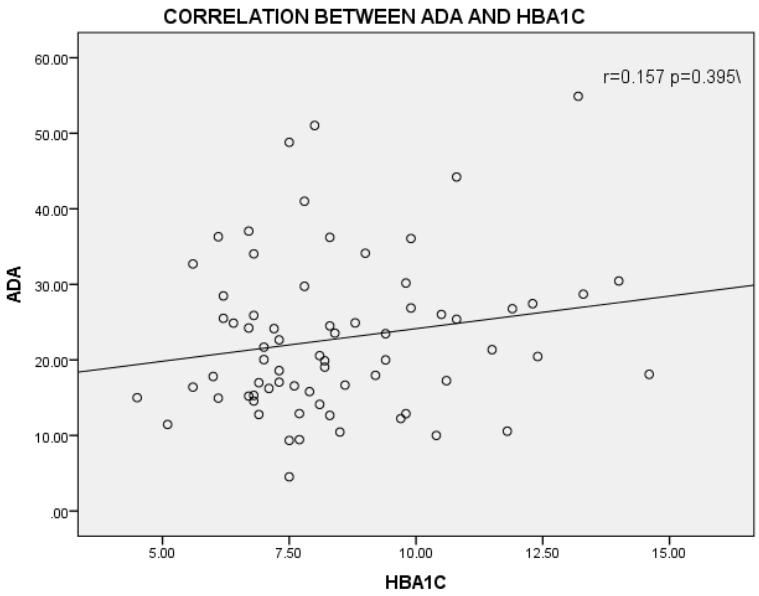

Figure – 5

Association between fasting serum insulin and fasting blood glucose level:

There was a mild positive correlation between fasting serum insulin and fasting blood glucose level with r value= 0.144 and p value = 0.234 **(Figure 6)**.

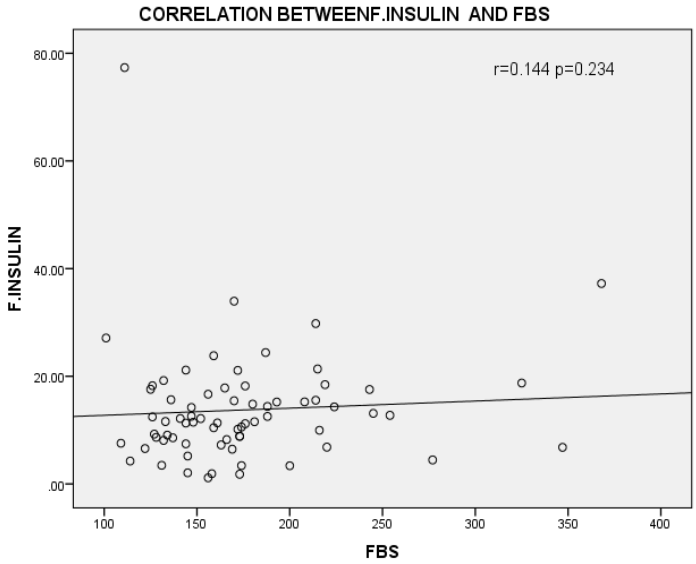

Figure – 6

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Association between fasting serum insulin and PPBS:

We observed a weakly positive connection between serum fasting insulin level and serum PPBS level that was statistically significant with r value= 0.265 and p value =0.027 **(Figure 7)**. This result can be explained on basis of a study done by Gopalratnam Raman et al which states that increased PPBG produces dyslipidaemia, which activates prothrombotic activity and reduces insulin sensitivity which further causes insulin resistance and an increase in fasting serum insulin. [16]

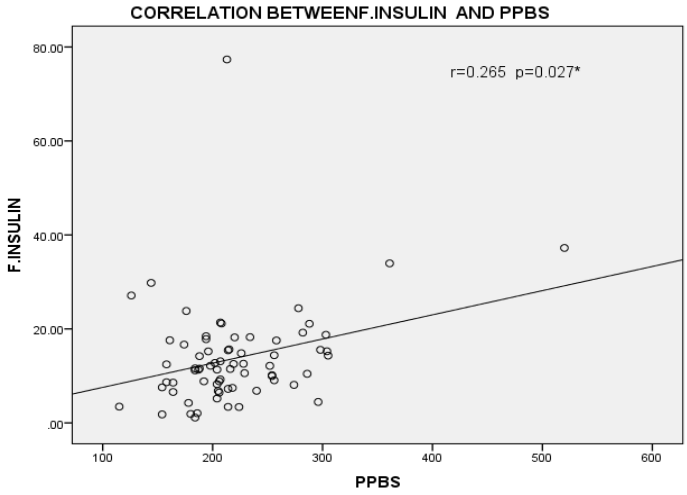

Figure – 7

Association of fasting serum insulin level and HbA1C level:

Another statistically significant finding in this study was the mild positive correlation between serum fasting insulin level and HbA1C level with r value = 0.273 and p value= 0.022 **(Figure 8)**. A study by Sitasuwan et al also shows HbA1c being significantly correlated with insulin resistance. [17]

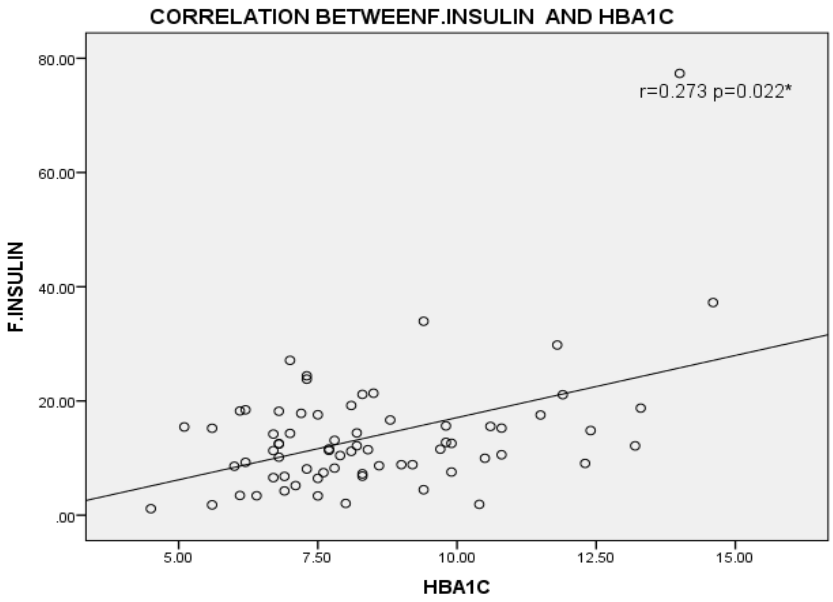

Figure – 8

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Association between fasting serum insulin and serum ADA levels:

This study demonstrated a mild positive correlation between serum ADA level and serum fasting insulin levels with r value= 0.201 and p value = 0.991 **(Figure 9)** which is also shown by Jae-GeunL et al.[11] ,Dharamveer et al.[12], Amandeep et al.[13] , Nisha et al.[14] and also by Muthiah et al. [15]

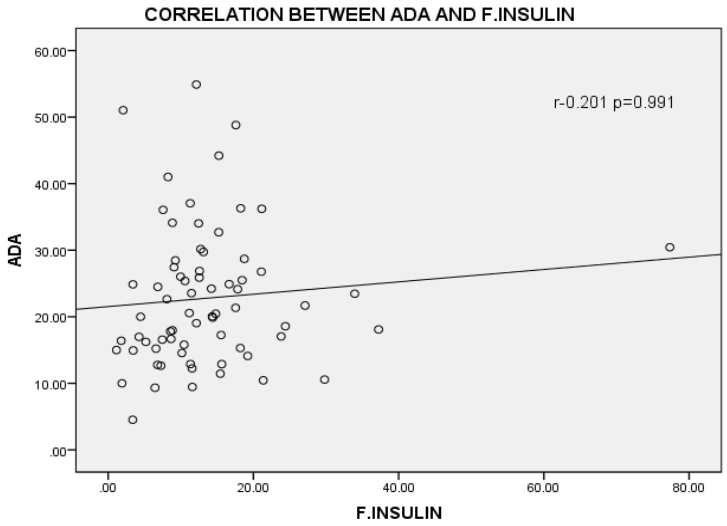

Figure – 9

Association between serum ADA and QUCKI:

A mild negative correlation between serum ADA and QUCKI was noted with r=0.126 and p=0.299 **(Figure 10)** which is also shown by Muthiah et al. [15] which demonstrates a decrease in ADA levels in patients with rising QUICKI values.

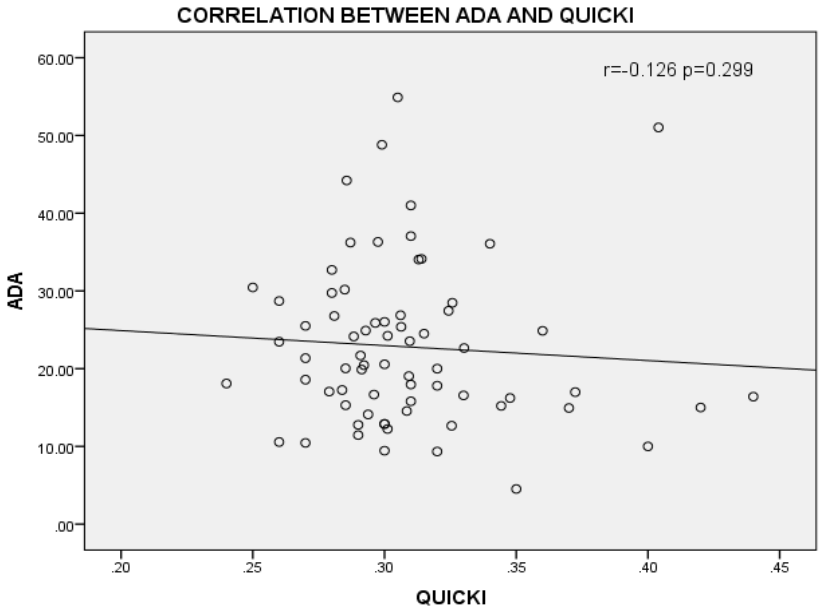

Figure – 10

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Association of serum fasting insulin with QUCKI:

A strong statistically significant correlation between serum fasting insulin and QUCKI was obtained with r=0.901 and p= 0.0001 **(Figure 11)** which is also noted by Katz et al [18] , Navneet et al and Gitanjali et al [19].

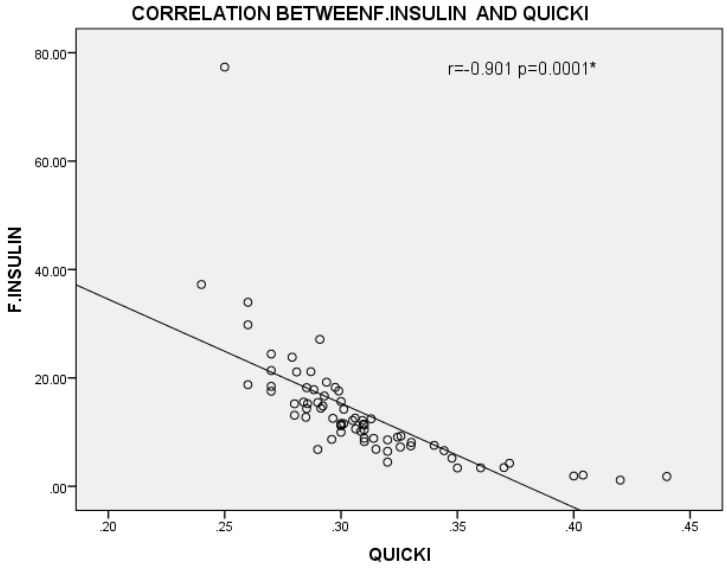

Figure – 11

4. Discussion

ADA is distributed differently in various tissues, with lymphoid and fatty tissues containing the most of it. [(20)]. Adenosine plays a known role in absorption of glucose by insulin in skeletal muscle; when its activity increases, cells become less likely to absorb glucose, which adds to insulin resistance. [21] Adenosine enhances gluconeogenesis and glycogenolysis both in vitro and in vivo research, boosts glucose synthesis, and affects cardiac functions through its receptors, primarily A1 and A2 adenosine receptors. [22]

In T2DM, an increase in ADA blood levels affects insulin metabolism, particularly in adipose tissues where it increases lipolysis, disrupts anti-lipolysis function, and intensifies hyperlipidemia. Adipocytes' oxidative phosphorylation and ATP retention are brought about by the substantial amount of fatty free acids (FFA) due to enhanced lipolysis activity.[23] Additionally, ADA decreases sensitivity to insulin in adipocytes and affects PKB (protein kinase B) synthesis in the insulin post receptor phase. [24] Along with that, ADA reduces GLUT4 availability to the cell membrane for glucose transporters.[25] As a result, diabetic adipocyte cells might demand greater insulin concentration. [26] The interactivity of ADA with DPP-4 may enable T-cells to proliferate and produce more cytokines, which can disrupt insulin signalling because adenosine induces apoptosis and prevents T-lymphocyte differentiation by activating P1 adenosine receptors [27]. Proteins in the acute phase are stimulated by cytokines. The acute phase protein regulates homeostasis and has survival benefits in the short term, but it is troublesome in the long run. [28] Certain proinflammatory cytokines, such IL-6 and TNF-alpha are increased by insulin resistance and hyperglycemia.[29] Other significant risk factors for T2DM include ageing, sedentary lifestyle, obesity, food intake, smoking, and increased peripheral cytokines.[30] ADA deficiency is also linked to compromised immunological processes. Thus, by lowering ADA activity, T-lymphocyte activity, inflammation, and insulin sensitivity—all of which are linked to the pathophysiology of T2DM—can be improved. Further evidence suggests that ADA modifies insulin's bioactivity.

The increased serum ADA levels in T2DM patients and glycemic control have rarely been linked in studies.[31] It has also been noted that ADA levels are higher in those with type 2 diabetes and are favourably connected with fasting blood sugar and insulin levels. Our study demonstrates higher serum ADA levels in T2DM

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participants, which is consistent with other studies. A favourable association between the fasting blood insulin level and the serum ADA level was also observed, which may contribute to the pathogenesis of T2DM patients.

5. Conclusion

DM type-2 is an important health problem globally. Diabetes is becoming more common and prevalent at an alarming rate. We discovered that patients with poorly managed type-2 DM had the highest levels of ADA. FBS and PPBS had a favourable correlation with ADA. It was discovered that ADA levels and QUICKI had an inversely proportional association. Even if a positive correlation was discovered, more future studies are required to adequately evaluate the function of ADA in the beginning and development of type-2 DM.

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