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"Prospective Study on Fetuin-A and Adiponectin in Type 2 Diabetes Mellitus: Correlations with HbA1c, C-Reactive Protein, Insulin, and Waist Circumference"

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KEYWORDS

Fetuin-A, Adiponectin, Type 2 Diabetes Mellitus, HbA1c, CRP, Insulin, Waist Circumference

Abstract

Background: Recent evidence highlights significant associations between fetuin-A and, adiponectin, characterized by hyperglycemia, central obesity, and insulin resistance. Notably, these two proteins exhibit opposing biological functions.

Objective: To assess prospective correlations between Fetuin-A, Adiponectin, and metabolic/inflammatory markers in T2DM patients.

Methods: The present prospective case-control study was conducted over 160 newly diagnosed T2DM patients and compare with age-matched apparently 160 healthy subjects. Insulin levels were quantitated by the electrochemiluminescence method. Serum C-reactive protein (CRP) concentrations were detected by the rate nephelometry assay. Serum levels of fetuin-A and adiponectin were determined by an ELISA method. Unpaired *t*-test was employed for the numerical investigational data, chi-squared test for categorical data, and Pearson's correlation for the correlation analysis.

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Results: The mean age of cases (53.77 ± 9.40 years) was significantly higher than that of controls (52.42±9.30 years) (p =0.198). Cases had higher levels of CRP (5.46 ± 2.00 mg/L), Fetuin-A (812.24 ± 100.54 µg/mL), and Fetuin-A/Adiponectin ratio (163.14 ± 41.51) compared to controls (2.54 ± 1.00 mg/L, 650.81 ± 82.61 µg/mL, and 74.83 ± 16.67, respectively) ($p < 0.001$ for all). Conversely, Adiponectin levels were significantly lower in cases (5.21 ± 1.12 µg/mL) compared to controls (8.95 ± 1.41 µg/mL) ($p < 0.001$). Adiponectin showed strong negative correlations with waist circumference ($r = -0.462$), HbA1c ($r = -0.750$), insulin ($r = -0.550$), and CRP ($r = -0.548$) ($p < 0.001$ for all). In contrast, fetuin-A and the fetuin-A/adiponectin ratio showed positive correlations with these markers, with fetuin-A/adiponectin ratio having the strongest correlation with HbA1c ($r = 0.732$) and CRP ($r = 0.561$).

Conclusion: The strong correlations between adiponectin and fetuin-A with HbA1c, CRP, insulin, and waist circumference underscore the potential role of these biomarkers in predicting and managing type 2 diabetes and its complications.

Introduction

Diabetes mellitus (DM) is defined as a heterogeneous group of diseases, characterized by a state of chronic hyperglycemia, resulting from a diversity of etiologies, environmental and genetic, acting jointly. The underlying cause of diabetes is the defective production or action of insulin, a hormone that controls glucose, fat, and amino acid metabolism.[1]

Insulin resistance is a multifaceted syndrome responsible for the future development of type-2 diabetes, obesity, hypertension, dyslipidaemia and atherosclerotic cardiovascular diseases (CVD).[2] There is increasing evidence supporting the fact that by the time glucose tolerance or, fasting glucose levels become impaired, appreciable β-Cell destruction may have already occurred.[3] Thus, it seems likely that attempts to prevent type-2 diabetes will be more successful if intervention is commenced when blood glucose levels are still in the normal range.

There are, however, patterns of diabetes incidence that are related to the geographical distribution of diabetes in India. Rough estimates show that the prevalence of diabetes in rural populations is one-quarter that of urban population for India and other Indian sub-continent countries such as Bangladesh, Nepal, Bhutan, and Sri Lanka.[4,5] Preliminary results from a large community study conducted by the Indian Council of Medical research (ICMR) revealed that a lower proportion of the population is affected in states of Northern India (Chandigarh 0.12 million, Jharkhand 0.96 million) as compared to Maharashtra (9.2 million) and Tamil Nadu (4.8 million).[4]

T2DM usually develops gradually—the early stages of the disease may be asymptomatic and undetected for several years. Initial symptoms commonly include polydipsia, polyuria, polyphagia, and eventually, weight loss. This chronic disease triggers a series of complications with a high degree of morbidity and mortality, resulting in a significant number of medical consultations, hospitalizations, disabilities, and deaths. Examples of these multisystemic complications include microvascular events, such as retinopathy, nephropathy, and neuropathy, and macrovascular events, including ischemic heart disease, stroke, and peripheral vascular disease.[6] A significant fraction of T2DM patients often present advanced complications that can be difficult to manage and costly to treat. In this context, the high incidence of T2DM presents a heavy burden on worldwide public health systems. Screening strategies have a positive impact on the quality of life and reduction of health costs since they allow early diagnosis lowering the prevalence of underdiagnosis, thus reducing the generation of complications, which in the long run decreases the pressure on health systems.[7,8]

Currently, there are several challenges in the management of T2DM that need to be addressed. On the technical side, there is a need for novel, more comprehensive strategies for optimal screening, early diagnosis, and adequate management of T2DM. Approaches combining the use of resources for risk assessment, such as Finnish Diabetes Risk Score

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(FINDRISC),[9] along with more effective biomarkers for screening and progression T2DM, have a higher probability of success in managing the global diabetes epidemic.

Adiponectin, the most abundant anti-atherogenic and anti-inflammatory adipocytokine found in circulation has direct effects on glucose and lipid metabolism, improves insulin sensitivity and central fat distribution.[10] Adiponectin levels are inversely correlated with visceral adiposity.[11] A lower level of adiponectin is associated with insulin resistance, obesity, metabolic syndrome (MetS) MetS and CVD.[12] Low level of circulating adiponectin may be used as a possible biomarker for MetS.[13]

Lower levels of adiponectin were observed in patients with high blood pressure, hyperglycaemia, low HDL-C, and hypertriglyceridemia, also in obese patients with MetS.[14] Adiponectin increases the sensitivity to insulin through several mechanisms. AdipoR1 and AdipoR2 are transmembrane receptors, whose carboxyl terminal group(C-terminal) is located outside the membrane, and the amino terminal group (N-terminal) inside.[15] When adiponectin attaches to its receptor it activates adenosine mono phosphate (AMP) kinase,[16] promoting so glucose uptake by muscles via intracellular translocation of the GLUT4 transporters. Simultaneously, it hampers gluconeogenesis by inhibiting the hepatic enzyme phosphoenolpyruvate carboxylase, inhibits the synthesis of fatty acids and stimulates their oxidation.[17]

Fetuin-A, known as alpha-2-Heremans–Schmid glycoprotein, has a multifunctional effect on human health, especially in adulthood, and has roles in the pathogenesis of many different chronic diseases.[18] The literature on the relationship between serum fetuin-A and obesity has yielded conflicting results.[19] It is indicated that obese individuals often exhibit higher serum fetuin-A levels.[20] Zhang et al. found a positive association between serum fetuin-A levels and waist circumference (cm).[21] El-shaer et al. concluded that serum fetuin-A levels are directly proportional to the degree of obesity and its complications and that exercise may play a protective role by reducing fetuin-A levels.[22] However, the relationship between fetuin-A and obesity and the mechanisms underlying this relationship have not yet been fully clarified.[23] Some potential mechanisms have been proposed for how fetuin-A increases the risk of developing obesity. One of these potential mechanisms is that fetuin-A may increase the risk of obesity by inhibiting endogenous insulin receptor tyrosine kinase and decreasing hepatic insulin sensitivity.[24]

Therefore, fetuin-A and adiponectin are speculated to work together in the metabolic balance, and fetuin-A/adiponectin ratio (F/A ratio) is expected to show more sensitive performance in assessing metabolic disorder than fetuin-A or adiponectin alone. In this study, we attempt to Fetuin-A and Adiponectin level in Type 2 Diabetes Mellitus and Correlations with HbA1c, C-Reactive Protein, Insulin, and Waist Circumference.

Materials and Methods

Subject Selection: The present prospective case-control study was included a total 320 subjects. In which, 160 of newly diagnosed T2DM and 160 of age-matched healthy controls individuals aged 18-70 years were enrolled from the Out Patient Department of Medicine, Medical College of University. Subjects aged between 18-70years.

Cases: Newly diagnosed T2DM subjects (0-1years disease duration without any microvascular complications) were defined based on the HbA1c (≥6.5%) (ADA 2025). Individuals with other types of diabetes, severe co-morbidities and those on medications affecting adipokine levels were excluded from the study. Ethical approval from the Institutional Ethical Committee was secured before initiating the study. Written informed consent was taken from each study subject.

Anthropometric Parameters: Waist circumference (WC) was measured at the umbilicus level. The WC cut-offs WC for diagnosis of abdominal obesity in Asian Indian males and females are ≥90 cm and ≥80 cm, respectively.[25]

Sample Collection: Blood samples were collected from patients in morning, after an overnight fasting for at least 10 hours. 4 mL of venous blood (1.5 mL EDTA, 1.5 mL fluoride, and 1mL plain) was withdrawn from the antecubital vein. To separate plasma, blood samples were centrifuged at 3000 rpm for 10 minutes.

Laboratory Investigations: Serum C-reactive protein (CRP) concentrations were detected by the rate nephelometry assay.[26] Insulin levels were quantitated by

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the electrochemiluminescence method.[26] Serum levels of fetuin-A and adiponectin were determined by an ELISA method. The adiponectin-to-fetuin-A ratio was calculated.[26]

Statistical analysis

The SPSS version 23.0; SPSS Inc; Chicago IL, USA was used to interpret the statistical data. For the descriptive research data, categorical variables were expressed as frequency and percentages, whereas continuous variables were expressed as mean ± standard deviation. Unpaired *t*-test was employed for the numerical investigational data, chi-squared test for categorical data, and Pearson's correlation for the correlation analysis. A $p < 0.05$ level of significance was applied.

Result

The mean age of cases (53.77 ± 9.40 years) was insignificantly higher than that of controls (52.42 ± 9.30 years) ($p = 0.198$) [Table 1].

Cases had a significantly higher mean waist circumference (101.10 ± 11.94 cm) compared to controls (86.28 ± 10.36 cm) ($p < 0.001$). Additionally, cases had significantly higher mean HbA1c levels (8.53 ± 1.01%) and insulin levels (17.65 ± 4.92 µIU/mL) compared to controls (5.21 ± 0.31% and 10.04 ± 2.83 µIU/mL, respectively) ($p < 0.001$ for both comparisons) [Table 1].

Cases had higher levels of CRP (5.46 ± 2.00 mg/L), Fetuin-A (812.24 ± 100.54 µg/mL), and Fetuin-A/Adiponectin ratio (163.14 ± 41.51) compared to controls (2.54 ± 1.00 mg/L, 650.81 ± 82.61 µg/mL, and 74.83 ± 16.67, respectively) ($p < 0.001$ for all). Conversely, Adiponectin levels were significantly lower in cases (5.21 ± 1.12 µg/mL) compared to controls (8.95 ± 1.41 µg/mL) ($p < 0.001$) [Table 1].

Adiponectin showed strong negative correlations with waist circumference ($r = -0.462$), HbA1c ($r = -0.750$), insulin ($r = -0.550$), and CRP ($r = -0.548$) ($p < 0.001$ for all). In contrast, fetuin-A and the fetuin-A/adiponectin ratio showed positive correlations with these markers, with fetuin-A/adiponectin ratio having the strongest correlation with HbA1c ($r = 0.732$) and CRP ($r = 0.561$) [Table 2].

Discussion

Type 2 diabetes is characterized by inadequate insulin secretion and insulin resistance in the target tissues. Insulin mediates its action through phosphorylation of the insulin receptor. Fetuin-A inhibits insulin receptor autophosphorylation.[27] In present study was conducted to find the associations between fetuin-A, adiponectin, and F/A ratio, were correlated with HbA1c, CRP, insulin, and waist circumference in newly diagnosed T2DM patients.

The cases had an insignificantly higher mean age (53.77 ± 9.40 years) compared to controls (52.42 ± 9.30 years) ($p = 0.198$). Both groups had a similar gender distribution, with males accounting for 52.5% of cases and 53.8% of controls, and females accounting for 47.5% of cases and 46.3% of controls. **Tiwari ON &Nigoskar S[28]** reported that in case groups, 66.2% male and rest were female and in control groups 60.0% were male and 40.0% were female patients. They found that the statistically insignificant higher older age population in case group distribution in compare to control group ($p > 0.05$).

The present study noted that Cases had a significantly higher mean waist circumference (101.10 ± 11.94 cm) compared to controls (86.28 ± 10.36 cm) ($p < 0.001$). Additionally, cases had significantly higher mean HbA1c levels (8.53 ± 1.01%) and insulin levels (17.65 ± 4.92 µIU/mL) compared to controls (5.21 ± 0.31% and 10.04 ± 2.83 µIU/mL, respectively) ($p < 0.001$ for both comparisons). **Tiwari ON & Nigoskar S[28]** reported that the waist circumference in case group in compare to control group ($p > 0.05$). Blood Sugar level (Insulin level and HbA1c) was significantly higher in the case group in comparison to control group ($p < 0.001$).

The present study showed highly significant increase in serum insulin, and serum fetuin-A, in the diabetic group compared with the control group. Our results showed significant positive correlations between fetuin-A levels and fasting insulin levels in patients with T2DM and this agreed with the studies conducted by **Jung et al[29]** who demonstrated that serum fetuin-A is significantly associated with IR, and **Graham et al[30]** who showed that fetuin-A is positively correlated with insulin resistance. These results were previously reported by **Wallace et al[31]**, who demonstrated that fetuin-A levels were correlated with fasting insulin levels in obese patients, suggesting a potential link between fetuin-A and insulin

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resistance. **Stefen et al**[32] had demonstrated that fetuin-A was correlated with insulin resistance and fat accumulation in the liver. **Li et al**[33] reported that fetuin-A, which is predominantly secreted by the liver, is found to be related to the accumulation of fat in the liver, insulin resistance, type 2 diabetes, and cardiovascular diseases.

Dasgupta et al[34] reported that the liver secreted protein fetuin-A induces insulin resistance, and circulating fetuin-A is elevated in insulin resistance and fatty liver in humans. In agreement with these data, **Emoto et al**[35] had shown that high levels of circulating fetuin-A are associated with insulin resistance in humans, suggesting that fetuin-A may represent a mechanism involved in the pathophysiology of type 2 diabetes.

In our results, CRP also showed marked increase in type 2 diabetes when compared with group control. This agrees with a study reported by **Kotronen and Yki-Järvinen**[36] which showed that serum fetuin-A levels were increased in diabetic patients when compared with case–control individuals and demonstrated a positive correlation between serum fetuin-A and CRP levels. These results agreed with our results, as there was a positive correlation between serum fetuin-A and CRP levels ($r = 0.561$, $P < 0.01$). In another study, **Ahmed et al**[37] reported that there was a positive correlation between serum fetuin-A and CRP levels ($r = 0.786$, $P < 0.01$).

Adiponectin is an anti-inflammatory adipokine. The level of adiponectin was apparently less in diabetes group in comparison to the healthy control group. Supporting evidence shows significantly low adiponectin level in prediabetic and hyperglycaemic group as compared to normoglycemic group.[38] Further supporting studies also show significant differences in adiponectin level in both the subject groups as compared to control group.[39,40] According to **Maggio CA et al**[41] adiponectin has been found as one of the important factors predicting prediabetes. The protective role of adiponectin against hyperglycaemia can be correlated with its insulin-sensitizing effects.[42] Mechanistic approaches demonstrate that adiponectin stimulates AMP-dependent protein kinases,[43] and thereby triggers insulin sensitivity by enhancing glucose cellular uptake and fatty acid oxidation in the liver. According to Okada-Iwabu *et al*., oral supplementation of AdipoR agonist can serve as a promising therapeutic option for insulin resistance and diabetes.[44]

Strength and Limitations of Study

This study benefits from a prospective design enabling causal inference and includes well-matched case-control groups (160 T2DM and 160 healthy controls), allowing robust comparative analysis. It investigates correlations between fetuin-A, adiponectin, and key metabolic markers like HbA1c, CRP, insulin, and waist circumference. However, its single-center nature may limit generalizability, and the age range (18–70 years) might overlook age-related variations. Additionally, confounding factors such as medication and lifestyle were not explicitly controlled, and the absence of follow-up data restricts longitudinal insights.

Conclusion

In conclusion, this prospective study highlights the significant associations between fetuin-A, adiponectin, and various metabolic markers in type 2 diabetes mellitus. The findings suggest that fetuin-A and adiponectin have opposing effects on metabolic health, with adiponectin exhibiting protective effects and fetuin-A being associated with adverse metabolic outcomes. The strong correlations between adiponectin and fetuin-A with HbA1c, CRP, insulin, and waist circumference underscore the potential role of these biomarkers in predicting and managing type 2 diabetes and its complications. These results have implications for the development of novel therapeutic strategies targeting fetuin-A and adiponectin to improve glycemic control and reduce inflammation in patients with type 2 diabetes.

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Table 1: Distribution of Anthropometric and clinical Characteristics of case and control

Variables	Case (n=160) Mean ± SD	Control (n=160) Mean ± SD	p value

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Age Group (Years)		53.77±9.40	52.42±9.30	0.198 #
Gender	**Male**	84 (52.5%)	86 (53.8%)	0.823 *
	Female	76 (47.5%)	74 (46.3%)	
Waist Circumference (cm)		101.10±11.94	86.28±10.36	**<0.001 #**
HbA1c (%)		8.53±1.01	5.21±0.31	**<0.001 #**
Insulin (µIU/mL)		17.65±4.92	10.04±2.83	**<0.001 #**
CRP (mg/L)		5.46±2.00	2.54±1.00	**<0.001 #**
Adiponectin (µg/mL)		5.21±1.12	8.95±1.41	**<0.001 #**
Fetuin-A (µg/mL)		812.24±100.54	650.81±82.61	**<0.001 #**
Fetuin-A/ Adiponectin ratio		163.14±41.51	74.83±16.67	**<0.001 #**

*Chi Square test; # Independent Sample t test

HbA1c: Glycated Heamoglobin; CRP: C-Reactive Protein; WC: Waist Circumference.

Table 2: Bivariate Pearson Correlation Analysis of variables among cases.

	Adiponectin (r-value)	**Fetuin-A (r-value)**	**Fetuin-A/Adiponectin Ratio (r-value)**
WC (cm)	-0.462**	0.366**	0.456**
HbA1c (%)	-0.750**	0.582**	0.732**
Insulin (µIU/mL)	-0.550**	0.457**	0.543**
CRP (mg/L)	-0.548**	0.417**	0.561**

**. Correlation is significant at the 0.01 level (2-tailed).

HbA1c: Glycated Heamoglobin; CRP: C-Reactive Protein; WC: Waist Circumference.

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