DOI: 10.7860/NJLM/2022/56393.2652

Original Article

Biochemistry Section

Association of Serum Adiponectin Level with Dyslipidaemia in North Indian Male Population: A Case-control Study

RANADIP MUKHERJEE[1], MANISH KUMAR MISRA[2], SUMERU SAMANTA[3], KAJAL MAHAJAN[4], DEVAJIT SARMAH[5], MOHUA ROY MUKHERJEE[6]

ABSTRACT

Introduction: Adiponectin is the most abundant adipocytokines secreted from adipose tissues and circulates in considerably high concentration in human plasma. Circulating adiponectin levels are decreased in obese subjects and this decrease has been thought to play a crucial role in the early development of atherosclerosis and cardiovascular diseases. Changes in adiponectin concentration has been reported in dyslipidaemic subjects, but the evidence is controversial and no study has been conducted in north Indian population. Moreover, low molecular adiponectin seems to be linked with a worse lipid profile leading to dyslipidaemic through an association with triglyceride but the exact role of adiponectin in modulating lipid fraction is not well established.

Aim: To correlate the level of serum adiponectin with lipid fractions in dyslipidaemic male subjects and also to compare them with apparently healthy individuals.

Materials and Methods: This case-control study was conducted from April 2015 to November 2016 in the Biochemistry Department of Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India. A total of 70 non diabetic dyslipidaemic male subjects between the age group 35 years to 55 years were selected and all the biochemical parameters (adiponectin, fasting plasma glucose, lipid profile) were evaluated and compared with 70 apparently healthy controls. Statistical analysis was performed by licensed version of Statistical Package for Social Sciences (SPSS) 16.0 software. All the data were expressed in "mean±SD". Student 't' test was also applied to see statistical significance in adiponectin levels between dyslipidaemic subjects and healthy controls.

Results: The study shows mean±SD of age in dyslipidaemic group was 43.61±4.85 years and for control group was 43.53±5.53 years. The mean±SD of BMI in dyslipidaemic group 25.72±2.43 was significantly higher than control group 23.42±1.56 with p-value <0.0001. The serum adiponectin concentration was significantly reduced in dyslipidaemic subjects 5.11±2.04 µg/mL as compared to healthy control 6.79±1.37 µg/mL with p-value <0.0001. Serum total cholesterol, triglyceride and Low Density Lipoprotein (LDL)-cholesterol were found to be negatively correlated with serum adiponectin (r= -0.89, -0.76 and -0.74) and positively correlated with High Density Lipoprotein (HDL)-cholesterol (r=0.70).

Conclusion: The present study revealed that hypoadiponectinemia is associated with dyslipidaemic in men. The main observation of our present study, however, is that in dyslipidaemic subjects, lower levels of adiponectin were associated with high total cholesterol, triglyceride, LDL-cholesterol and reduced HDL cholesterol, though more extensive, multicentric, prospective research with increase sample size could obtain wider insights.

Keywords: Adipocytokines, Atherosclerosis, Deranged lipid profile, Obesity

INTRODUCTION

Adipose tissue is nowadays recognized as a highly active metabolic endocrine gland which secretes a variety of biologically active substances, including adipocytokines, growth factors into blood stream [1]. These molecules have effects on autocrine, endocrine and also paracrine glands and show control over various tissues like brain, liver and skeletal muscle. Adiponectin molecule also control thermogenesis, production and secretion of thyroid and reproductive hormones, body immunity and nutrition [2]. Among the various adipokines, adiponectin is the most abundant adipocytokines which consists of 244 amino acid peptides [3]. Though various adipocytokines are secreted from adipose tissue, only adiponectin represents anti-inflammatory and antiatherogenic properties [4]. Though adiponectin is secreted from adipose tissue its levels are surprisingly decreased in obesity and also in type 2 diabetes [5-7]. A reduce level of adiponectin is an independent risk factor for metabolic syndrome, insulin resistance and diabetes mellitus [2,7]. It is also suggested that low-adiponectin levels are correlated with coronary artery disease [4]. Adiponectin circulates at high levels in human plasma represents for approximately 0.01% of all plasma proteins in normal individual [3].

Studies suggest that adiponectin correlates with various parameters of lipoprotein fractions and mainly its association with High Density Lipoprotein-Cholestrol (HDL-C) level and triglyceride [8-12]. Moreover, adiponectin exerts its effect by inducing an increase in serum HDL concentration and, in addition, it decreases serum Triglyceride (TG) [9,10]. Adiponectin is known to lower the synthesis of free fatty acids and to stimulate β-oxidation [11,13]. A study carried out on obese subjects suggested that impaired lipid fraction is characterised by reduce levels of serum adiponectin levels compared with obese subjects with normal lipid fraction who were associated with high adiponectin concentration [14].

Nowadays worldwide dyslipidaemia becomes a highly prevalent disorder which is associated with various metabolic complications which results in decreased longevity and increased morbidity. Dyslipidaemia results from various metabolic complications like insulin resistance, obesity, severe hypertension, and also from cardiovascular disease [9]. The Indian Council of Medical Research (ICMR)-INDIAB study revealed that dyslipidaemia is highly prevalent in urban as well as rural India [15]. Dyslipidaemia has been closely associated to the pathophysiology and also is a key independent risk factor for Cardiovascular Disease (CVD) worldwide. Low Molecular Weight (LMW) adiponectin seems to be linked with a worse lipid profile leading to dyslipidaemic through an association with triglyceride but the exact role of adiponectin in modulating lipid fraction is not well established [11,13]. Studies

regarding serum adiponectin levels in dyslipidaemic are very limited and they were conducted mainly on Japanese and European population [8,10] and to our best knowledge no data is available in north Indian population which drag our interest to evaluate serum adiponectin in cases with dyslipidaemia. We chose male subjects only because sex difference had been reported to alter levels of serum adiponectin [6,13]. Thus, the present study was conducted to check the correlation of adiponectin level with lipid parameters in North Indian dyslipidaemic males and also to compare it with apparently healthy individuals.

MATERIALS AND METHODS

This case-control study was conducted in Biochemistry Department with collaboration with Medicine Department at Rajshree Medical Research institute, Bareilly from April 2015 to November 2016. Ethical clearance permission was obtained from RMRI Institutional Ethical Committee to conduct the study (reference number RMRI. Bly/2014-14/101).

Inclusion criteria: Newly diagnosed dyslipidaemic male subjects, aged between 35-55 years were included in present study. Selection criteria for dyslipidaemic subjects were based on the American Heart Association's classification and National Cholesterol Education Programme (NCEP) guidelines: total cholesterol >200 mg/dL, triglycerides >150 mg/dL, LDL >130 mg/dL and HDL <40 mg/dL [16,17]. Age and sex matched apparently healthy subjects whose lipid profile were within normal reference range were selected as control [18]. The control subjects were selected from the population who turned up for general health check-up in RMRI hospital.

Exclusion criteria: Known cases of diabetes, HIV, any renal or liver disorders, morbidly obese subjects with BMI >35, hypertensive, cancer patients and patients taking hypolipidaemic agents or medication were excluded.

Sample size calculation: Sample size was calculated by the formula [19].

$$n = \frac{r+1}{r} \frac{\sigma^2 (Z_{\alpha/2}+Z_{1-\beta})^2}{d^2}$$

Where n = sample size

r = n1/n2 is the ratio of sample size required for two groups; r=1 when cases are equal to controls

$Z_{\alpha/2}$ is the normal deviate at a level of significance which is equivalent to 1.96 for 5% level of significance

$Z_{1-\beta}$ is the normal deviate at 1-β% power with β% of type II error which is equivalent to 0.84 at 80% power.

σ = pooled standard deviation between two groups.

d = difference of means between two groups.

The values of 'σ' and 'd' were obtained from previous studies of similar hypothesis [8,9]. Calculations were done for each parameter to derive sample size for dyslipidaemic subjects. Calculated sample size was 70 subjects in each group.

Written informed consents were taken from all the subjects who participated in present study after explaining the purpose of the study. Detail history was obtained from all the patients and height and weight also measured to calculate BMI as per the following formula [20].

$$BMI = \frac{\text{Weight in Kg}}{\text{Height in m}^2}$$

Biochemical Analysis

After an overnight fasting approximately 4 mL of venous blood sample was collected from an antecubital vein with aseptic condition without adding anticoagulant and allowed to clot. Immediately the serum was extracted and stored at -20°C until analysation done. The sample collection and test procedure were followed and standardised as per the guidelines of the National Committee for Clinical Laboratory Standards (NCCLS) [21].

The [Table/Fig-1] [3,18,22,23] shows all the parameters assayed, the methods used and reference ranges as per availability of commercial diagnostic kits [3,18,22,23].

Tests Performed	Methodology	Manufacturer	Reference Range
Serum Adiponectin [3]	Sandwich ELISA	CUSA Biotech USA	5.0-30.0 µg/mL
Fasting Plasma Glucose [22]	Colorimetric GOD POD	Erba Mannheim	70.0-100.0 mg/dL
Serum Total Cholesterol [18]	Colorimetric CHOD PAP Method	Erba Mannheim	<200.0 mg/dL
Serum Triglycerides [18]	Colorimetric GPO POD	Erba Mannheim	<150.0 mg/dL
Serum HDL-C [18]	Colorimetric Direct End point	Erba Mannheim	40.0-60.0 mg/dL [26,34]
Serum LDL-C [23]	Mathematically, Friedwald and Frederickson formula		<130.0 mg/dL

[Table/Fig-1]: Tests performed for various parameter analysis, their methodologies and reference range [3,18,22,23].

STATISTICAL ANALYSIS

All the data was processed and statistical analysis was performed by licensed version of SPSS 16.0 software (SPSS Inc., Chicago, IL, USA) for windows 10. Data were expressed in "mean±SD" and student 't' test was applied to see statistical significance in adiponectin levels between dyslipidaemic subjects and healthy controls. A p-value <0.05 was considered statistically significant. Coefficient of correlation ('r') was obtained between different biochemical parameters by help of Pearson product moment correlation.

RESULTS

The demographic distribution and BMI in present study population for the dyslipidaemic cases and control group is shown in [Table/Fig-2]. The mean age group of dyslipidaemic cases was 43.61±4.85 years and mean age of controls was 43.53±5.53 years. The mean BMI of dyslipidaemic cases were significantly higher than the control group BMI for dyslipidaemic cases 25.72±2.43; for control 23.42±1.56 with p-value <0.0001.

Parameters	Controls (n=70) (Mean±SD)	Dyslipidaemic cases (n=70) (Mean±SD)	p-value as per unpaired Student's 't' test
Age (Years) (only male population)	43.53±5.53	43.61±4.85	0.92
BMI (Kg/m^2)	23.42±1.56	25.72±2.43	<0.0001

[Table/Fig-2]: Demographic characteristic and BMI of study population.

Comparison of various biochemical parameters (plasma glucose, serum adiponectin and lipid profile) is shown in [Table/Fig-3]. Plasma glucose level in both controls and dyslipidaemic cases was within normal reference range, but in dyslipidaemic cases the mean plasma glucose level (89.22±8.91 mg/dL) was raised compared to control group (84.15±11.58 mg/dL) and p-value was statistically significant (p-value=0.004). Serum mean adiponectin level was (5.11±2.04 µg/mL) in dyslipidaemic cases which was less than that of (6.79±1.37 µg/

Parameters	Controls (Mean±SD)	Dyslipidaemic cases (Mean±SD)	p-value as per unpaired Student's 't' test
Fasting plasma glucose (mg/dL)	84.15±11.58	89.22±8.91	0.0042
Adiponectin (µg/mL)	6.79±1.37	5.11±2.04	<0.0001
Total Cholesterol (mg/dL)	166.96±33.85	246.60±37.33	<0.0001
Triglyceride (mg/dL)	129.67±42.28	195±54.76	<0.0001
HDL-C (mg/dL)	45.75±11.01	31.39±8.28	<0.0001
LDL-C (mg/dL)	95.26±32.99	168.71±32.81	<0.0001
VLDL-C (mg/dL)	25.93±8.45	38.89±10.91	<0.0001

[Table/Fig-3]: Comparison of biochemical parameters between two groups.

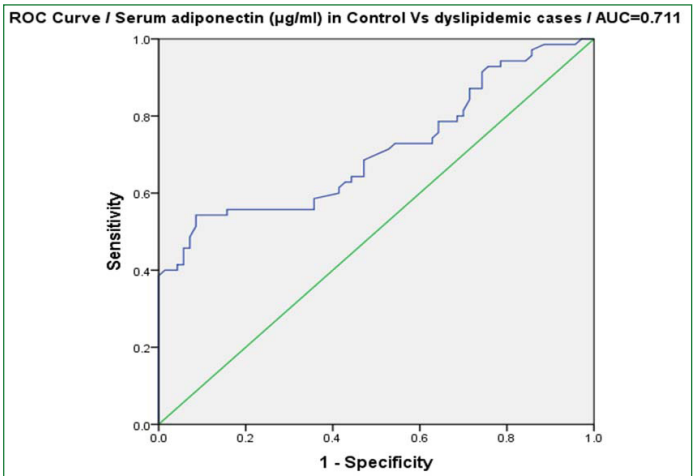
mL) as in control group. When compared with unpaired student 't' test p-value was <0.0001 which is highly statistically significant. The levels of total cholesterol (246.60±37.33 mg/dL), triglyceride (195±54.76 mg/dL), LDL-C (168.71±32.81 mg/dL), were significantly higher in dyslipidaemic case in comparison with the control group (p-value <0.0001). The level of HDL-cholesterol (31.39±8.28) significantly reduced in dyslipidaemic cases with p value statistically significant (p-value <0.0001).

Among the parameters, serum adiponectin levels had strong negative correlation with BMI (r=-0.83), serum cholesterol (r=-0.89), serum triglyceride (r=-0.76), LDL-C (r=-0.74) but positive correlation with HDL-C (r=0.70) [Table/Fig-4].

Parameters versus serum adiponectin value	'r' value	p-value as per Student's unpaired 't' test
BMI	-0.83	<0.001
Total Cholesterol	-0.89	<0.001
Triglyceride	-0.76	<0.001
HDL-C	0.70	<0.001
LDL-C	-0.74	<0.001
VLDL-C	-0.76	<0.001

[Table/Fig-4]: Correlation of different biochemical parameters with adiponectin in dyslipidaemic cases.

When plotted in ROC curve in dyslipidaemic cases vs. control groups best cut off value obtained for serum adiponectin was 6.32 µg/mL with 63% sensitivity and 58% specificity with AUC 0.711 [Table/Fig-5].



[Table/Fig-5]: ROC curve analysis of serum adiponectin in dyslipidaemic cases versus control.

DISCUSSION

In this present study it was found that serum adiponectin level was decreased in dyslipidaemic cases (5.11±2.04 µg/mL) as compare to healthy subjects (6.79±1.37 µg/mL) which was statistically highly significant with p-value <0.0001. Significant negative correlation (r= -0.83) between serum adiponectin and BMI in dyslipidaemic cases were observed. Such decrease level of adiponectin and inverse relation with BMI also reported by several other studies conducted by Ryo M et al, Matsubara M and Hu E et al., [2,9,24].

In this study serum adiponectin shows significant negative correlation with total cholesterol (r=-0.89), triglyceride (r=-0.76) and LDL cholesterol (r=-0.74). But HDL cholesterol shows significant positive correlation (r=0.70) with serum adiponectin levels. Such findings were in accordance with Matsubara M et al. who observed that dyslipidaemic is associated with low adiponectin levels in non-diabetic women and found significant negative correlation between adiponectin and serum triglyceride and positive correlation with HDL-C [9]. This finding was also in agreement with another study conducted by Vander Vleuten GM et al. who observed that patients with familial hyperlipidemia a reduction of 25% serum adiponectin levels led to reduction of 7.3% HDL-C level and this reduction of serum adiponectin level is associated with increase of atherogenic lipids such as high LDL-C, TG and low HDL-C [11]. Findings from several other studies also showed that serum adiponectin level inversely correlated with TG level which is in accordance to this present study [12,13,25]. Another study conducted by Hotta K et al. in type 2 diabetic patients shows significant negative correlation between adiponectin and TG levels, and positive correlation between adiponectin and HDL-C levels [26].

The present study results extend this finding by revealing that plasma adiponectin concentrations are not only inversely correlated to BMI, total cholesterol, TG, LDL-C, but also positively correlated to serum HDL-C in non-diabetic north Indian male subjects. Another similar study conducted by Daniela G et al., clearly suggest that subjects with relatively lower plasma levels of adiponectin show decreased HDL cholesterol which may be involved in the pathogenesis of coronary endothelial dysfunction in patients with dilated cardiomyopathy [27]. These observations strongly suggest that the anti-atherosclerotic actions of adiponectin might be due to its effects on modulating lipids metabolism.

The exact physiological role of adiponectin in lipid modulation is not properly understood but experimental studies suggest its role as a potential antiatherogenic and anti-inflammatory marker [4]. At the early onset of atherosclerosis endothelial cells get activated by numerous inflammatory stimuli like Tumour Necrosis Factor (TNF), which leads to the synthesis of adhesion molecules and increases the adherence of monocytes. Such type of adhesion monocyte is very crucial for the initial development of vascular disease [4]. Adiponectin has been believed to down regulate both the production and action of TNF [27]. The expression of scavenger receptor A-1 also gets inhibited by adiponectin molecule. As a result, decrease uptake of oxidized LDL cholesterol occur and foam cell formation also gets inhibited. This step is vital in fighting the onset of atherosclerosis. Studies suggested that patients with decrease serum adiponectin level had a significant two-fold increase in coronary artery disease prevalence, which is found to be independent of well-known Coronary Artery Disease (CAD) risk factors [4,27].

Adiponectin is associated with key rate limiting enzymes in lipid metabolism which directly influence the concentrations of circulating lipids, particularly TG and HDL cholesterol [4,5]. Adiponectin is known to lower the synthesis of free fatty acids and to stimulate β-oxidation [11,13]. These metabolic effects resulted partly from the ability of adiponectin to increase carnitine palmitoyl transferase I activity and increase hepatic fatty acid oxidation. The reduce synthesis of fatty acid is due to the decrease activities of two main regulating enzymes involved in fatty acid synthesis, which includes acetyl-CoA carboxylase and fatty acid synthase [14]. Furthermore, High Molecular Weight (HMW) adiponectin seems to decrease the release of apolipoprotein apo B and apo E from the liver which reduces the release of lipoproteins rich in TG e.g., Very Low Density Lipoprotein (VLDL) and increasing HDL-C levels [13,14].

In this present study VLDL-C levels were inversely correlated (p-value= -0.76) with adiponectin levels independently of age, body mass index (BMI). The observed association of serum adiponectin with VLDL strongly suggests that the regulation of serum VLDL-C by adiponectin may involve VLDL catabolism. A probable explanation for the adiponectin-induced elevation in TG catabolism is the control of LPL activity by adiponectin [28]. It has been observed that the TG-reduction effect of adiponectin was not due to a reduction in hepatic VLDL-TG secretion, but rather was due to VLDL-TG catabolism through arise in postheparin plasma Lipoprotein Lipase (LPL) activity which is independently of insulin resistance and inflammation [28]. Another possible mechanism which probably explain TG reduction by adiponectin would be the adiponectin mediated reduction in serum APOCIII as because APOCIII is well established inhibitor of LPL and negative correlation observed between APOCIII with serum adiponectin by various studies [11,12,14,28].

Studies suggest that insulin resistance boost the overall activity and also the expression of hormone-sensitive lipase in adipose tissue

[5,7,28,29]. This help in catalysing the breakdown of TG, releasing Free Fatty Acids (FFA) [5,7,29]. As a result, increased FFA enter the liver and rise the production of VLDL. Therefore, an improvement of insulin resistance by adiponectin may reduce Hormone Sensitive Lipase (HSL) activity and result in a decrease of VLDL overproduction. The effect of serum adiponectin in HDL-C elevation is through via an increase in the hepatic production of major apo protein of HDL-C, i.e apo-AI and via an elevation in the production of ATP-binding cassette transporter A1 (ABCA1), which initiates HDL assembly [11].

Regarding the association of LDL-C with serum adiponectin, several studies observed no direct significant association unlike this present study [7,9]. Studies conducted by Kazumi T et al., Okada T et al. showed that adiponectin concentrations were inversely associated with LDL cholesterol level which is in accordance to this present study [14,30]. Adiponectin induced improvement of TG, HDL and may decrease the atherogenic lipoprotein small dense-LDL [30]. Remnant lipoproteins, produced from VLDL and chylomicrons, have been thought to be atherogenic [4,30]. Therefore, adiponectin decreases the TG, which causes the reduction in remnant lipoproteins, thus adiponectin may attribute to the antiatherogenic effects.

Limitation(s)

In the present study, cases and controls is pooled from single hospital which may lead to selection bias and does not allow the calculation of incidence rate so further multicentric prospective study could provide more insights.

CONCLUSION(S)

The present study shows significant decrease in serum adiponectin levels in dyslipidaemic group and it is negatively correlated with serum total cholesterol, triglycerides and LDL-Cholesterol while positively correlated with serum HDL-Cholesterol by Pearsons Moment Correlation. Above said findings supports the hypothesis that lower levels of serum adiponectin increased the risk factor for developing CVD due to its hyperlipidemic effect. This study could be further validated if a prospective multicentric study is undertaken.

Acknowledgement

The authors are very much obliged to all the staff of Department of Biochemistry of RMRI, Bareilly, India for their technical support.

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PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of Biochemistry, RDASMC, Ayodhya, Uttar Pradesh, India.
2. Associate Professor, Department of Biochemistry, RDASMC, Ayodhya, Uttar Pradesh, India.
3. Associate Professor, Department of Biochemistry, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India.
4. Tutor, Department of Biochemistry, RDASMC, Ayodhya, Uttar Pradesh, India.
5. Professor and Head, Department of Biochemistry, RDASMC, Ayodhya, Uttar Pradesh, India.
6. Dietitian, Department of Dietetics, RDASMC, Ayodhya, Uttar Pradesh, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Ranadip Mukherjee,
Assistant Professor, Department of Biochemistry, RDASMC, Ganja, Ayodhya, Uttar Pradesh, India.
E-mail: ranadip_mukherjee83@yahoo.co.in

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Mar 21, 2022
- Manual Googling: Apr 27, 2022
- iThenticate Software: May 06, 2022 (25%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Mar 15, 2022**
Date of Peer Review: **Mar 31, 2022**
Date of Acceptance: **May 03, 2022**
Date of Publishing: **Jul 01, 2022**