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ADIPONECTIN AS A BIOMARKER FOR CARDIOMETABOLIC SYNDROME IN PREHYPERTENSIVE SUBJECTS

Health Science

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ABSTRACT

Prehypertension (PHTN) is a global major health risk that focuses individuals to double the risk of cardiovascular disease (CVD) independent of progression to hypertension. Its prevalence rate varies considerably from country to country ranging between 21.9% and 52%. Several laboratory studies shows that adiponectin suppresses certain pathophysiological processes related to endothelial dysfunction, inflammation and atherosclerosis but its association with prehypertension and risk for cardiomeabolic syndrome remain sparse. Hence the present study was undertaken to evaluate role of adiponectin as a biomarker for the early prediction of cardiometabolic syndrome in prehypertensive subjects. The present study included 75 subjects with prehypertension and 48 healthy age and sex matched controlssubjects. The mean adiponectin value of study subjects was lower than healthy control subjects. This indicates that when adiponectin level decreases the chances of having cardiometabolic syndrome increases. Thus adiponectin has an inverse or strong negative association with cardiometabolic syndrome. Therefore the study suggests that modification of lifestyle and proper medications will help to improve the level of adiponectin, thus lower the risk of CMS in prehypertensive subjects.

KEYWORDS

Prehypertension, Cardiometabolic Syndrome, Adiponectin, Atherosclerosis

INTRODUCTION

The concept of prehypertension (PHTN) was introduced in 1939 by Robinson and Brucer who were first to draw attention to the range of blood pressure (BP) between 120-139 mmHg (systolic) and 80-89 mmHg (diastolic) as being of value in determining clinically overt hypertension. Prehypertension (PHTN) is a global major health risk that subjects individuals to double the risk of cardiovascular disease (CVD) independent of progression to overt hypertension. Its prevalence rate varies considerably from country to country ranging between 21.9% and 52%. Many hypotheses are proposed to explain the underlying pathophysiology of PHTN. The most notable of these implicate the renin-angiotensin system (RAS) and vascular endothelium. However, other processes that involve reactive oxygen species, the inflammatory cytokines, prostaglandins and C-reactive protein as well as the autonomic and central nervous systems are also suggested (Albarwani et al., 2014).

Metabolic Syndrome (MS) was diagnosed according to the guidelines of the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III – ATP III), in the presence of three or more of the following criteria: abdominal obesity according to waist circumference (>102cm for men and >88cm for women), hypertriglyceridemia (≥150mg/dL), low HDL-C (<40mg/dL for men and <50mg/dL for women), pre-hypertension or hypertension (≥130mmHg and ≥85mmHg) and fasting glucose (≥110mg/dL and/or diagnosed diabetes mellitus) (Silva et al, 2015).

Cardiometabolic syndrome (CMS) is a constellation of metabolic dysfunction characterized by insulin resistance and impaired glucose tolerance, atherogenic dyslipidemia, hypertension and intra-abdominal adiposity (IAA). Cardiometabolic syndrome is now recognized as a disease entity by the American Society of Endocrinology, National Cholesterol Education Program (NCEP) and World Health Organization (Castro et al., 2003). Around 25% of the world's adults are suffering from cardiometabolic syndrome (International Diabetes Federation, 2011). Adipose tissues are considered to involve in the release of several inflammatory and immune mediators. The onset of MS and CVD is thought to be partly mediated by aberrant expression of adipokines (Gremese E et al., 2011). Levels of circulating adiponectin are inversely correlated with BMI, waist-to-hip ratio and percent body fat (Baldwin W et al., 2011).

Laboratory studies show that adiponectin suppresses several pathophysiological processes related to obesity, including insulin resistance, endothelial dysfunction, inflammation and atherosclerosis (PengliBao et al., 2015). Despite a number of cross-sectional and case-control studies showing an inverse association between plasma adiponectin and BP levels or hypertension status, prospective studies of adiponectin in association with prehypertension and risk for cardiomeabolic syndrome remain sparse (Xi B et al., 2013). Hence the present study was undertaken to evaluate role of adiponectin as a biomarker for the early prediction of cardiometabolic syndrome in prehypertensive subjects.

MATERIALS AND METHOS

75 subjects with prehypertension and 48 healthy age and sex matched subjects were selected for this study. The samples were referred from various centers of Kerala to Genetika, Centre for Advanced Genetic Studies, Thiruvananthapuram, Kerala. Detailed demographic, clinical and lifestyle characteristics were recorded using proforma. Five ml of blood was transferred into a plain tube. Blood was allowed to clot and the serum was separated immediately. Biochemical parameters were estimated using semi-automated clinical chemistry analyzer. The level of serum adiponectin was determined using ELISA method.

OBSERVATION AND RESULTS

Based onadiponectin analysis, 75 study subjects showed a mean adiponectin value of 8.08µg/mL and control subjects showed10.73µg/mL. The mean adiponectin value of study subjects was lower than healthy control subjects. This indicates that when adiponectin level decreases the chances of having cardiometabolic syndrome increases. Thus adiponectin has an inverse or strong negative association with cardiometabolic syndrome.

Distribution of Adiponectin value according to demographic characteristics in prehypertensive subjects

Table 1:

Category	Variable	Number	Adiponectin (µg/mL)
Age	21-30	3	5.06
	31-40	12	7.6
	41-50	37	7.5
	51-60	23	6.8
Sex	Male	35	6.6
	Female	39	7.7

Birth order	≤3	47	7.08
	>3	28	7.5
Residence	Rural	26	8.1
	Urban	41	7.1
	Coastal	8	4.9
Occupational type	Non sedentary	65	7.3
	Sedentary	10	6.7
Socio-economic status	Low	14	7.4
	Average	30	5.6
	High	31	8.7

Distribution of mean adiponectin value according to demographic characteristics of the study subjects were given in the table 1. Among the 75 study subjects, 3 subjects within the age range of 21-30 years showed lowest mean adiponectin value of 5.06µg/mL. The mean adiponectin value was comparatively lower for male subjects (6.6µg/mL) than female subjects (7.7µg/mL). Subjects with birth order ≤3 had lowest adiponectin value (7.08µg/mL). Subjects those residing in the coastal area showed the lowest mean adiponectin value of 4.9µg/mL. Sedentary subjects were showed lowest mean value for adiponectin (6.7g/mL) than non- sedentary (7.3 µg/mL) subjects. Subjects with average socioeconomic status showed the lowest mean adiponectin value (5.6µg/mL).

Distribution of Adiponectin value according to lifestyle characteristics in prehypertensive subjects

Table 2:

Category	Variable	Number	Adiponectin (µg/mL)
Regular exercise	Yes	20	7.5
	No	55	5.8
Physical activities	Poor	11	6.1
	Good	64	7.4
Water intake per day	Poor	20	8.61
	Good	55	6.7
BMI	≤30	33	14.3
	>30	42	1.71
Systolic pressure	≤130	31	5.50
	>130	44	8.48
Diastolic pressure	≤90	28	5.14
	>90	47	8.24

Distribution of mean adiponectin value according to lifestyle characteristics of the study subjects are given in table 2. The study subjects without the habit of regular exercise and those with poor physical activity showed lowest adiponectin value. Majority of the subjects were obese (BMI >30) and they showed lowest adiponectin value when compared to non-obese subjects. Subjects with good water intake showed low adiponectin value. While considering the systolic and diastolic pressure the subjects with normal blood pressure showed the lowest adiponectin value.

Distribution of Adiponectin value according to biochemical parameters in prehypertensive subjects

Table 3:

Category	Variables	Number	Adiponectin (µg/mL)
FBS (mg/dL)	≤ 110	16	6.3
	>110	59	7.48
TC (mg/dL)	≤200	39	5.8
	>200	36	9.06
TG (mg/dL)	≤150	19	5.9
	>150	56	7.6
HDL (mg/dL)	≤40	28	8.3
	>40	47	6.61
LDL (mg/dL)	≤100	9	6.3
	>100	66	7.3
hs CRP (mg/L)	≤3.0	3	12.65
	>3.0	72	7.18
Creatinine (mg/dL)	≤1.2	3	12.35
	>1.2	72	7.04
Urea (mg/dL)	≤20	4	4.7
	>20	71	7.3
Uric acid (mg/L)	≤7.0	31	7.17
	>7.0	44	6.3

Distribution of mean adiponectin value according to biochemical parameters of the study subjects are given in table 3. The study subjects with normal range of total cholesterol, fasting blood sugar, triglycerides, low density lipoprotein cholesterol, high density lipoprotein cholesterol and urea showed lowest adiponectin value. While considering the high sensitive C-reactive proteins, creatinine and uric acid the subjects with abnormal levels showed the lowest adiponectin value.

DISCUSSION

In the study of Moller et al., (2005) it is mentioned high blood pressure is a frequent component of MS and generally associated with central obesity and insulin resistance. In the present study, the subjects with prehypertension showed 132-93 mmHg range. In a study done by Salazar et al., (2011) reported that insulin resistance is a determining factor and early feature in the development of MS. It is frequently linked to a range of comorbidities such as obesity, type 2 diabetes and atherosclerotic diseases.

According to the study by Arunkumar and Sushil (2017) an acute bout of aerobic exercise results in a significant increase in adiponectin levels in individuals with abdominal obesity. Adiponectin levels are inversely proportional to both total and abdominal fat mass. Evidence suggests that an acute bout of vigorous aerobic exercise may result in a significant increase in plasma adiponectin levels in trained athletes. In contrast, immediately following the cessation of exercise, adiponectin levels are reported to be unchanged or even reduced (Jurimae et al, 2005) in trained individuals. In the present study the adiponectin levels were found to be higher among the subjects who had a habit of doing regular exercise.

Tobias et al, 2004 suggest that adiponectin has been shown to result in activation of the adenosine monophosphate–activatedprotein kinase in skeletal muscle and liver, leading to phosphorylation of acetyl coenzyme A carboxylase, increased fatty acid oxidation and glucose uptake, reduced fatty acid synthesis and reduction of molecules involved in gluconeogenesis. Downstream effects may include reduced triglyceride content in liver and skeletal muscle and suppression of hepatic glucose production. This may also explain the finding that adiponectin is inversely associated with triglyceride levels and positively associated with HDL cholesterol levels. The present study reveals that lipid profile and fasting blood sugar had a positive correlation with adiponectin.

Adiponectin may lower CRP and other inflammatory cardiovascular risk factors (Meda et al, 2002). Thus, injection of an adiponectin-producing adenovirus was shown to reverse the significantly increased adipose tissue, tumor necrosis factor (TNF-) messenger RNA andplasma TNF-levels in adiponectin knockout mice. In line with these animal experiments, human studies found inverse associations between adiponectin and the inflammatory markers TNF, interleukin (Halleux et al., 2001) and CRP (Kern et al., 2003). In the present study also the inflammatory markers showed an inverse relationship with adiponectin.

CONCLUSION

Although risks that cluster in metabolic syndrome are common, it is likely that the cluster occurs coincidentally. Adiponectin is a unique and essential adipocytokine that is produced very abundantly in adipocytes and stably present in the plasma at very high concentration. In healthy subjects, adiponectin carries out its roles for preventing development of vascular changes and the impairment of glucose and lipid metabolism, which may be induced by a variety of attacking factors. The present study highlights the inverse and independent association of adiponectin with the presence of cardiometabolic syndrome in prehypertensive subjects. The major finding of the current study was that adiponectin exhibited a significantly higher probability of having cardiometabolic syndrome and was dependent of BMI and other factors such as inflammatory markers and lifestyle behaviors. Therefore the study suggests that modification of lifestyle and proper medications will help to improve the level of adiponectin, thus lower the risk of CMS in prehypertensive subjects.

REFERENCES

1. Achari, A. and Jain, S, Adiponectin, a therapeutic target for obesity, diabetes and endothelial dysfunction, International journal of molecular sciences, 2017 18(6), p.1321.
2. Castro JP, El-Atat FA, MacFarlane SI, Aneja A, Sowers JR. Cardiometabolic syndrome: Pathophysiology and treatment. CurrHypertens Rep 2003;5:393-401.
3. Halleux CM, Takahashi M, Delporte ML, et al. Secretion of adiponectin and regulation of apM1 gene expression in human visceral adipose tissue. BiochemBiophys Res Commun. 2001;288:1102-1107.

4. International Diabetes Federation. Available from: http://www.idf.org. [Last accessed on 2011 Jan 25].
5. Jürimäe, J.; Purge, P.; Jürimäe, T., Adiponectin is altered after maximal exercise in highly trained male rowers.Eur. J. Appl. Physiol., 2005 93, 502–505.
6. Kern PA, Di Gregorio GB, Lu T, Rassouli N, Ranganathan G. Adiponectin expression from human adipose tissue: relation to obesity, insulin resistance, and tumor necrosis factor-alpha expression. Diabetes.2003; 52:1779-1785.
7. Maeda N, Shimomura I, Kishida K, et al. Dietinduced insulin resistance in mice lacking adiponectin/ ACRP30. Nat Med. 2002;8:731-737.
8. Moller DE, Kaufman KD., Metabolic syndrome: a clinical and molecular perspective. Annu Rev Med 2005;: 45–62.
9. Pischon, T., Girman, C.J., Hotamisligil, G.S., Rifai, N., Hu, F.B. and Rimm, E.B., Plasma adiponectin levels and risk of myocardial infarction in men. 2004 Jama, 291(14), pp.1730-1737.
10. Robinson SC, Brucer M. Range of normal blood pressure: a statistical and clinical study of 11,383 persons. Arch Int Med 1939;64: 409–444.
11. Salazar MR, Carbajal HA, Espeche WG, Dulbecco CA, Aizpurúa M, Marillet AG, Echeverría RF, Reaven GM. Relationships among insulin resistance, obesity, diagnosis of the metabolic syndrome and cardio-metabolicrisk. DiabVascDisRes. 2011;8:109–116.