

# To Correlate the Serum Levels of Adiponectin with Systolic and Diastolic Blood Pressure in Hypertensive's and Healthy Individuals

1. Humera Nawab Ghori 2. Uzma Tasneem 3. Gul Afshan

1. Asstt. Prof. of Physiology 2. Asstt. Prof. of Physiology 3. Associate Prof. of Physiology, Karachi Medical and Dental College (Abbassi Shaheed Hospital), Karachi.

## ABSTRACT

**Background:** To correlate the serum levels of adiponectin with systolic and diastolic blood pressure in hypertensive's and healthy individuals.

**Study Design:** Cross-sectional, case control study.

**Place and Duration of study:** This study was conducted at the Department of Physiology BMSI JPMC in collaboration with Abassi Shaheed hospital karachi from Dec 2008 to June 2009.

**Materials and Methods:** This study included total 100 subjects, 50 subjects with known case of hypertension and 50 normal healthy age and gender matched controls

**Results:** Adiponectin serum concentration was found lower in coronary artery disease patients when compared with control subjects. (7.10 ) verses (11.98).

**Conclusion:** This suggests that higher levels of serum adiponectin is protective against the development of hypertension.

**Key Words:** Coronary heart disease, Adiponectin, Case Control study.

## INTRODUCTION

Hypertension is a major risk factor for cardiovascular disease, it is the leading cause of morbidity and mortality worldwide. In developed countries, hypertension ranks as the top contributing factor for mortality and third in causing disability-adjusted life years.<sup>1</sup> Hypertension is a polygenic and complex disease with rising prevalence. More than 25% of the adult population is affected by hypertension, and two thirds of those individuals reside in developing countries.<sup>2</sup> With the present trends, the prevalence of hypertension is predicted to increase to 30%, or  $\approx$ 1.5 billion people, on the globe in the next 20 years.<sup>3</sup>

Hypertension causes injury to the endothelium leading to endothelial dysfunction which may develop atherosclerotic changes<sup>4</sup>. In hypertension the concentration of angiotensin II, the principal product of renin angiotensin system are often elevated, angiotensin is the potent vasoconstrictor. In addition to cause hypertension it contributes to atherosclerosis by stimulating the growth of smooth muscle<sup>5</sup>.

Adiponectin is the most abundant known secreted factor produced by adipocytes, was originally identified by four independent groups in mid 1990's, in both mice and human<sup>6</sup>. The adipose tissue has traditionally been regarded as a silent organ that passively stores excess energy. However the recent evidence suggests that adipose tissue especially visceral fat is to be considered as an endocrine organ, directly involved in the pathophysiology of metabolic syndrome and cardiovascular diseases.<sup>7</sup> As the biologically active molecule, adiponectin appears to protect the vasculature

at each stage of atherosclerosis. First stage of atherosclerosis involves endothelial dysfunction in response to the traditional cardiovascular risk factor, such as hypertension<sup>13</sup>.

## MATERIALS AND METHODS

A total of 100 subjects were recruited in the study, and divided into 2 groups:-

A group consisting of 50 normal healthy control subjects with no history of hypertension. The other group included 50 subjects which were known cases of hypertension. Normal and diseased subjects were from both sexes of age ranging from 40 years to 80 years.

A written consent was taken from all the participants of the study six ml of the venous blood was drawn from the subjects under all aseptic procedures. The blood sample was transferred to the gel tube. After 30 min to 60 min the blood was centrifuged for 10 minutes at the speed of 3000 rounds per minutes (rpm). Serum was separated to dry clean aliquot tube and stored at -20 degree centigrade. Before analyzing, the samples were thawed and allowed to attain the room temperature. Serum adiponectin was measured by enzyme linked immunosorbent assay, using the kit provided by Biosource France.

## RESULTS

Table 1 shows the comparison of mean ages among 2 groups i.e. normal healthy control group and the group having diagnosed cases of hypertension. Mean ages of 2 groups were comparable on average, as the samples were collected from the matched cases. Table 1 also

shows the comparison of height among the 2 groups i.e. between cases and control subjects; non significant changes were recorded in the height among the 2 groups. On comparison of weight among the 2 groups i.e. between cases and the control subjects, weight in group B was significantly increased as compared to control subjects ( $73.5 \pm 0.5$ ) and ( $66.0 \pm 0.10$ ) respectively ( $p < 0.001$ ). Independent t-test was applied.

Table 2 shows comparison of systolic and diastolic blood pressure among hypertensive subject and control group. The systolic blood pressure shows statistically significant increase in hypertensive patients ( $170.1 \pm 19.1$ ) than the systolic blood pressure in control group ( $146.7 \pm 20.9$ ). Similarly diastolic blood pressure shows statistically increased significance ( $p < 0.001$ ) in hypertensive patients ( $95.7 \pm 12.6$ ) than in control subjects ( $82.8 \pm 13.2$ ).

Table 3 shows comparison between serum adiponectin concentrations in hypertension subject with that of control. The adiponectin concentration shows significant ( $p < 0.001$ ) decrease in hypertensive patients ( $7.10 \pm 1.40$ ) than in controls ( $11.98 \pm 0.59$ ).

**Table No.1: Comparisons of age, height and weight in control & hypertensive groups**

Variables	Group A	Group-B
Age(yrs)	Control	hypertensive
	Mean $\pm$ SEM	Mean $\pm$ SEM
Height (meter)	$55.18 \pm 1.12$	$54.17 \pm 2.24$
	$1.60 \pm 0.02$	$1.56 \pm 0.02$
Weight (kg)	$66.0 \pm 0.10$	$73.5 \pm 0.19^{**}$

\*\* $P < 0.001$  highly significant when compared to controls

**Table No.2: Comparison of systolic and diastolic blood pressure in hypertensive patients (group B) with normal healthy (Groups).**

Variables	Group A	Group B
SBP (mmHg)	Control	Hypertensive
	Mean $\pm$ SEM	Mean $\pm$ SEM
	$146.7 \pm 20.9$	$170.1 \pm 19.1^{**}$
DBP(mmHg)	$82.8 \pm 13.2$	$95.7 \pm 12.6^{**}$

\*\* $P < 0.001$  highly significant when compared to controls.

**Table No.3: Comparison of serum adiponectin concentration in normal control group a with hypertensive subjects Group B**

Variables	Group A	Group B
Serum Adiponectin ( $\mu\text{gm}/\text{ml}$ )	Control	Hypertensive's
	Mean $\pm$ SEM	Mean $\pm$ SEM
	$11.98 \pm 0.579$	$7.10 \pm 1.40^{**}$

\*\* $P < 0.001$  highly significant when compared to controls

Table 4 shows correlation coefficient ( $r$ ) among serum adiponectin with systolic and diastolic blood pressure, among the two groups. Highly significant negative

correlation was found between control group and hypertensive patients ( $r = 0.55$  and  $r = -0.76$ ). Similarly significant negative correlation was found between diastolic blood pressures among controls and hypertensive ( $r = 0.65$  and  $r = -0.86$ ).

**Table No.4: Correlation of serum adiponectin concentration with systolic and diastolic blood pressure in Group A & B**

Variables	Group A	Group B
	Serum adiponectin	Serum adiponectin
SBP mmHg	$r = -0.54$	$r = -0.76^{**}$
DBP mmHg	$r = -0.65$	$r = -0.86^{**}$

\*\*Correlation is significant at the 0.01 level.

## DISCUSSION

Hypertensive subjects are always at high risk of developing coronary heart disease, endothelial dysfunction caused by hypertension leads to the inflammatory process which may cause the initiation of atherosclerotic phenomenon<sup>8</sup>. As expected from our results the relationship between hypertension and adiponectin was inversely related. Our study showed that decrease in serum adiponectin levels in hypertensive subjects as compared to control group. Our observations suggested that subjects having low serum adiponectin levels are more prone to develop hypertension and this observation is in agreement with the studies of Adamezeck et al. (2005) and Kasumi et al. (2002) who did their study in Japanese hypertensive men. Our results showed strong negative correlation of systolic and diastolic blood pressure with serum adiponectin which are in agreement with the study done by Funuhashi et al. (2003) and Mallamaki et al. (2002). According to Ouchi et al. (2003) serum adiponectin levels were independently correlated with vasodilator response to reactive hyperemia, so it can be an independent parameter for endothelial dysfunction which is an important feature of hypertension and atherosclerosis<sup>9</sup>. Our study agreed with the above statement and suggests that hypo adiponectinemia may affect the pathogenesis of hypertension at very early stage.

Lower concentration of serum adiponectin has been associated with both hypertension and dyslipidemia.<sup>12</sup> Patients with essential hypertension appears to have significantly lower levels of plasma adiponectin when compared with normotensive patients<sup>13</sup>. In another case-control study, after adjusting for confounding factors such as obesity, insulin resistance, and diabetes, significantly lower concentrations of circulating adiponectin were present in patients with hypertension compared with those without. (Iwashima et al., 2004). Adiponectin may also be involved in the progression of hypertension. On a high-salt diet, Ohashi et al showed that adiponectin-deficient animals display significantly

higher systolic blood pressure compared with wild-type control animals independent of insulin resistance.

## CONCLUSION

This suggests that higher levels of serum adiponectin is protective against the development of hypertension.

## REFERENCES

1. Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ. Selected major risk factors and global and regional burden of disease. *Lancet* 2002; 360: 1347–1360.
2. Kearney PM, Whelton M, Reynolds K, Muntner P, Whelton PK, He J. Global burden of hypertension: analysis of worldwide data. *Lancet* 2005;365: 217–223.[ Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. *J Clin Invest* 2006; 116: 1784–1792.
3. Wolf-Maier K, Cooper RS, Banegas JR, Giampaoli S, et al. Hypertension prevalence and blood pressure levels in 6 European countries, Canada, and the United States. *JAMA* 2003;289:2363–2369.
4. Ohashi K, Kihara S, Ouchi N, Kumada M, Fujita K, Hiuge A, et al. Adiponectin replenishment ameliorates obesity-related hypertension. *Hypertension* 2006; 47: 1108–1116.
5. Iwashima Y, Katsuya T, Ishikawa K, Ouchi N, Ohishi M, Sugimoto K, et al. Hypoadiponectinemia is an independent risk factor for hypertension. *Hypertension* 2004; 43: 1318–1323.]
6. Funahashi T, Matsuzawa Y, Matsubara K. DNA cloning and expression of a novel adipose specific collagen-like factor, apM1 (AdiPose Most abundant Gene transcript 1). *Biochem Biophys Res Commun* 1996; 221: 286–289
7. Adamczak M, Wiecek A, Funahashi T, Chudek J, Kokot F, Matsuzawa Y. Decrease plasma adiponectin concentration in patients with essential hypertension, *Am J Hypertension* 2003;16:72-75.
8. Iwashima Y, Horio T, Suzuki Y. Hypoadiponectinemia is an independent risk factor for Hypertension 2004;42: 1318-1323.
9. Kumada M, Kihara S, Ouchi N. Adiponectin specifically increased tissue inhibitor of metalloproteinase – 1 through interleukin – 10 expression in human macrophage circulation 2004; 109: 2046-2049.
10. Nakamura Y, Shimada K, Fukuda D, Shimada Y, hirose M, et al. Implication of plasma concentration of adiponectin in patients with coronary artery diseases. *Heart* 2004;90:528-533.
11. Baretta R, Amato S. Adiponectin relationship with lipid metabolism is independent of body fat mass: *J Clin Endocrinology Metabolism* 2004;89:2665-2671.
12. Lacy F, O'Connor DT, et al. Plasma hydrogen per oxide produce in hypertensive and normotensive subjects at genetic risk of hypertension 1998;16:291-303.
13. Kobayashi H, Ouchi N, Kihara S, Walsh K, Kumara M. Selective suppression of endothelial cell receptors by the high molecular weight form of adiponectin *Circ Res*. 94: e 27 – e 31, 2004. Matsuzawa YM. Pathophysiology and molecular Mechanism of visceral fat syndrome. *Diabetes Metab Rev* 2005; 13:13-13.

### Address for Corresponding Author:

**Dr. Humaira Nawab**

Karachi Medical and Dental College  
(Abbassi Shaheed Hospital), Karachi  
Cell No.0313-2461330