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STUDY OF SERUM RESISTIN LEVEL IN ESSENTIAL HYPERTENSION

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Abstract:

Hypertension is a persistent elevation of B.P. above the normal level. Approximately 1 billion people have hypertension globally. Resistin is a Cysteine – rich protein secreted by adipose tissue. In this study, 60 patients with stage I & stage II hypertensives and 60 normotensives are included. Venous blood sampling was done for estimation of serum resistin. Serum resistin was found to be positively correleated with BP ($P < 0.001$) and negatively correleated with GFR ($P < 0.0001$). This indicates that augumented level of serum resistin may be a risk factor for kidney dysfunction and it can be considered as early predictor of kidney dysfunction in hypertensive subjects.

Keywords: Hypertension, Resistin, kidney dysfunction, B.P.

Introduction:

Hypertension is a persistent elevation of B.P. above the normal level. About 95% of all cases of hypertension have no single identifiable causes and are termed primary Hypertension (or) essential Hypertension. (Lancet 2005; 365: 217-223).

Approximately 1 billion people (around 26% of all adults) have hypertension globally. Hypertension is often called 'the silent Killer' as it frequently has no obvious symptoms and consequently, often goes undetected [2].

The clinical significance of hypertension stems from the organ damage caused by elevated B.P including. Left ventricular hypertrophy (LVH) which may progress to congestive heart failure (CHF) arteriosclerosis, and increases the incidence of arteriosclerosis [3]; Retinopathy, which may cause a progressive worsening of vision; and kidney damage leading to fibrosis, impaired renal function and eventual renal failure.

As a result of these effects, increases in BP are clearly associated with increases in the risk of death from organ damage for every 20 mmHg systolic or 10 mmHg diastolic increases in BP, mortality from ischemic heart disease, heart failure and stroke doubles. Importantly organ damage begins early in the course of Hypertension [3,4]. Organ can sustain damage long before symptoms appear; therefore the early intervention is increasingly recognized as generalized vasoconstriction and increased BP. It also increases the release of nor-adrenaline from sympathetic nerve terminals, reinforcing vasoconstriction an increasing both the rate and farce of heart contractions. It increases the reabsorption of salt and water. Through its aldosterone releasing effect from adrenal cortex. Increasing sodium levels in extracellular fluid, leading to water retention leads to ↑ Co, PVR and BP [5].

Based on levels of systolic blood pressure and diastolic blood pressure reading, hypertension can be classified into 4 stages (National Heart, Lung and Blood institute (NHLB1) of US].

Accordingly

SBP ≤ 120 and DBP ≤ 80 considered as normal level of Blood pressure

Pre hypertension	SBP 120 – 139 mm of Hg
	DBP 80 - 89 mm Hg
Stage I Hypertension	SBP 140 - 159 mm Hg
	DBP 90 - 99 mm Hg
Stage II Hypertension	SBP 160 - 179 mm Hg
	DBP 100 - 109 mm Hg
Stage III Hypertension	SBP 180 - 209 mm of Hg
	110 – 119 mm of Hg
Stage IV Hypertension	SBP > 210
	DBP > 120

Resistin

Resistin is a Cysteine – rich protein secreted by adipose tissue of mice and rats. In mammals, resistin is secreted by immune and epithelial cells [6]. Resistin is found in inflammatory zones 3 (FIZZ3), or "adipocyte – specific secretory factor" (ADSF). The length of resistin pre-peptide in human is 108 aminoacids (in the mouse and rat it's 114 aa); the

molecular weight is ~ 12.5 KDa. Among the proteins synthesized and released from adipose tissue (adiponectin, angiotensin, estradiol, IL-6, leptin, PA1-1 TNF-a) resistin is a cytokine with potent proinflammatory properties.

Resistin was discovered in 2001 by the group of Dr. Michell A. Lazar from the University of Pennsylyania school of Medicine. It was called "resistin" because of the observed insulin resistance in mice injected with resistin. Resistin was found to be produced and released from adipose tissue to serve endocrine functions likely involved in insulin resistance. This idea primarily stems from studies demonstrating that serum resistin levels increase with obesity in several model systems (humans, rats, and mice). Since these observations further research has linked resistin to other physiological systems such as inflammation and energy homeostasis. This study is focused on serum resistin level in essential hypertension and their relationship between GFR & serum resistin level. Serum resistin level can also be correlated with stages of hypertension.

Materials and methods:

This study was carried out in a tertiary care centre, Thanjavur Medical college Hospital, Thanjavur.

Study Participants:

The study group consisted of 60 patients with stage I (> 140/90mmHg) and stage II (>160/110 mm of Hg) Hypertension and 25 narmotensives as control

Clinical Examination:

B.P was recorded in sitting posture with sphygmomanometer, Height and weight were measured.

Inclusion Criteria:

1. Age (36-80years)
2. BP $\geq$140/90mm of Hg

Exclusion Criteria:

Diabetes Mellitus

Dyslipidemia

Chronic renal failure

Obesity

H/O Intake of Drugs:

1. Statins (Atrova statin, Lova statin etc)
2. Thiazolidinediones

Determination of Serum Resistin By Elisa:

Sample Collection & Storage:

Venous blood is collected without any anti-coagulant. Allow the blood to clot at room temperature for 30 min. centrifuge the clotted blood at 2000-3000xg for 15 minutes at $4\pm2^{o}C$. Samples are stored at $\leq 20^{o}C$.

Principles of Procedure:

This assay is based, sequentially, on

1) Capture of human resistin from sample by a monoclonal antibody immobilized in the wells of a microwell plate.
2) Washing off unbound materials including free materials from samples.
3) Binding of the biotinylated monoclonal human resistin antibody to the other side of captured human resistin molecule.
4) Conjugation of SA-HRP (PolyHRP-labelled streptavidin) enzyme to biotinylated antibody
5) Quantification of bound detection conjugates by monitoring SA-HRP enzyme activity in the presence of tetramethylbenzidine substrates.

The enzyme activity is measured spectrophotometrically by the absorbency at 450 nm. Since the amount of photometric product is directly proportional to the concentration of human resistin in the unknown sample its concentration can be calculated.

Results and Discussion:

Group = Test

		BP (S)	**BP (D)**	**GFR**	**SERUM RESISTIN**
BP (S)	Pearson Correlation	1	.157	-.669**	.733**
	Sig. (2-tailed)		.232	.000	.000
	N	60	60	60	60

BP (D)	Pearson Correlation	.157	1	-.092	.096
	Sig. (2-tailed)	.232		.487	.466
	N	60	60	60	60
GFR	Pearson Correlation	-.669**	-.092	1	-.546**
	Sig. (2-tailed)	.000	.487		.000
	N	60	60	60	60
SERUM RESISTIN	Pearson Correlation	.733**	.096	-.546**	1
	Sig. (2-tailed)	.000	.466	.000	
	N	60	60	60	60

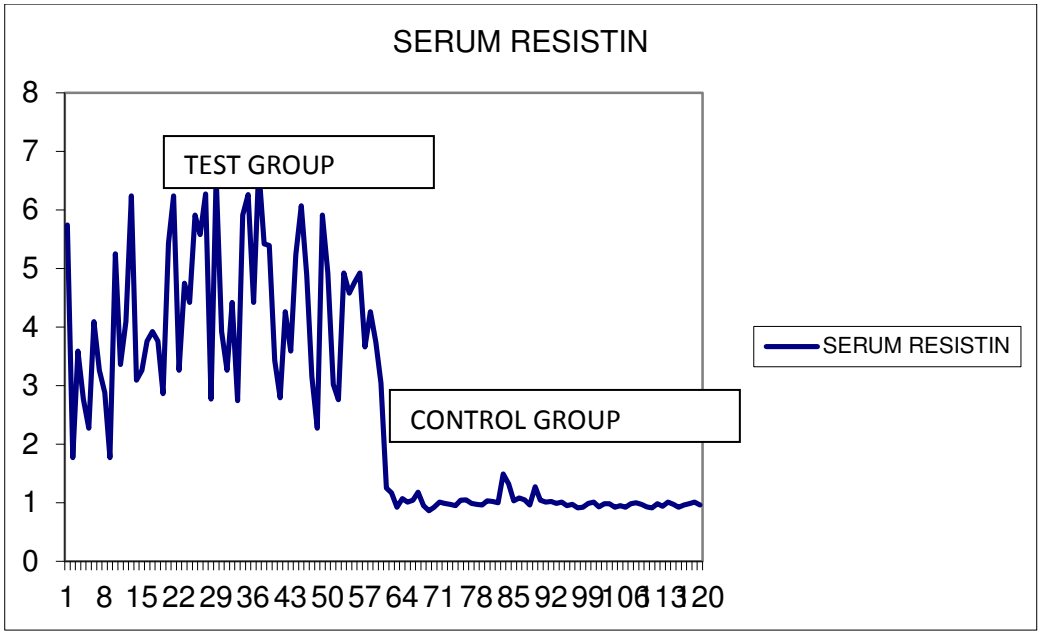
**. Correlation is significant at the 0.01 level (2-tailed).

Group = Control

		BP (S)	**BP (D)**	**GFR**	**SERUM RESISTIN**
BP (S)	Pearson Correlation	1	.013	-.165	.159
	Sig. (2-tailed)		.919	.208	.224
	N	60	60	60	60
BP (D)	Pearson Correlation	.013	1	.237	-.133
	Sig. (2-tailed)	.919		.068	.312
	N	60	60	60	60
GFR	Pearson Correlation	-.165	.237	1	-.116
	Sig. (2-tailed)	.208	.068		.376
	N	60	60	60	60
SERUM RESISTIN	Pearson Correlation	.159	-.133	-.116	1
	Sig. (2-tailed)	.224	.312	.376	
	N	60	60	60	60

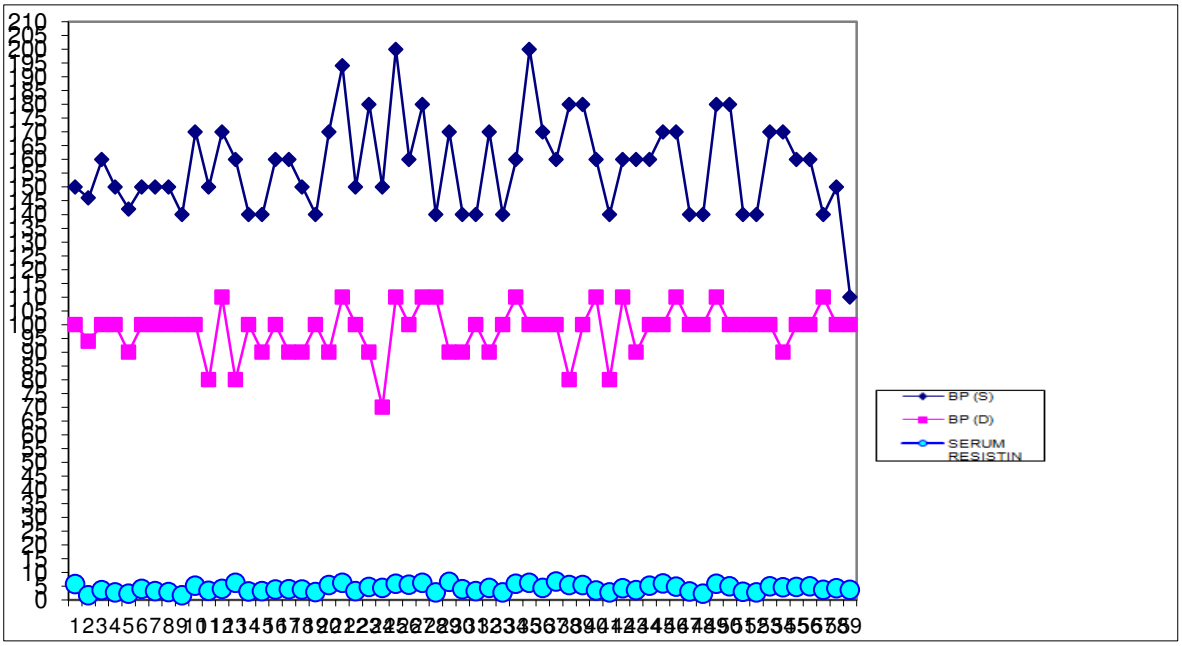
When compared to control group, in test group serum resistin level was found to be highly positively correlated with BP **(0.733**)** and it was significant at ($P < 0.000$). Serum resistin level was found to be negatively correlated with GFR (**-0.546****) and it was significant at ($P < 0.001$).

COMPARISON OF SERUM RESISTIN LEVEL IN TEST AND CONTROL GROUP:



This graph shows increased serum resistin level in test group compared to control group.

COMPARISON BETWEEN SERUM RESISTIN LEVEL AND BP IN TEST GROUP:



This graph shows correlation between serum resistin level and blood pressure in test group. It shows that serum resistin has positive correlation with blood pressure.

Conclusion:

Essential hypertension is major risk factor for chronic kidney disease. This study of serum resistin level in essential hypertension without known diabetes mellitus, myocardial infarction or stroke reveals that there is marked increase in serum resistin level in patients with essential hypertension compared to normotensive. The salient finding of our study is that hypertensive subject with higher resistin levels are characterized by lower GFR values, the most established index of kidney dysfunction. The remarks of our study exhibits that hypertensive subjects with augmented resistin level shows impaired GFR and suggests the major role of resistin in the progression of kidney damage even in early stages of essential hypertension. These findings indicate that augmented levels of resistin may be a risk factor for kidney and it can be considered as an early predictor of kidney dysfunction in hypertensive subjects.

References:

1. P.M. Kearney, M. Welton, K,Reynolds, et al; Global burden of Hypertension : analysis of world wide data; Lancet 2005, 365 : 217 – 223.
2. Harrison, Principles of Internal Medicine, 17th edition, Vol 2, 1522 – 1533.
3. JD Neaton, L.Kuller, J.Stamier et al; Impact of systolic and diastolic blood pressure on cardiovaruluar Mortality. In : Laragh, B.M. Brenner editor, Hypertension Pathophysiology, diagnosils and management, 2nd Edition, 1995 : 127– 144.
4. D.L.clement, D.E Buyzere, De Bacquel. Et al: prognostic value of ambulatory blood pressure recordings in patients with treated hypertension; N Engl J Med 2003; 24: 2407 – 2415.
5. G.Beevers, G.H. Lip, o' Brien; ABC of Hypertension: the pathophysiology of hypertension, BMJ 2001; 322: 912 – 916.S.

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