IJBPAS, March, Special Issue, 2025, 14(3): 662-671

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

CLINICAL ASSESSMENT OF RESISTIN IN CARDIOVASCULAR DISEASE PATIENTS

APPANABOYINA SK AND PULIGILLA S*

Department of Pharmacy, Chaitanya deemed to be University, Warangal-506001, Telangana, India

***Corresponding Author: Dr. Puligilla Shankaraiah; E Mail: drspuligilla@gmail.com**

Received 25th Dec. 2024; Revised 9th Jan. 2025; Accepted 23rd Feb. 2025; Available online 15th March 2025

https://doi.org/10.31032/IJBPAS/2025/14.3.1084

ABSTRACT

Objective: The study is to assess the resistin concentrations in Cardiovacular diseases and Other Co-morbidities of Cardiac patients. The underlying cause of cardiovascular diseases (CVD) and coronary artery disease (CAD) are understood to be inflammation. A significant factor in the etiology of CAD and HF are subclinical inflammations by resistin. Resistin adipokine is one of several pro-inflammatory indicators that can be used to identify the onset of a disease. In order to ascertain its correlation with CVD and other Co-morbidities.**Method**: There is 206 CVD patients were recruited for this study and then split into five groups based on the co-morbidities that were linked to them. Numerous biochemical and anthropometric markers like Resistin, Lipid profiles, blood glucose and BMI were evaluated. **Results**: The results were obtained that when compared to controls, CVD patients of Males, Type-2 Diabetes and CAD, HF were greater resistin levels than the females of CVDs and its subgroups had considerably higher and significant circulating levels of resistin. Point to a critical function of biomarker resistin in the onset of coronary artery disease and other cardiac problems. **Conclusion**: The current results found that there are co-morbid conditions such as type 2, diabetes, CAD, Hypertension can be a major contributing causes to the higher resistin levels in cardio vascular diseases.

Keywords: Cardiovascular diseases, Resistin, Obesity, lipid profile and Type-2 Diabetes

INTRODUCTION

In a developing nation like India, cardiovascular disorders are the main causes of death of patients. Cardiovascular disease is believed to be mostly caused by lifestyle disorders such as obesity, diabetes and hypertension. The underlying causes of the early onset of these metabolic syndromes include unhealthy diets, over weight and obesity, lifestyle of sedentary and excessive consumption of alcohol and tobacco use around the world, like the Diabetes mellitus and cardiovascular diseases (CVDs) constitute the leading cause of death **[1]**.

According to estimates CVDs were responsible for the millions of deaths **[2]**, there are the two main causes of cardiovascular diseases related to health problems worldwide were ischemic heart disease (IHD) and stroke by 2030 and there would claim the lives of millions of people annually. There is 75% of CVD fatalities now occurring in populations among the low and middle income countries, which has caused by a decline in their GDPs and fails the implementation of various efforts aimed at promoting early illness identification and management as well as population-wide shifts toward healthy behavior, is necessary to reduce the burden of CVD **[3]**.

The numerous scientific research organizations have conducted significant studies on the pathophysiology of coronary artery disease (CAD) within CVD; nevertheless, recent research continues to find various additional aspects involvement of multiple biomolecules in the progression of CVD and in most studies associated with the onset and progression of CAD disease **[4]**. Adipose tissue releases adipokines, which are bioactive molecules that are linked to a number of inflammatory changes linked to CAD. These chemicals also play a major role in atherogenesis **[5]**, resistin is an adipokine that is thought to be linked to inflammatory changes in CAD. It has a negative impact on the arterial wall by promoting causes of endothelial dysfunctions. The stromal vascular component of adipose tissue cells, which comprises bone marrow cells, macrophages are the main source of resistin is a cysteine-rich peptide hormone **[6]**. Human mononuclear cells primarily express and secrete circulating resistin in response to inflammatory stimuli, which raises the amount of resistin in the bloodstream **[7].** The pathophysiology of obesity, cardiovascular diseases and type 2 diabetes mellitus is known to be significantly influenced by inflammation **[8]**. Elevated serum resistin levels have been linked to the clinical atherosclerotic

progression of cardiovascular disease and have been implicated in a significant role **[9]**. The current study therefore sought to assess the resistin in CVD and Type-2 Diabetes mellitus.

MATERIALS AND METHODS

Study design

This study was conducted from at tertiary care hospital in warangal after Human Ethical Committee approvals and written inform consent was acquired from all the patients., from the Cardiology department, 206 patients in all both male and female were enrolled.

As per the predetermined parameters of inclusions and exclusions of patients were chosen for the study. The CVD groups were further divided according to the existence of co-morbid conditions such as 1. hypertension (HTN) alone, 2. HTN & CAD group, 3. CAD & Heart failure (HF)group, 4. CAD & type 2 diabetes mellitus (DM) Group, 5.HTN+CAD & type 2 diabetes mellitus (DM) Groups were Clinically proven.

The Anthropometric measurements were taken into consideration for all chosen subjects. Age sex, Scocial history, Dietary history and exposure to metabolic disorders (obesity, diabetes, and HTN) were documented for each participant.

Biochemical analysis:

Following a 12-hours fasting the patients' blood samples were taken and after being separated the serum was kept in aliquots at -40°C until it could be examined further. The clinical laboratory measured all of the standard biochemical tests, such as blood **[10],** glycosylated hemoglobin **[11]** and Resistin **[12]** and lipid profiles **[13]**.

Statistical analysis:

The mean ± SD was used to express all data with statistically. Using their frequencies, the gender distribution within the CVD subgroups was examined. The independent student's t test was used to compare the CVD patients with controls. The CAD subgroups were compared using one-way analysis of variance (ANOVA). Statistical significance was defined as probability values less than 0.05. Version 23 of IBM SPSS statistical software was used for all analyses.

RESULTS

Patients categorized according to their co-morbid conditions such as diabetes mellitus, CAD, HF, DM and hypertension. The distribution of the subjects by gender is shown in **Table 1**.

There were 55.2% of men and 44.8% of women with HTN. Males are more susceptible to the conditions according to the current study but this needs to be confirmed the given a bigger population **(Table 1)**. Group 2

CAD+HTN showed a similar pattern with 17.5.2% of patients being female and 82.5% of patients being males. Males with CAD were more prevalent in groups 3 (CAD+HF), group4 (CAD+DM) and 5 (CAD+DM+HTN), both with and without co-morbidities.

The mean age of control group is 46.5±1.3, while the control group's was 57.4± 4.1 years **(Table 2)**. The current investigations found between the control and CVD patients of mean fasting sugar ($p<0.001$) and HbA1c ($p<0.001$) **(Table 2)**. It appears that hyperglycemia speeds up to atherogenesis by increasing the production of glycated end products as well as by aggravating endothelial dysfunctions.

All CVD Patient cases and control subjects of their Lipid profiles were assessed. When the CAD patients were compared to control patients, the mean total cholesterol, triglycerides, LDL-cholesterol, and VLDL-cholesterol increased significantly ($p<0.001$) while HDL-cholesterol was significantly ($p<0.001$) lower in CVD patients **(Table 2)**, when compared to one another. The patients' serum concentrations have the resistin level of 15.72±4.5 ng/ml, while the control group's was 6.2±0.42. Comparing the control group to the CVD patients group raised the significant mean of resistin concentrations ($p<0.001$) **(Table 2 and Figure 1)**.

This illustrates to how elevated resistin levels are linked to cardiovascular diseases and additionally variations in CVD **(Table 3)**. (CAD+DM) group and group (CAD+HTN) had higher resistin levels out of all the subgroups and Group (CAD+DM+HTN) had the highest mean resistin levels, it indicating that resistin's superior potential as a biomarker for CVD related. In humans, resistin has been shown to increases the vascular smooth muscle cells' abnormal activity. CVD sub-groups and cholesterol and LDL levels likewise showed significant differences **(Table 3)**.

Table 1: Comparison of cardiovascular diseases on Gender distribution subjects in the study

Genders/Parameters	Male N (%)	Female N (%)	Total
HTN	39(55.2)	32(44.8)	71
CAD with HTN	21(82.5)	04(17.5)	25
CAD with HF	14(85.7)	02(14.3)	16
CAD with DM	44(79.2)	12(20.8)	56
CAD with DM +HTN	34(88.9)	04(11.1)	38

Table 2: Comparison of Anthropometric and biochemical parameters of Normal and CVD patients

CVD/Parameters	Normal (mean ±SD)	CVS DISEASE (mean ±SD)	p-value
Age (years0	36.3±2.4	42.5±1.8	0.6
BMI (kg/m2)	25.64±3.2	26..22±2.41	0.5
Fasting Sugar (mg/dl)	96.23±5.7	143.34 ±13.28	0.001
HbA1c(%)	5.64±1.12	6.94±1.2	0.001
Cholesterol(mg/dl)	151.21±8.24	192.52±9.2	0.001

Triglyceride(mg/dl)	**124.15±9.12**	**198.23±8.21**	**0.001**
HDL(mg/dl)	**48.23±4.3**	**32.21±2.4**	**0.01**
LDL(mg/dl)	**75.4±5.3**	**104.23 ±6.12**	**0.01**
VLDL(mg/dl)	**24.83±1.04**	**39.64±2.6**	**0.001**
Resistin (ng/ml)	**6.23±0.24**	**15.74±2.26**	**0.001**

(Data values Mean±SD and p- values *p<0.05, ** p<0.01, *** p<0.001, Compared Normal with CVD).

Table 3: Serum Resistin & lipid profile levels in CVD and sub-clinical CAD patients compare with Diabetes mellitus

Groups/ Parameter	HTN (n=71) (Mean ±SD) 34.4%	CAD+HTN (n=25) (Mean ±SD) 12.1%	CAD +HF (n=16) (Mean ±SD) 0.07%	HTN+DM (n=56) (Mean ±SD) 27.18%	CAD+DM+ HTN (n=38) (Mean ±SD) 18.4%	*p*-value
Age of Patients	**46.4±3.04**	**53.1±4.02**	**59.3±4.2**	**59.2±3.41**	**61.32±2.4**	**0.658**
BMI (kg/m2)	**24.5.2±3.2**	**26.31±2.1**	**25.3±1.06**	**25.62±1.5**	**27.8±1.3**	**0.642**
Fasting sugar(mg/dl)	**82.6±13.2**	**92.3±11.21**	**86.4±13.1**	**187.3±12.4**	**232.4±21.8**	**0.001**
HbA1c(%)	**4.98±0.31**	**4.98±0.31**	**5.02±0.30**	**7.92±1.04**	**8.96±1.21**	**0.001**
Cholesterol(mg/dl)	**183.24±11.4**	**167.12±13.1**	**196.4±13.2**	**178.8±21.2**	**188.22±14.3**	**0.054**
Triglycerides(mg/dl)	**195.2±14.3**	**182.83 ±15.3**	**198.2±11.4**	**165.8±17.2**	**221.1±16.3**	**0.055**
HDL(mg/dl)	**43.8±1.02**	**41.4±2.4**	**35.3±3.21**	**35.6±3.2**	**33.12±2.7**	**0.0643**
LDL(mg/dl)	**145.6±15.2**	**132.44 ±12.1**	**106.11±14.2**	**114.57 ±11.2**	**141.22±22.3**	**0.012**
VLDL(mg/dl)	**39.04±2.1**	**36.56±4.1**	**39.64±2.8**	**33.16 ±3.2**	**44.22±5.2**	**0.623**
Resistin (ng/ml)	**6.1±0.31**	**7.4±0.66**	**6.8±0.65**	**11.3±0.5**	**15.23±1.03**	**0.001**

(Data values Mean±SD and p- values *p<0.05, ** p<0.01, *** p<0.001, Compared Normal with CVD and CAD+DM)

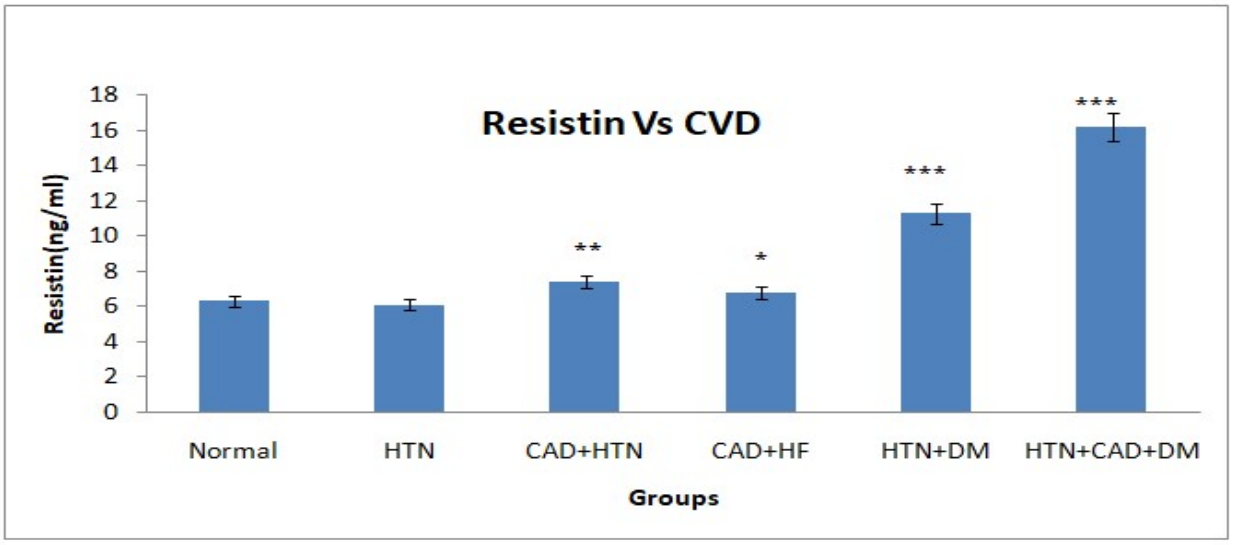

Figure 1: Comparison of serum resistin levels in cardiovascular diseases and CAD Patients (Data values Mean±SD and p-values *p<0.05, ** p<0.01, *** p<0.001, Compared Normal with CVD and CAD+DM)

DISCUSSION:

The development of risk factors and their relationships to the occurrence of different diseases have been established by global prospective epidemiological study **[14]**. These investigations have consistently shown a correlation between the occurrence of CVD and characteristics examined in healthy persons. The relationship between age and the prevalence of an illness has always been crucial. According to our research the incidence of CAD rises as sub-groups of

people age. it consistent with risk to getting CAD rises with age, including for men over 45 and women over 55. The data remained the non-significant when comparing the CAD patients' ages across the subgroups created based on co-morbidities **(Table 3) [15]**, a low body mass index (BMI) is linked to a higher risk of CAD in its early stages.

The BMI values of the control and CVD groups and the subgroups did not vary in the current investigations. Obesity and excess weight are regarded as risk factors for metabolic syndrome and cardiovascular diseases but not all overweight people get CAD, the wide range of factors involved has left clinicians perplexed. Numerous studies have connected a person's fat distribution, particularly the buildup of intra-abdominal fat, to metabolic abnormalities linked to diabetes mellitus, coronary artery disease, and atherosclerosis **[16]**.

DM has been linked to an increased incidence and risk of developing CVD and CAD. Glycated hemoglobin (HbA1c) and impaired fasting blood sugar have long been utilized to identify diabetes mellitus. Since HbA1c readings give an average glucose concentration in plasma over a period of two to three months, they are more useful as a gauge of long-term glycemic status **[17]**. Both microvascular and macrovascular diseases are expected to be exacerbated by these direct effects of hyperglycemia. In patients of CVD, our findings are consistent with other research showing a link between fasting blood sugar levels and HbA1c **[18]**.

The biggest risk factor for CVD is still hyperlipidemia **[19]**. The significance of the lipid profile in the pathophysiology of cardiovascular disease has been demonstrated by numerous research reports. Elevated triglycerides and total cholesterol levels may have an impact on heart artery constriction and abstractions, which are closely associated with cardiovascular events (CAD, HF, HTN). Atherogenesis may result from elevated LDL levels, On the other hand, people who have greater HDL-cholesterol levels might be reduces the cardiovascular diseases and may have a lower risk of CAD **[20, 19]**.

Insulin resistance is the result of resistin adipokine's interference with insulin signalings, which prevents insulin secretions from doing its job By stimulating inflammatory molecules including TNF-Alpha, IL-6, and others, resistin has been found to play a significant role in enhancing inflammation and additionally it is essential for promoting NF-KB expressions, which amplifies the various inflammatory cascades **[21, 22]**.

This suggests a strong correlation between resistin and inflammatory markers **[23]**. By altering the expression of cell adhesion molecules and producing oxidative stress, resistin biomarker have more impact on vascular endothelial dysfunctions like inflammatory mediators.

Atherosclerosis is ultimately CAD may result from this process. Research in to resistin's function as a stand-alone as biomarker and the effects of its imbalance on CVD and CAD conditions is becoming more and more popular. In order to determine the resistin has a potential correlation with CAD **[24]**.

According to previous research, resistin has linked to coronary heart disease and diabetes mellitus. Studies of resistin have shown similar to those of normal populations, in addition to showing that there are increased in individuals with CVD and CAD association patients.

CONCLUSION

According to the current study, CVD patients have greater levels of resistin and Cholesterol than controls. Additionally, it has been noted that CAD patients with co-morbidities such as HTN, HF and type 2 diabetes mellitus had incriminatingly elevated the resistin levels. The onset of CVD, metabolic diseases, and early inflammation may all be likely caused by an imbalance in resistin levels.

ACKNOWLEDGEMENTS

All authors are thankful to Chaitanya deemed to be University for providing the research facilities and to all of the participants for taken part of the study.

CONFLICT OF INTEREST: No conflict of interest

REFERENCES

[1] Sreeniwas Kumar, A., & Sinha, N. Cardiovascular disease in India: A 360 degree overview. Medical Journal, Armed Forces India 76(1) (2020) 1-3.

[2] Ruan, Y., Guo, Y., Zheng, Y., Huang, Z., Sun, S., Kowal, P., Wu, F. (2018). Cardiovascular disease (CVD) and associated risk factors among older adults in six low-and middle-income countries: Results from SAGE Wave 1.BMC Public Health 18(1) (2018) 775-778.

[3] Mc Namara, K., Alzubaidi, H., & Jackson, J.K. Cardiovascular disease as a leading cause of death: How are pharmacists getting involved? Integrated Pharmacy Research & Practice 8 (2019) 1-11.

[4] Libby, P., & Theroux, P. (2005). Pathophysiology of Coronary

Artery Disease. Circulation, 2005; 111(25), 3481-3488.

[5] Sinan, U. Y., Cetinarslan, O., Oktay, V., Calpar, I., Dalgic, Y., Kaya, A. Ersanli, M. K. The Association between Serum Resistin Levels and Major Adverse Cardiac Events. World Journal of Cardiovascular Diseases 7(1) (2017) 332-339.

[6] Pang, S. S., & Le, Y. Y. Role of resistin in inflammation and inflammation- related diseases. Cellular & Molecular Immunology 3(1) (2006) 29-34.

[7] Reilly, M.P., & Rader, D.J. The Metabolic Syndrome. Circulation 108(13) (2003) 1546-1551.

[8] Libby, P., Ridker, P.M., & Hansson, G.K. Inflammation in Atherosclerosis: From Pathophysiology to Practice. Journal of the American College of Cardiology 54(23) (2009) 2129-2138.

[9] Emamalipour, M., Seidi, K., Jahanban-Esfahlan, A., & Jahanban-Esfahlan, R. Implications of resistin in type 2 diabetes mellitus and coronary artery disease: Impairing insulin function and inducing pro-inflammatory cytokines. Journal of Cellular Physiology 234(12) (2019) 21758-21769.

[10] Trinder. P. Determination of Glucose in Blood using Glucose Oxidase. Annals of Clinical Biochemistry 6(1) (1969) 24-27.

[11] Rochman H. Hemoglobin A1c and Diabetes Mellitus. Ann Clin Lab Sci. 10(2) (1980) 111-115

[12] Bo S, *et al*. Relationships between human serum resistin, inflammatory markers and insulin resistance. Int J Obes (Lond) 29 (2005) 1315-1320.

[13] Nordestgaard BG, Langsted A, Mora S, *et al*. Fasting is not routinely required for determination of a lipid profile: clinical and laboratory implications including flagging at desirable concentration cut-points a joint consensus statement from the European Atherosclerosis Society and European Federation of Clinical Chemistry and Laboratory Medicine. Eur Heart J 37 (2016) 1944-1958.

[14] Bhori, M., Rastogi, V., Tungare, K., & Marar, T. A review on interplay between obesity, lipoprotein profile and

nutrigenetics with selected candidate marker genes of type 2 diabetes mellitus. Molecular Biology Reports 49(1) (2022) 687-703.

[15] Alkhawam, H., Nguyen, J., Sayanlar, J., Sogomonian, R., Desai, R., Jolly. J. Rubinstein, D. Coronary artery disease in patients with body mass index ≥30 kg/m2: A retrospective chart analysis. Journal of Community Hospital Internal Medicine Perspectives 6(3) (2016) 10.

[16] Singh, R., Prakash, M., Dubey, R., Roy, K., Kotwal, A., & Mahen, A. Body composition parameters as correlates of coronary artery disease. The Indian Journal of Medical Research 138(6) (2013) 1016-1019.

[17] Hong, L.F., Li, X. L., Guo, Y. L., Luo, S. H., Zhu, C. G., Qing, P. Li, J. Glycosylated hemoglobin A1c as a marker predicting the severity of coronary artery disease and early outcome in patients with stable angina. Lipids in Health and Disease 13 (2014) 89.

[18] Saha, A., Kuila, M., & Sharma, R. K. Relationship of severity of coronary artery disease with fasting blood sugar and glycosylated hemoglobin. Asian Journal of Medical Sciences 13(3) (2022) 38-42.

[19] Haddad, F. H., Omari, A. A., Shamailah, Q. M., Malkawi, O. M., Shehab, A. I., Mudabber, H. K., & Shubaki, M. K. Lipid profile in patients with coronary artery disease. Saudi Medical Journal 23(9) (2002) 1054-1058.

[20] Zhao, X., Wang, D., & Qin, L. Lipid profile and prognosis in patients with coronary heart disease: A meta-analysis of prospective cohort studies.BMC Cardiovascular Disorders 21(1) (2021) 69.

[21] Zhang, J. Z., Gao, Y., Zheng, Y. Y., Liu, F., Yang, Y. N., Li, X. M., Xie, X. Increased serum resistin level is associated with coronary heart disease. Oncotarget, 8(30) (2017) 50148–50154.

[22] Jamaluddin, M. S., Weakley, S. M., Yao, Q., & Chen, C. Resistin: Functional roles and therapeutic considerations for cardiovascular disease. British Journal of Pharmacology 165(3) (2012) 622-632.

[23] Yıldırım Ö, T. (2018). Can resistin be used as a biomarker in coronary artery disease: Archiview. Frontiers in Drug, Chemistry and Clinical Research 1(1) (2018) 1-4.

[24] Verma, S., Li, S.H., Wang, C. Fedak, P. W. M., Li, R, Weisel, R. D., & Mickle, D. A. G. Resistin Promotes Endothelial Cell Activation. Circulation 108 (6) (2003) 736-740.