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Original Research Article

A Hospital-Based Study to Assess the Level of Apelin and Lipid Profile in Hypertensive Patients: A Retrospective Study

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Abstract

Aim: The aim of the present study was to assess the level of Apelin and lipid profile in hypertensive patients.

Methods: The project was a classic sectional study done at ESICMCH, Bihta, Patna, Bihar, India from August 2022 to September 2023. Eighty-nine people were recruited (56 females and 33 males) and divided into two groups: the control group included 25 healthy people and the patients' group included 64 hypertensive patients.

Results: The current study included (89) participants, with a group of patients (n=64) compared to the control group of healthy adults (n=25). There are statistically significant differences between the patients and the control group in all biochemical variables ($p \leq 0.01$). There was a significant increase in hypertension. On the other hand, there was a significant decrease in apelin and UHDL-c. There was an inverse relationship was observed in DLDL-c (LDL-c cal.) and cholesterol in both patients and control, while with systolic and diastolic pressure an inverse relationship appeared with the control group only whereas a direct relationship with patients. The non-HDL-c change is significant in the control group but in the patients' group nonsignificant with UHDL-c. The UHDL-c and Non-HDL-c were in positive correlation in the control group significant ($p<0.000$) and patients group nonsignificant. Non-HDL-c appears direct correlation with DLDL-c, as well as triglycerides and cholesterol ($p<0.001$) in both patients and control groups. Also, the systolic for the control group was significant with non-HDL-c ($p<0.027$), but the Diastolic was nonsignificant with non-HDL-c ($p<0.143$).

Conclusion: The present concluded that high blood pressure is associated with increased harmful fats (Non-HDL-c) in the body, which are deposited on the walls of the arteries causing them to narrow, which leads to high blood pressure. Also, an increase in apelin peptide concentration in blood is a good indicator. On the other hand, a deficiency of apelin combined with high blood pressure.

Keywords: Lipid profile, apelin, hypertension

Introduction

The significant global burden of hypertension is a risk factor for cardiovascular morbidity and mortality. Per Current guidelines, hypertension is classified into isolated systolic (ISH), isolated diastolic (IDH), and systolic and diastolic combined hypertension. [1-3] The seventh Joint National Committee (JNC-7) report first reported prehypertension. Elevated blood pressure is due to a variety of risk factors, which among obesity is proven as closely interrelated to hypertension. [4] Studies reported that body mass index (BMI) and waist circumference (WC), fat accumulation, abnormal fat distribution, accumulation of subcutaneous adipose tissue and visceral adipose tissue, and dyslipidemias are seen in hypertension. [5,6] Apelin is a hormone peptide in adipose, cardiovascular, pulmonary, and cerebral tissues. Various active apelin peptides exist in 36, 17, and 13 amino acids, originating from preproproteins consisting of 77 amino acid residues. [7,8] So, apelin-13 has the highest activity of endogenous ligand for the G protein-coupled receptor apelin receptor (APJ) and plays an essential role in the cardiovascular system. [9]

Cardiovascular diseases (CVDs) are the leading cause of death worldwide, accounting for nearly 4 million fatalities (45 percent of all deaths) in Europe each year. [10] Important risk considerations are lipoprotein changes are responsible for around half of all CVDs. Include high levels of total cholesterol (TC), low levels of low-density lipoprotein cholesterol (LDL- c) and low levels of high-density lipoprotein cholesterol (HDL-c) concentrations. LDL- cholesterol, intermediate-density lipoproteins, VLDLs, and non-HDL-c cholesterol show the total cholesterol carried by all potentially atherogenic particles and lipoprotein remnants. European

guidelines propose lowering TC and LDL-c levels as primary targets in treatment approaches because it includes remnant cholesterol and is unaffected by triglyceride fluctuation. Non-HDL-c considers a better measure. [11]

Apelin is an adipokine discovered by Tatemoto et al. in 1998. Apelin is expressed and secreted by both mouse and human adipocytes. In adipocytes, insulin can upregulate apelin expression. The expression of apelin in adipose tissue of humans swiftly cleaved from circulation with a half-life of <5 minutes. Furthermore, apelin and its receptor, the orphan G protein-coupled receptor (OGPR) and APJ receptor are expressed in pancreatic islet cells. [12] Apelin has a regulator effect of glucose stimulation on insulin production. [13]

The aim of the present study was to assess the level of Apelin and lipid profile in hypertensive patients.

Materials and Methods

The project was a classic sectional study done at ESICMCH, Bihta, Patna, Bihar, India from August 2022 to September 2023. Eighty-nine people were recruited (56 females and 33 males) and divided into two groups: the control group included 25 healthy people and the patients' group included 64 hypertensive patients.

Eligibility Criteria

In this study, all patients were diagnosed as hypertensive and attending Internal medicine consultants. Patients with high blood pressure accompanied by one of the following diseases are excluded: diabetes, kidney failure, and thyroid disease.

Measurements

The pressure was measured electronically with a device from the Beurer company in a sitting position after 5 min of rest. Body mass index (BMI) was calculated after measuring weight and height. Five ml of blood was collected the next day after an overnight fast (10–12 h) to assess the fasting serum lipid profile. The lipid profile includes: total cholesterol (TC), triglyceride TG, Ultra high-density lipoprotein-cholesterol (UHDL-c), low-density lipoprotein- cholesterol (LDL-c) both Direct LDL-c (DLDL-c) and calculated (LDL-c cal.). They were analyzed using an enzymatic method on an automated chemistry analyzer, Selecta Pro M (ARCHITECT c4000). Non-HDL-c was calculated as TC minus UHDL-c. LDL-c cal. was calculated using the Friedewald formula as following: LDL-c cal. = (Total Cholesterol) − (UHDL-c) − (TGs/5).

Definition of Terms

According to the National Cholesterol Education Program (NCEP), dyslipidemia occurred at an elevated TG level of 1.7 mmol/L, reduced HDL-c to 1.04 mmol/L, LDL-c level >3.37 mmol/L, and/or a TC level of 5.2 mmol/L. [14] Hypertension is systolic blood pressure (SBP) of 140 mm Hg and/or diastolic blood pressure (DBP) of 90 mm Hg on the report of the Joint National Committee. [15] Blood pressure is controlled within an SBP <140 mm Hg and a DBP < 90 mm Hg by antihypertensive drug(s) and/or no pharmacological methods. Uncontrolled or poorly controlled BP is an SBP ≥140 mm Hg and/or DBP ≥90 mm Hg.

Results

Table 1: Variables and characteristics of patients and control groups

Group	n	n %	M/F	M/F%	Age (year)	BMI (Kg/m^2)
Control	25	29.1	11/14	44.8/55.2	34.4±12.18	30.61±7.73
Patients	61	70.9	32/29	52.4/47.5	52.13±12.56	28.93±7.23

The current study included (89) participants, with a group of patients (n=64) compared to the control group of healthy adults (n=25).

Table 2: Lipid profile, blood pressure and Apelin of patients and control groups

Biochemical	Control	Patients
Stolic. mmHg	117.4±14.53	154.8±18.6
Diastolic. mmHg	71.92±11.72	91.73±16.6
LDL-c cal. mmol/L	1.267±0.611	1.792±0.872
DLDL-c. mmol/L	1.336±0.607	1.973±0.703
Cholesterol. mmol/L	1.094±0.471	1.622±0.473
Triglycerides. mmol/L	1.097±0.638	2.311±0.559
Non-HDL-c. mmol/L	1.773±0.773	2.808±0.891
UHDL-c. mmol/L	0.791±0.375	0.949±0.246
Apelin. ng/L	73.60±20.75	43.30±53.1

There are statistically significant differences between the patients and the control group in all biochemical variables ($p \leq 0.01$). There was a significant increase in hypertension. On the other hand, there was a significant decrease in apelin and UHDL-c.

Table 3: The relationship of Apelin with other parameters of patients and control groups in terms of probability (P) and correlation coefficient (R)

Biochemical Parameter	Apelin			
	Control		Patients	
	R	P	R	P
Systolic	-0.223	0.295	0.19	0.885
Diastolic	-0.306	0.146	0.022	0.866
LDL-c cal.	-0.035	0.872	-0.103	0.430
DLDL-c	-0.028	0.89	-0.087	0.507
Cholesterol	-0.033	0.878	-0.010	0.941
Triglyceride	0.158	0.460	0.065	0.617
Non-HDL-c	0.032	0.883	0.034	0.796
UHDL-c	-0.094	0.663	0.003	0.985

There was an inverse relationship was observed in DLDL-c (LDL-c cal.) and cholesterol in both patients and control, while with systolic and diastolic pressure an inverse relationship appeared with the control group only whereas a direct relationship with patients.

Table 4: The relationship of UHDL-c with other parameters of patients and control groups in terms of probability (P) and correlation coefficient (R)

Biochemical Parameter	UHDL-c			
	Control		Patients	
	R	P	R	P
Systolic	-0.065	0.759	-0.084	0.519
Diastolic	-0.098	0.640	-0.067	0.607
LDL-c cal.	0.729	0.000	0.501	0.000
DLDL-c	0.710	0.0001	0.456	0.000
Cholesterol	0.737	0.000	0.446	0.000
Triglyceride	0.549	0.004	-0.011	0.932
Non-HDL-c	0.698	0.000	0.248	0.054

The non-HDL-c change is significant in the control group but in the patients' group nonsignificant with UHDL-c. The UHDL-c and Non-HDL-c were in positive correlation in the control group significant ($p<0.000$) and patients group nonsignificant.

Table 5: The relationship of non-HDL-c with other parameters of patients and control groups in terms of probability (P) and correlation coefficient (R)

Biochemical Parameter	Non-HDL-c			
	Control		Patients	
	R	P	R	P
Systolic	-0.443	0.027	0.178	0.169
Diastolic	-0.192	0.358	0.143	0.272
LDL-c cal.	0.954	0.000	0.524	0.000
DLDL-c	0.965	0.000	0.849	0.000
Cholesterol	0.967	0.000	0.965	0.000
Triglyceride	0.788	0.000	0.602	0.000

Non-HDL-c appears direct correlation with DLDL-c, as well as triglycerides and cholesterol ($p<0.001$) in both patients and control groups. Also, the systolic for the control group was significant with non- HDL-c ($p<0.027$), but the Diastolic was nonsignificant with non-HDL-c ($p<0.143$).

Discussion

Hypertension affects millions of patients and is recognized as a risk factor for cardiovascular events and cardiovascular mortality. Hypertension results from a complex interaction between environmental

factors, genetic influences, unhealthy lifestyle and abnormalities in the control mechanisms of the cardiovascular system. Consequently, identifying a specific cause for this condition is impossible in many patients, which explains the use of the terms essential or primary hypertension. [10]

Many theories offer to explain the hypertension phenomenon. The incidence of blood pressure (BP) increases anomalies in salt and water levels in a person handled by the kidneys. Especially through the intrarenal route of the renin-angiotensin-aldosterone system in the body. Also, deregulation of neuronal autonomic modulation of the circulatory system. These mechanisms are not mutually exclusive, and they could all have a role in the rise in blood pressure seen in many people with essential hypertension. Endothelial dysfunction and inflammation linked to heart disease in recent studies of the emergence of a hypertensive state. [16]

The current study included (89) participants, with a group of patients (n=64) compared to the control group of healthy adults (n=25). There are statistically significant differences between the patients and the control group in all biochemical variables ($p \leq 0.01$). There was a significant increase in hypertension. On the other hand, there was a significant decrease in apelin and UHDL-c. There was an inverse relationship was observed in DLDL-c (LDL-c cal.) and cholesterol in both patients and control, while with systolic and diastolic pressure an inverse relationship appeared with the control group only whereas a direct relationship with patients. The non-HDL-c change is significant in the control group but in the patients' group nonsignificant with UHDL-c. The results indicate there is a correlation between high blood pressure and an increase in non-HDL-c concentration, it is a direct relationship where R is positive. Also, there is a positive correlation between women and men with Non-HDL-c. It is no secret to everyone that the cause of high blood pressure is excess body fat. [17,18] However, high blood pressure has an inverse correlation with UHDL-c, and high UHDL-c is considered beneficial to health. [19]

The UHDL-c and Non-HDL-c were in positive correlation in the control group significant ($p<0.000$) and patients group nonsignificant. Non-HDL-c appears direct correlation with DLDL-c, as well as triglycerides and cholesterol ($p<0.001$) in both patients and control groups. Also, the systolic for the control group was significant with non-HDL-c ($p<0.027$), but the Diastolic was nonsignificant with non-HDL-c ($p<0.143$). The natriuretic peptide system is an endocrine system that controls salt and water balance and arterial blood pressure (BP). The natriuretic peptide precursor gene (NPPA) encodes the atrial natriuretic peptide (ANP), which is produced from atrial myocardium cells and helps to reduce cardiac stress by promoting bedwetting, vasoconstriction and other physiological actions mediated by natriuretic peptide receptors. [20] Since the action of apelin is to stimulate the ANP (apelin is a part of the natriuretic peptide system with ANP), Apelin works to inhibit the Angstein II hormone by competing with him to bind with receptors in the arteries to relax and expand the arteries. [21]

Conclusion

The present concluded that high blood pressure is associated with increased harmful fats (Non-HDL-c) in the body, which are deposited on the walls of the arteries causing them to narrow, which leads to high blood pressure. Also, an increase in apelin peptide concentration in blood is a good indicator. On the other hand, a deficiency of apelin combined with high blood pressure. Furthermore, a decrease in apelin levels is associated with increased non-HDL-c levels in the blood, which increase hypertension risk factors.

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