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Assessment of Atrial Natriuretic Peptide (ANP) and Lipid Profile for Hypertension Patients in Thi-Qar province, Iraq

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***Abstract*—The present study was designed to assessment of atrial natriuretic peptide (ANP) and lipid profile of patients with hypertension in Thi-Qar province, Iraq. The sample included 70 patients, who were attending Al- Nasiryiah heart center during the period from February 2019 to June 2019, with 30 healthy as a control group. The results showed a significant increase.(P≤0.05) of systolic blood pressure (SBP) and diastolic blood pressure (DBP) in hypertension patients compared with the control group. The level of atrial natriuretic peptide (ANP) showed a significant increase (P≤0.05) in patients compared with control group. Also the result indicated a significant increase (P≤0.05) of TC, TG, LDL and VLDL in patients compared with the control, while there was a significant decrease in HDL of patients compared with the control.**

Keywords*— *hypertension, Atrial natriuretic peptide (ANP), lipid profile

I. INTRODUCTION

Hypertension is characterized as a systolic blood pressure (SBP) greater than 140 mHg or a diastolic blood pressure (DBP) greater than 90 mmHg; the diagnosis is based on an average of 2 or more readings per 2 or more visits after initial screening (Holm *et al*., 2006) . Hypertension is a major health problem, particularly because the symptoms are not apparent. Most people get hypertension without being aware of it. Modifier factors such as obesity, overweight measured by body mass index, visceral adiposity measured by waist circumference, increased age, are now well known to be correlated with the high prevalence of hypertension (Dibby, 2015). High blood pressure is one of the most common world disorders which cause many effects on human body (Al-Hamdani, 2010). It is the main risk factor for cardiovascular disease, congestive cardiac failure, stroke and end-stage of kidney disease (Casey *et al*., 2006). Hypertension is one of manifestation of metabolic syndrome (Hassan *et al*., 2016).

Atrial natriuretic peptide (ANP) is a potent diuretic, vasorelaxant hormone, which is synthesized predominantly in the cardiac atria. In response to intravascular volume expansion and blood pressure (BP) elevation, this peptide controls sodium-water balance (Kato *et al*., 2000). Under normal hemodynamic conditions, it is predominantly synthesized, stored, and secreted in a regulated fashion by modified myocytes of the cardiac atria. However, in patho-physiological conditions of hemodynamic overload (in congestive heart failure, ventricular synthesis of the peptide) it is reactivated and contributes significantly to the circulating pool of the peptide (Ahmed *et al*., 2012).

The majority of hypertensive patients are diagnosed with elevated lipid rates such as raised cholesterol , triglycerides and/or reduced lipoproteins in high density (Lamina and Okoye, 2012). Although a specific pattern of dyslipidemia was not identified in people with high blood pressure, several studies have shown that cholesterol, triglycerides and other fatty proteins are abnormal in people with high blood pressure (Osuji *et al*., 2012). Dyslipidemia is more common in untreated hypertensive patients than healthy individuals and lipid profile values were increased as blood pressure in pattern of dyslipidemia and has been consistently reported among hypertensive patients (Murtadha, 2017). Also hypertension is known to be related with change of lipid metabolism which causes increase of lipid levels and progression of cardiovascular disease (CVD) (Chales *et al*., 2012). The present study aimed to assessment of ANP level and lipid profile of patients with hypertension in Thi-Qar province, Iraq.

II. MATERIALS AND METHODS

A. Study Population

The target population of this study was 70 patients (male 40 and female 30) who were already diagnosed as hypertension patients were attending the AL-Nasiriyah Heart Center in Thi- Qar province, Iraq during the period from February 2019 to June 2019 with age ranged between (30 - 80 years) . A control group was composed of 30 (male 16 and female 14) healthy with the same age range.

B. Blood pressure Measurement

Blood pressure was measured using a device sphygmomanometer.

C. Blood Sample

Blood samples were obtained by venipuncture from patients and control group. The blood sample was dispensed in a plain tube, and left for 15 minutes at room temperature to clot. Then, it was centrifuged at 3000 rpm for 10 minutes to collect serum and kept

in the freezer (-20ºC) until use unless used immediately to analyze biochemical parameters.

D. Atrial natriuretic peptide assay

The Atrial natriuretic peptide levels were analysed using commercially available Human ANP ELISA Kit from elabscience company, This ELISA kit uses the Competitive-ELISA principle. The micro ELISA plate provided in this kit has been pre-coated with Human ANP. During the reaction, Human ANP in the sample or standard competes with a fixed amount of Human ANP on the solid phase supporter for sites on the Biotinylated Detection Ab specific to Human ANP. Excess conjugate and unbound sample or standard are washed from the plate, and Avidin conjugated to Horseradish Peroxidase (HRP) are added to each microplate well and incubated. Then a TMB substrate solution is added to each well. The enzyme-substrate reaction is terminated by the addition of stop solution and the color change is measured spectrophotometrically at a wavelength of 450 nm ± 2 nm. The concentration of Human ANP in the samples is then determined by comparing the OD of the samples to the standard curve.

E. Determination of serum lipid profile

The lipid profile (total cholesterol, triglycerides, HDL, LDL and VLDL) was determined in the serum using COBAS INTEGRA systems.

F. Statistical analysis

Statistical analysis was done using the software SPSS version 23.0.The results were expressed as mean ± standard deviations (mean ± SD) with LSD. One way ANOVA-test was used to compare parameters in different studied groups. P-values ($P \leq 0.05$).

II. Results and discussion

A. Blood pressure

The results showed a significant increase ($P \leq 0.05$) of systolic blood pressure (SBP) (148.33±15.12) and diastolic blood pressure (DBP) (96.18±7.22) in hypertension patients compared with control group (124.48±6.46, 81.03±4.89) (table 1).

Table 1: Level of systolic blood pressure (SBP) and diastolic blood pressure (DBP) in patients and control group

Parameters / Groups	SBP (mmHg) Mean± SD	DBP (mmHg) Mean ±SD
Control	124.48±6.46 [b]	81.03±4.89 [b]
Patients	148.33±15.12 [a]	96.18±7.22 [a]
LSD	3.23	1.72

The different letters refers a significant different at $P \leq 0.05$

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) of male and female hypertension patients increased significantly ($P \leq 0.05$) compared with the control group (table 2).

Table 2: Systolic blood pressure (SBP) and diastolic blood pressure (DBP) of male and female patients with control group.

gender	Groups	SBP(mmHg) Mean± SD	DBP(mmHg) Mean ±SD
Male	Control	122.15±5.30 [b]	80.16±5.10 [b]
	Patients	147.13±12.22 [a]	98.15±8.19 [a]
	LSD	3.52	2.55
Female	Control	124.08±5.90 [b]	81.01±4.81 [b]
	Patients	145.18±11.89 [a]	94.20±7.15 [a]
	LSD	4.05	2.63

-The different letters refers a significant different at $P \leq 0.05$

For most high blood pressure patients the cause is unknown. This is classified as primary or essential hypertension. More than 90 % of high blood pressure patients have primary hypertension. Primary hypertension cannot be healed, but it can be managed with appropriate treatment (including improvements in lifestyle and medication). Genetic factors may play a significant role in primary hypertension growth. This form of hypertension tends to evolve gradually over several years (Olin and Pharm, 2018), while less than 10% of patients with high blood pressure have secondary HT. The most common cause of secondary HT is associated with kidney impairment such as chronic kidney disease (CKD) or renovascular disease. This form of high blood pressure tends to appear suddenly and often causes higher blood pressure than primary HT (Saseen, 2014; CDC, 2015). many previous epidemiological studies have revealed that older age, obesity,smoking, longer duration of hypertension, higher baseline blood pressure level, excessive salt consumption, diabetes , most of these factors perse contribute to blood pressure elevation (Cai *et al*., 2017)

B. Atrial natriuretic peptide (ANP)

The results showed a significant increase ($P \leq 0.05$) of atrial natriuretic peptide (ANP) level in hypertension patients (185.03±20.13) compared with control group(98.62±9.15) (table 3).

Table 3: level of atrial natriuretic peptide (ANP) in patients and control group

Parameters / Groups	ANP (Pg\ml) Mean± SD
Control	98.62±9.15 [b]
Patients	185.03±20.13 [a]
LSD	4.38

-The different letters refers a significant different at $P \leq 0.05$

Table (4) show atrial natriuretic peptide (ANP) of male and female hypertension patients increased significantly ($P \leq 0.05$) compared with the control group.

Table 4: level of atrial natriuretic peptide (ANP) of male and female patients with control group.

Parameters	Groups	Male (Mean± SD)	Female (Mean± SD)
ANP (pg\ml)	Control	100.86±13.01 [b]	97.89±12.32 [b]
	Patients	189.01±28.13 [a]	186.96±26.57 [a]
	L.S.D	8.15	8.86

-The different letters refers a significant different at $P \leq 0.05$

ANP is excreted from the heart. It binds in the kidney and blood vessels to its receptor, and promotes salt excretion, lowers blood volume, and relaxes the vessel. This endocrine system binds the heart and the kidney to maintain a perfect balance between electrolytes and body fluid (Zhou *et al*., 2009) . The possible reason for the elevated ANP levels is that the increased atrial stretch, resulting from volume overload, increases atrial peptides release rates. With the atrial problem of changes in left ventricular function induced by hypertension, this is likely to be a strong combined trigger for the release of auricular peptides (Nicholls *et al*., 1987). The elevated levels of ANP in hypertensive subjects can be associated with a tendency towards decreased kidney sodium excretion, either as a result of hereditary kidney abnormality or as a result of high blood pressure. This could cause sodium retention and thus increase compensatory mechanisms to excrete the sodium

excess. Most patients with essential hypertension, however, either have normal or low blood volume. (Tarazi, 1983).

C. Lipid profile level

The result indicated a significant increase (P≤0.05) of TC, TG, LDL and VLDL in patients of hypertension (213.53±19.20, 193.19±15.25, 137.40±8.63, 38.19±3.20) compared with the control group(158.96±13.58, 113.30±12.25, 77.78±4.37, 22.66±2.11), while there was a significant decrease in HDL of patients(37.94±6.15) compared with the control group(58.53±9.10) (table 5).

Table 5: Level of lipid profile (TC, TG, HDL, LDL and LDL) in hypertension patients and control group.

Groups / Parameters	Control Mean± SD	Patients Mean± SD	LSD
TC (mg\dl)	158.96±13.58 [b]	213.53±19.20 [a]	4.67
TG (mg\dl)	113.30±12.25 [b]	193.19±15.25 [a]	3.88
LDL (mg\dl)	77.78±4.37 [b]	137.40±8.63 [a]	1.92
HDL (mg\dl)	58.53±9.10 [a]	37.94±6.15 [b]	2.17
VLDL (mg\dl)	22.66±2.11 [b]	38.19±3.20 [a]	0.76

-The same letters refers non-significant different at P≤0.05

-The different letters refers a significant different at P≤0.05

The results indicated a significant increase (P≤0.05) in TC, TG , LDL and VLDL in patients of male and female patients compared with the control group, while the HDL showed a significant decrease in male and female patients compared with control group as (table 6).

Table 6: Lipid profile (TC, TG, HDL, LDL and LDL) of male and female hypertension patients with control group.

Parameters	Groups	Male Mean± SD	Female Mean± SD
TC(mg\dl)	Control	159.06±11.30 [b]	156.43±11.03 [b]
	Patients	205.19±13.33 [a]	203.93±11.23 [a]
	LSD	4.85	4.80
TG(mg\dl)	Control	108.23±11.09 [b]	103.20±10.55 [b]
	Patients	193.15±16.09 [a]	183.20±15.58 [a]
	LSD	5.14	5.74
LDL(mg\dl)	Control	82.31±4.81 [b]	79.59±3.95 [b]
	Patients	132.72±4.55 [a]	132.74±4.96 [a]
	LSD	1.40	1.27
HDL(mg\dl)	Control	55.10±6.33 [a]	56.20±6.03 [a]
	Patients	33.84±5.56 [b]	34.55±5.08 [b]
	LSD	2.21	2.41
VLDL(mg\dl)	Control	21.65±2.22 [b]	20.64±2.11 [b]
	Patients	38.63±3.22 [a]	36.64±3.12 [a]
	LSD	1.01	1.15

-The different letters refers a significant different at P≤0.05

The association between hyperlipidemia and hypertension has been shown in several previous studies. An excessive dietary intake of saturated fats, cholesterol and other calorie sources and subsequent lipid profile disruption leading to hypertriglyceridemia and hypercholesterolemia are related to obesity and hypertension . In a study Brown, 2000 confirmed the association between several factors including BMI, serum cholesterol, HDL and hypertension (Ghooshchi *et al*., 2014). Hypertension is considered to be associated with lipid metabolism alterations that give rise to serum lipid and lipoprotein levels abnormalities.It has also been documented that the presence of hyperlipidaemia makes the prognosis in hypertensive patients significantly worse (Osuji *et al*., 2012). High total blood cholesterol level increase the risk of many large vascular complications such as coronary artery disease (CHD) and stroke (Albucher *et al*., 2000). The study by Akuyam *et al*. (2009) In both hypertensive patients and normotensive controls, a positive and significant serum TC relationship with systolic and diastolic blood pressure was found in. High total cholesterol levels may be attributed to a number of causes including stress , increased intake of animal fat, lack of physical activity and genetic factors.

III. Conclusions

The present study indicated increase of atrial natriuretic peptide (ANP) in hypertension patients and show a positive association between hypertension and serum lipid profile included triglycerides, total cholesterol, LDL cholesterol levels and VLDL. High fats may be due to eating large amounts of nutritional fat, lack of physical activity and weight gain.

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