CASE STUDY

Assessment of Blood Lipids in People with Chronic Hypertensive

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ABSTRACT

High blood pressure remains one of the world's leading health problems. High blood pressure is already manageable, and early detection can prevent complications by using therapy or changing lifestyle to healthy habits. High blood pressure and dyslipidemia are two of the main hazard factors for cardiovascular disease. The current study was conducted on 60 male and female samples aged 35 to 70 years, which divided the samples into three groups each group including 20 samples. The first group are healthy people who don't have high blood pressure or chronic diseases (control group), the second group of patients with chronic hypertension without treatment but follow lifestyle modification, and the third group of patients with chronic hypertension with treatment the active substance hydrochlorothiazide and Losartan potassium, The regulated questionnaire information was recorded for all members of the sample and was measured the lipid profile test of triglycerides, cholesterol, lipoprotein (HDL, VLDL, LDL) in serum, atherogenic index plasma, body mass index (BMI), waistline. Levels of triglycerides and cholesterol were a non-significant increase in group of patients hypertensive without treatment and a significant increase in group of patients hypertensive with treatment than the control group. The results of lipoproteins showed non-significant differences than the control group while there was a significant increase in the VLDL level of group patients hypertensive with treatment than the control group. The results also indicated non-significant differences in the groups of atherogenic index plasma, body mass index and waistline measurement except for group of patients hypertensive without treatment that showed a significant increase in BMI than control group.

Keywords: Blood pressure, Lipid profile, Atherogenic index, Body mass index, Cardiovascular disease.

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INTRODUCTION

In low and middle-income nations, high blood pressure and dyslipidemia are substantial hazard factors of cardiovascular disease, accounting most than 80% of deaths and disability.[1] A number of hazard factors impacts cardiovascular disease development; high blood pressure is the leading reason of cardiovascular disease among all the hazard factors for the disease.[2] A poor diet is thought to be responsible for nearly half of all instances of hypertension in addition to increased salt consumption is linked to roughly 30% of instances, and low dietary potassium is linked to about 20% of cases low intake to fruit and vegetables, physical inactivity is also associated with about 20% of hypertension, while obesity is linked to approximately 30% of hypertensive.[3] Overweight and obese people, especially those with central obesity, have an increased risk of cardiovascular disease, according to research.[4]

A number of mechanisms have been hypothesized to relate a high BMI to cardiovascular disease. A high BMI raises blood pressure and serum cholesterol.[5] Body mass index BMI is a comprehensive indication of acquired lifestyle outcomes linked to hypertension incidence.[6] Obesity is concomitant with a risk of high blood pressure and cardiovascular disease mortality, according to a review of meta-analytic studies. General obesity is measured by BMI, central and abdominal obesity is measured by anthropometric indictors like waistline or waist-to-hip ratio.[7] Although lifestyle changes are crucial in high blood pressure management, most high blood pressure people require two anti-hypertension medications (combination therapy) to lower their blood pressure and keep it below tolerable values.[8] At least five medication types have been shown to be useful in treating hypertension and reducing cardiovascular events.[9] The anti-hypertension and lipid-lowering treatment to prevent heart attack trial found that thiazide diuretics are as effective as or more effective than other anti-hypertension medications in lowering cardiovascular actions.[10] Multiple negative effects have been linked to thiazide diuretics. The diuretic dose causes the majority of these adverse effects; the most prevalent metabolic consequences are hypokalemia and hyponatremia, followed by hyperuricemia, hypomagnesemia, hyperlipidemia, and elevated glucose levels.[11] Therefore, the

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main objective of this study was to mark serum lipid levels and BMI as hazard factors for cardiovascular disease in chronic hypertensive patients. The study also aimed to mark the independent associations of antihypertensive treatment on serum lipid levels and blood pressure.

Samples Collection

Current study conducted on 60 samples aged 35 to 70 years divided the samples into three groups each group of 20 samples. The first group are healthy people with no hypertensive and no chronic disease (control group), second group is patients with chronic hypertension do not have any treatment but follow lifestyle modification; third group of patients with chronic hypertension taking a drug (Angizaar H 50 mg), recorded the organized questionnaire information for all sample members and included age, gender, height and weight (to extract mass index measurement) waist measurement and treatment use period, as well as blood pressure measurement and recording pressure readings per sample the sampling of blood was carried out in the morning for a fasting period ranging from 8 to 12 hours by single-use injections and then emptied with clotting gel tubes. The samples were left at 37°C until they were clotted and then separated with the centrifuge at 3,500 rpm, to extract serum and then divided in special small-sized tubes stored to freeze samples were placed in plastic cans and each case was indicated with the sample symbol, and samples were then kept at freezing until the study's lipid profile was chemically tested (triglycerides, cholesterol, HDL, LDL, VLDL). Both triglycerides, total cholesterol, and HDL-cholesterol were determined by enzymatic method.[12-14] Friedewald formula was used in the calculation of LDL-Cholesterol and (VLDL).[15,16] BMI was calculated as (weight in kg/height in m^2).[17] Atherogenic index plasma was also calculated as LDL/HDL. This ratio is an indicator of whether or not heart disease occurs. If it exceeds number five, this is an indicator of the occurrence of the disease, the increase in LDL and the condition is abnormal. If the ratio is lower than number three , there an increase in HDL, which means that the disease does not occur and the condition is normal.[18]

Statistical Comparison

The average and standard deviation were calculated among groups using SPSS, and Duncan 's Test was adopted to compare the results between the groups.

RESULTS AND DISCUSSION

Data Description

The statistical analysis of demographic information collected was carried out with the structured questionnaire, mediated by the Duncan test as a percentage and a graph of circular sectors and histograms, which includes age, gender, period of treatment intake and illness as well as blood pressure readings.

Age

Table 1 shows the dispensation of the sample members in this study according to age, where we note that the highest percentage (40%) are from the second category 40 to 50 years old in the control group, and the lowest percentage (10%) are from the last category 60 to 70 years old in the control group, and the percentage was approximately equal in the patients with treatment. To clarify the age dispensation of the study sample using the graph in Figure 1.

Gender

Table 2 shows the dispensation of the sample members pursuant to gender, it was noted that the percentages are close between males and females in all study groups. The graph was utilized to clarify the percentage of males and females in the study samples in all groups, as shown in Figure 2.

Period of treatment and followers lifestyle of hypertensive patients

Table 3 and the graph of circular sectors as shown in Figure 3, shows the dispensation of the samples in the study groups pursuant to the period of treatment and followers lifestyle of hypertensive patients.

Bood pressure

Table 4 shows the dispensation of the sample members pursuant to high blood pressure.

Determination of Lipid Profile for the Groups under Study

Table 5 shows the mean ± standard deviation of lipid profile levels represented by biochemical measurements of HDL, LDL, VLDL, and atherogenic index, BMI, waistline scale of total groups under consideration, healthy group, patients with chronic hypertensive do not have any treatment but follow lifestyle modification and patients with chronic hypertensive taking a drug (Angizaar H 50 mg).

Determination of Triglycerides

Current study results showed that the levels of triglycerides moral differences between patients hypertensive without treatment group and patients hypertensive with treatment group compared to control group for healthy people at probability level $p \leq 0.05$, was the rate ± standard deviation

Table 1: Dispensation of the samples pursuant to age

Age category (years)	*Control*	*Patients without treatment*	*Patients with treatment*
	Percentage%	*Percentage%*	*Percentage%*
35–40	20	20	24
40–50	40	30	24
50–60	25	30	28
60–70	10	20	24

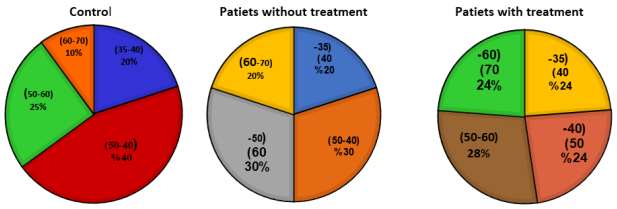

Figure 1: Dispensation of the study samples pursuant to age

Table 2: Dispensation of the sample members by gender

Groups	*Gender*	*Percentage%*
Control	Males	50
	Females	50
Patients without treatment	Males	55
	Females	45
Patients with treatment	Males	52
	Females	48

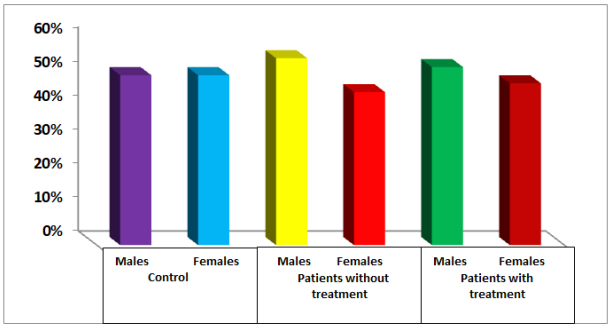

Figure 2: Percentage of males and females in the study sample

Table 3: Dispensation of the sample pursuant to the period of treatment and followers lifestyle of hypertensive patients

Period (years)	*Patients without treatment(lifestyle)*	*Patients with treatment*
	Percentage%	*Percentage%*
1-3	10	33
4-5	40	29
5-7	30	14
7-10	20	24

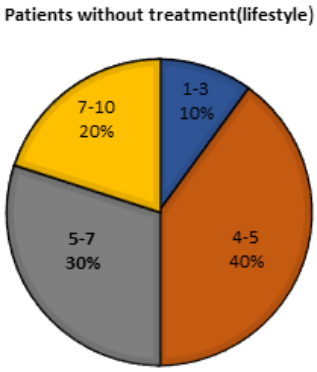

Figure 3: Dispensation of the study sample pursuant to the Period of treatment and followers lifestyle of hypertensive patients

of the mentioned groups was extracted as shown in Table 5 and Figure 4.

The mean ± S.D. of level triglycerides of the control group and patients hypertensive without treatment group and patients hypertensive with treatment group was (123.20 ± 69.991mg/dl), (141.35 ± 85.231 and 179.00 ± 82.132 mg/dl respectively. Current study results included a non-significant increase in the concentration of triglycerides in the serum of patients hypertensive without the treatment group and a significant

Table 4: Dispensation of the sample members by high blood pressure

Control		*Patients without treatment*		*Patients with treatment*	
Pressure	*Percentage %*	*Pressure*	*Percentage %*	*Pressure*	*Percentage %*
15/7	5	16/9	5	14/7	5
14/9	5	15/10	5	13/9	14
13/9	10	13/9	5	13/8	10
13/8	5	13/8	15	13/7	5
12/9	5	13/7	5	12/8	38
12/8	30	13/6	5	12/7	14
12/7	20	12/8	45	12/6	5
11/7	5	12/7	10	11/9	5
11/6	5	11/6	5	10/6	5
10/8	5				
10/7	5				

increase in patients hypertensive with treatment group than control group of healthy people. A study mediated by Kawamoto[19] on a community sample of Japanese individuals found triglycerides were positively linked to systolic and diastolic blood pressure. Current findings are consistent with a study by Huldani *et al.*[20] that concluded triglycerides had an effect on high blood pressure. According to other studies conducted by Huldani *et al.*, Achmad *et al.*[21,22] there is a substantial relationship between the level of blood triglycerides and systolic and diastolic blood pressure**.** Because triglycerides are a component of lipoproteins and therefore at increased levels of lipoproteins in the blood will have an effect on the levels of triglycerides in the blood, these studies reported blood viscosity is affected by triglycerides, the higher the levels of the blood triglycerides, the greater the blood viscosity, thus making blood flow more difficult this makes the heart work harder to pump blood leading boost blood pressure.

Determination of Cholesterol

The serum cholesterol level was measured for patients of the hypertensive without treatment group and patients hypertensive with treatment group than the control group for healthy people at the probability lewvel $p \leq 0.05$. The mean ± standard deviation of the mentioned groups was extracted as shown in Table 5 and Figure 5.

The mean ± S.D. of level cholesterol of control group and patients hypertensive without treatment group and patients hypertensive with the treatment group was 168.85 ± 23.946, 178.70 ± 36.015 and 202.81 ± 38.024 mg/dl, respectively. The results of the current study alluded to a non-significant increase in the concentration of cholesterol in the serum of patients hypertensive without the treatment group and a significant increase in serum of patients hypertensive with the treatment group than control group of healthy people. The current study agrees with the Znyk[23] study which pointed to the association of blood pressure and cholesterol level with lifestyle modification and which has a preventive role and is one of the easiest ways to reduce cholesterol level and hypertension and thereby

Table 5: HDL, LDL, VLDL, atherogenic index, BMI and waistline scale in the serum of total groups under consideration

Lipid profile	*Mean ± S.D*		
	Control	*Patients without treatment*	*Patients with treatment*
Triglycerides (mg/dl)	123.20 ± 69.991 b	141.35 ± 85.231 ab	179.00 ± 82.132 a
Cholesterol (mg/dl)	168.85 ± 23.946 b	178.70 ± 36.015 b	202.81 ± 38.024 a
dl) /mg)HDL	52.80 ± 16.315 a	44.90 ± 12.320 a	53.57 ± 18.696 a
LDL (mg/dl)	106.49 ± 24.318 a	105.70 ± 29.519 a	113.38 ± 34.707 a
VLDL (mg/dl)	23.61 ± 10.743 b	28.25 ± 17.477 ab	36.14 ± 16.502 a
Atheriogenic index	2.0685 ± 1.073 a	2.5933 ± 1.210 a	2.5546 ± 1.574 a
BMI	28.835 ± 4.109 b	34.9700 ± 9.372 a	31.1810 ± 6.363 ab
Waistline Scale	98.75 ± 11.026 a	102.45 ± 12.878 a	104.19 ± 19.167 a

*Different letters mean moral differences at p ≤0.05. ,*Similar letters mean no moral difference at p >0.05.

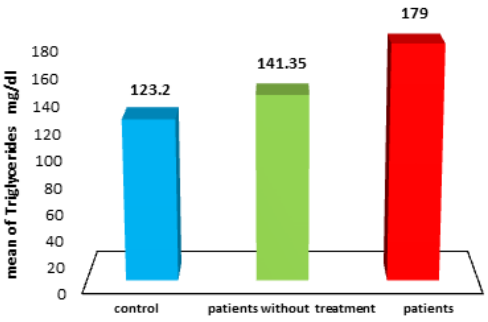

Figure 4: Mean of triglycerides (mg/dl) in the serum of the groups under study

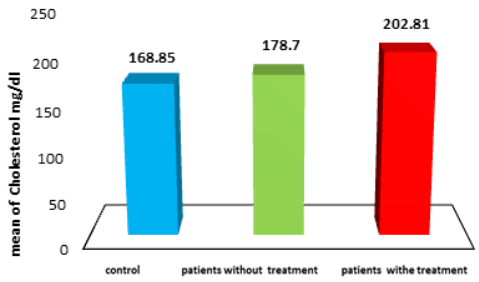

Figure 5: mean of cholesterol (mg/dl) in the serum of the groups under study

reduce risk of chronic illness. A subsequent study published by Sakurai[24] found similar results. It analyzed data from 4680 people aged 40 to 59 years from 17 different regions in Japan, China, the UK, and the US. Blood pressure, cholesterol levels, and diet were monitored 24 hours a day. The results showed that cholesterol was directly associated with blood pressure for all participants. As a result, high blood cholesterol may predict the future presence of high blood pressure. That's what Ruben and others.[25] They analyzed data from 3110 men who had not been diagnosed with hypertension or cardiovascular disease at the start and followed them for about 14 years just over 1000 of them developed hypertensive by the end of the study, the same researchers did a similar test on women with a follow-up of about 11 years and found comparable results healthy women with higher levels of cholesterol were more likely to develop hypertensive down the road than those with lower levels of cholesterol. Hypercholesterolemia promotes atherosclerosis or cholesterol deposits in the arterial lumen, which results in artery narrowing, hardening and stiffness, and increased peripheral vascular resistance and pressure.[26] Fibrous tissue formation calcination and alters in the endothelial artery walls can cause the arterial wall's thickening, and arteriosclerosis results from a buildup of plaque in the arteries. This suggests that endothelial dysfunction occurs early in the evolution of arteriosclerosis, resulting in raised blood pressure, while hypercholesterolemia takes a long time to cause alterations in arterial endothelium, culminating at elevated hypertension.[27]

Determination of Lipoprotein

The concentrations of HDL, LDL, VLDL were measured in the serum for patients of the hypertensive without treatment group and patients hypertensive with the treatment group compared to control group of healthy people at the probability level $p \leq 0.05$. The mean ± standard deviation of the mentioned groups was extracted as shown in Table 5 and Figures 6-8.

The mean ± SD of level HDL of the control group and patients hypertensive without treatment group and patients hypertensive with treatment group was 52.80 ± 16.315, 44.90 ± 12.320, 53.57 ± 18.696 mg/dl, respectively. The results of the current study indicated no moral difference at probability level p≤0.05 for groups under study. Results indicate non-significant decrease in concentration of HDL in serum of patients hypertensive without treatment group and non-significant increase in serum of patients hypertensive with treatment group than control group of healthy people.

The mean ± S.D. of level LDL of the control group and patients hypertensive without treatment group and patients hypertensive with treatment group was 106.49 ± 24.318, 105.70 ± 29.519, 113.38 ± 34.707 mg/dl, respectively. The results of the

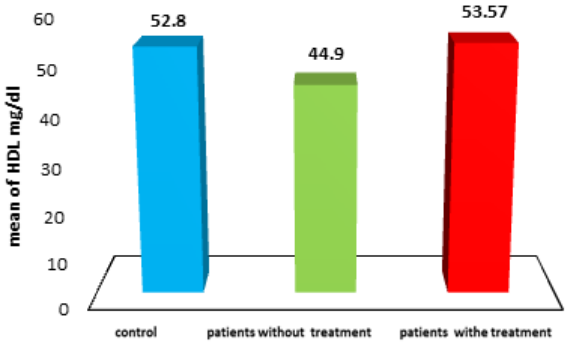

Figure 6: Mean of HDL (mg/dl) in the serum of the groups under study

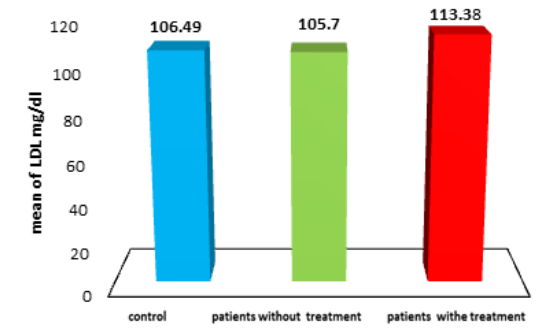

Figure 7: Mean of LDL (mg/dl) in the serum of the groups under study

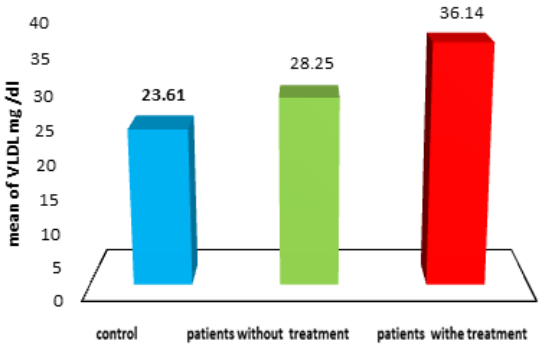

Figure 8: Mean of VLDL (mg/dl) in the serum of the groups under study

current study indicated no moral difference at probability level $p \leq 0.05$ of groups under study. Results non-significant decrease in concentration of LDL in serum of patients hypertensive without treatment group and non-significant increase in serum of patients hypertensive with treatment group than control group of healthy people**.**

The mean ± SD of level VLDL of the control group and patients hypertensive without treatment group and patients hypertensive with treatment group was 23.61 ± 10.743, 28.25 ± 17.477, 36.14 ± 16.502 mg/dl, respectively.

Results indicated a non-significant increase in the concentration of VLDL in the serum of patients hypertensive without the treatment group and a significant increase in the concentration of VLDL in serum of patients hypertensive with the treatment group than a control group of healthy people. The results correspond with a study by Onwubuya[28] that found Patients with hypertension had higher lipid and lipoprotein levels than controls, and values became more important as the intensity of hypertension increased and the difference was statistically significant for total cholesterol, LDL-C, and VLDL-C. Li *et al.*[29] also showed that high blood lipoprotein cholesterol was significantly related with central systolic hypertension in the Chinese population, apart from other fats. The most prevalent lipoprotein identified in hypertension patients was VLDL-C, which increased total cholesterol. In other investigations, an increase of total cholesterol is associated with an increase of LDL-C and VLDL-C levels.[30] This difference could explain that VLDL-C is formed mainly in the liver, and is made up of triglyceride derived from many fatty acids in the circulation and from dietary carbohydrates.[31,32] According to Bruce *et al.*[33] Excess VLDL-C can also be processed through their triglyceride exchange for cholesterol ester in LDL-C and HDL-C via the action of a cholesterol ester transfer protein, The resulting triglyceride-rich HDL-C particle serves as a substrate for hepatic lipase, which shrinking the particle and releasing apolipoprotein A1, which is excreted through the kidney. The endothelium-bound lipoprotein lipase hydrolyzes the triglyceride-rich LDL-C particle and produces tiny dense LDL-C particles, which were not quantified. This results in low HDL-C and LDL-C, which could explain the findings in this study.

Determination of Atherogenic Index Plasma

Atherogenic index plasma was measured for patients of the hypertensive without treatment group and patients hypertensive with treatment group compared to control group for healthy people at the probability level $p \leq 0.05$. Mean ± standard deviation of the mentioned groups was extracted as shown in Table 5 and Figure 9.

The mean ± SD of atherogenic index of the control group and patients hypertensive without treatment group and patients hypertensive with treatment group was 2.0685 ± 1.073, 2.5933 ± 1.210, (2.5546 ± 1.574), respectively. The results of the current study indicated no moral difference at probability level $p \leq 0.05$ for the groups under study. Results showed a non-significant increase in the atherogenic index of patients hypertensive without treatment group and patients hypertensive with treatment group compared to a control group of non-hypertensive people. Atherogenic index (AI) (LDL-C/HDL-C) can be a powerful marker for predicting atherosclerosis and coronary heart disease risk. In the Kazemi trial, 5207 patients were recruited and the atherogenic and coronary risk indexes were enrolled for each of them. Indicated in his results, age, body mass index, gender, and Atherogenic.[34] A study of 150 women living in Tabriz, Iran, evaluated atherosclerosis, fat levels and blood pressure indicators and revealed that most women studied had a high risk of cardiovascular disease depending on atherosclerosis indicators.[35]

Determination of Body Mass Index

Body mass index was measured for patients of the hypertensive without treatment group and patients hypertensive with treatment group compared to control group for healthy people at the probability level $p \leq 0.05$. The mean ± standard deviation of the mentioned groups was extracted as shown in Table 5 and Figure 10.

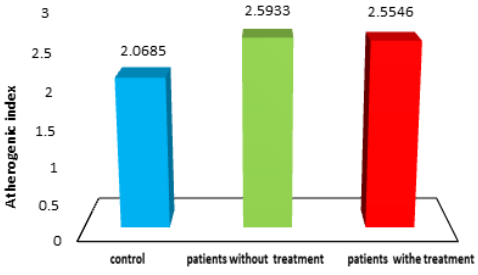

Figure 9: Mean of atherogenic index plasma of the groups under study

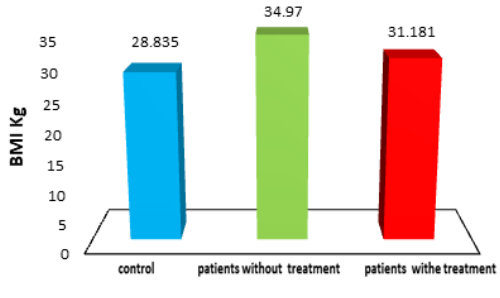

Figure 10: Mean of body mass index (Kg) of the groups under study

The mean ± SD of body mass index of the control group and patients hypertensive without treatment group and patients hypertensive with treatment group was 28.835 ± 4.109, 34.9700 ± 9.372, 31.1810 ± 6.363, respectively. The results of the current study indicated moral difference at the level of probability $p \leq 0.05$ for groups under study. Results shown a significant increase in the body mass index of patients hypertensive without treatment group and a non-significant increase of patients hypertensive with the treatment group in control group of healthy people. The results are in agreement with Landi's *et al*. study,[36] which demonstrated a gradient of rising blood pressure with increasing body mass index that systolic and diastolic blood pressure values are linearly correlated with body mass index and pointed that body mass index may have a direct effect on blood pressure irrespective of other clinical risk factors. Overweight status, which reflects a greater body fat mass, was inferred an independent risk factor of hypertension, which was consistent with previous researches[37-41] that found a link between high body lipid levels and hypertension. However, The mechanism behind the link between visceral fat and hypertension is uncertain. Inflammatory processes are discovered that play a major part in the mechanisms underlying hypertension development.[42] Fat cells are distinguished by their susceptibility to lipolysis and their ability to release large amounts of inflammatory cytokines. This inflammatory reaction contributes to high blood pressure and organ damage. Furthermore, increasing adipose tissue probably causes a reduction in the generation and usage of nitric oxide, which is vital for controlling vascular tone and suppressing vascular smooth muscle cell growth. Endothelial dysfunction and arterial hypertension hase been linked to a decreased nitric oxide effect.[43]

Determination of Waistline

Waistline was measured for patients of the hypertensive without treatment group and patients hypertensive with treatment group compared to the control group for healthy people at the probability level $p \leq 0.05$. The mean ± standard deviation of the mentioned groups was extracted as shown in Table 5 and Figure 11.

The mean ± SD of the waistline of the control group and patients hypertensive without treatment group and patients hypertensive with treatment group was 98.75 ± 11.026, 102.45 ± 12.878, 104.19 ± 19.167, respectively. The results of the current study indicated no moral difference at the probability

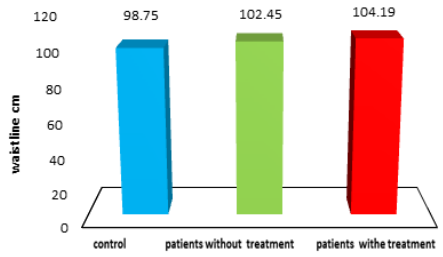

Figure 11: Mean of waistline (cm) of the groups under study

level $p \leq 0.05$ for the groups under study. Results shown non-significant increase in the waistline of patients hypertensive without treatment group and patients hypertensive with treatment group compared to control group of healthy people. Because waist circumference corresponds with abdominal fat mass and is correlated with cardio-metabolic illness risk, it is employed as a proxy marker to abdominal fat mass[44] based to a review of meta-analytic studies. General obesity was measured by BMI, central and abdominal obesity measured by anthropometric indictors such as waist circumference or waist-to-hip ratio, and obesity is associated with the risk of hypertension and cardiovascular disease mortality.[7] Yu-findings Sun also revealed that waist circumference was a helpful biomarker for assessing the risk of hypertension. When assessing the cardiometabolic risk associated with fat distribution, his findings justified the utilization of waist circumference independent of BMI.[45] Another study found that BMI, waistline, and composite index were all linked with incident hypertension in a Chinese community-based sample.[46] The study concluded, Kumar, that the waist to height ratio was a better indicator than BMI marked systolic hypertensive.[47]

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