How to Cite:
Alwaan, A. A., Albyati , M. A., & Shaker, M. K. . (2022). Study of aldosterone and malondialdehyde in obesity related hypertension without A diagnosis of chronic disease. *International Journal of Health Sciences*, *6*(S7), 5603-5616. https://doi.org/10.53730/ijhs.v6nS7.13287

Study of aldosterone and malondialdehyde in obesity related hypertension without A diagnosis of chronic disease

Ahlam A. Alwaan
Al-Nahrain University/College of Medicine/Department of Chemistry and Biochemistry / Iraq Mobile: 07733148098
Email: ahlamaltay@gmail.com

Mohammed A. Albyati ,
Al-Nahrain University /College of Medicine / Department of Chemistry and Biochemistry / Iraq

Mahmoud K. Shaker
Al-Nahrain University /College of Medicine / Department of Medicine / Iraq

***Abstract*---**Obese subjects often have hypertension and related cardiovascular and renal diseases. In obese subjects impaired renal-pressure natriuresis causes sodium retention, leading to the development of salt sensitive hypertension. Obesity is due to fluid retention and high fat in cells, so fat cells will active any factors to stimulate the secretion of aldosterone, and there will be salt sensitivity to blood pressure only. But it is not necessarily linked to cardiovascular and renal disease. The oxidative stress which rises with the presence of heart and kidney diseases associated with high blood pressure, so there will be increase in oxidative stress because people do not suffer from chronic diseases and obesity is considered fluid retention and fat accumulation in the body.

***Keywords*---**aldosterone; blood pressure; hypertension; oxidative stress ;obesity related hypertension.

Introduction

Over the past ten years, there has been a severe increase in the prevalence of obesity and the related cardiovascular, metabolic, and renal problems. More over 1.9 billion adults were overweight in 2014, of whom more than 600 million were obese, or 39% and 13% of adults, respectively, according to the World Health Organization's Obesity and Overweight Fact Sheet. Additionally, medical expenses for obese people were almost 30% greater than for their contemporaries who were

of normal weight. It indicates that obesity places a significant economic burden on many nations. The amount of extra weight correlates directly with a large rise in the risk of hypertension. Additionally, type 2 diabetes, stroke, dyslipidemia, and cardiovascular disease (CVD) are all linked to obesity. Obesity is closely tied to a number of metabolic and cardiovascular diseases and is associated with a higher risk of death. Contrarily, mounting data recently revealed that obese people had higher plasma and urinary aldosterone concentrations and that aldosterone plays a role in cardiovascular, renal, and metabolic disorders. Aldosterone has traditionally been thought to be crucial to the control of electrolyte and fluid volume as well as the preservation of blood pressure (BP) equilibrium. However, cardiovascular and renal damage are significantly influenced by the aldosterone/mineralocorticoid receptor (MR) system. These non-classical actions of aldosterone and the involvement of MR activation in CVD were proven by 3 large-scale randomized clinical trials, the Randomized Aldactone Evaluation Study (RALES), the Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study (EPHESUS), and the Eplerenone in Mild Patients Hospitalization and Survival Study in Heart Failure (EMPHASIS-HF). Additionally, recent clinical trials such as the Mineralocorticoid Receptor Antagonist Tolerability Study-Diabetic Nephropathy (ARTS-DN) and the Eplerenone Combination versus Conventional Agents to Lower Blood Pressure on Urinary Antialbuminuric Treatment Effect (EVALUATE) study showed that aldosterone/MR activation plays a pathogenic role in chronic kidney disease (CKD). We emphasize the involvement of the aldosterone/MR system in obesity-related hypertension in this review (1).

Obesity Related Hypertension

Weight, particularly instinctive stoutness, is firmly connected with hypertension. The connection between human weight and hypertension was first depicted by Vague in 1956.20 He separated corpulent patients into "android" type (chest area corpulence) and "gynoid" type (lower body stoutness) and announced that cardiovascular and metabolic confusions of stoutness were more pervasive in the previous. Be that as it may, such complications don't foster in every single hefty individual, and some ordinary weight grown-ups display metabolic disorder and its comorbidities. This is on the grounds that strange muscle to fat ratio distribution, like overabundance instinctive fat tissue, is a more significant element than weight file for grimness. Instinctive adiposity purportedly assumes a significant part in the happen rence of hypertension, diabetes mellitus, hyperlipidemia and atherosclerosis in corpulent people and in creature models.(1)

Oxidative Stress in Obesity Related Hypertension:

Numerous constant sicknesses are described by over the top oxidative pressure and aggravation. The new heightening of ongoing circumstances like heftiness, insulin obstruction, diabetes, hypertension, and other related cardio metabolic entanglements in all times of the populace including kids, immaturities, and grown-ups represents an extraordinary test to medical services systems.(2) Systemic oxidative pressure was decidedly corresponded with fat mass in people and rodents. Mitochondrial oxidative pressure markers, like protein carbonyls, lipid peroxidation items, and malondialdehyde (MDA), as well as ROS creation

were expanded in fat tissues of hefty subjects. In db/db mice, ROS creation was up managed in adipocytes, alongside lipid peroxidation item 4-hydroxynonenal (4-HNE) accumulation.(3) Lipid peroxidation (LPO) is a response that happened between free revolutionaries and polyunsaturated unsaturated fat on cell film structure, LPO is the cycle which free extremists take the temperamental particles from the lipids that will make progressive oxidations and leads lipid precariousness, cell harm and the development of malondialdehyde (MDA)(4)

Aldosterone in Obesity Related Hypertension:

A few studies suggest that aldosterone overproduction has a role in the etiology of weight-related hypertension and reveal that it is frequently prevalent in stoutness. showed that weight loss is accompanied by drops in aldosterone, regardless of salt intake, and this affects the drop in (BP) blood pressure in obese patients. (1) Aldosterone is controlled in the adrenal zona glomerulosa and connects to specific mineralocorticoid receptors found in the target epithelial cells' cytoplasm. The subsequent steroid receptor complex's relocation to the cell's nucleus improves the expression and understanding of specific proteins that regulate electrolyte and liquid balance. However, aldosterone has also been shown to have non-epithelial and rapid non-genomic activities that document for several aldosterone functions that contribute to pulse homeostasis. These recall significant endothelial cell and cardiovascular tissue functions. (5)

Definition of Obesity:

Overweight and obesity occur when excess fat accumulation (regionally, globally, or both) increases risk to health. It is the point at which health risk is increased that is most important because, as covered below, body weights and fat distributions that lead to expression of co-morbid diseases occur at different thresholds depending on the population.(2)

Methodology:

A case control study was employed. In This study was conducted from February 2022 to April 2021, where this study included 120 (60 female & 60 male) people and they were distributed as follows

The study subjects were divided into two main groups and each group subdivides into two subgroups.

Group I: including 60 subjects subdivides into

a. included 30 healthy subjects with no obese and no hypertension in the age group (above 18 year), who were used as a control Group. The control group samples will be collected from healthy people,

b. included 30 subjects with no obese with hypertension (dose not receive treatment) in the age group (above 18 year) who were used as a positive control
Group II : including 60 subjects subdivides into:

a.30 subjects with obese and hypertension) Patients dose not receive treatment) in the age group (above 18 year).

b.30 subjects with obese and no hypertension in the age group (above 18 year).

Exclusion Criteria

The requirements for exclusion criteria were as follow :

1. Patients with adrenal tumor or cancer.
2. Patients with any type of cancer or tumor will be excluded.
3. Patients with cardiac surgery.
4. People suffering from chronic diseases.

Inclusion Criteria:

1. People diagnosed with high pressure.
2. Do not take any medication for high pressure.
3. They do not suffer from kidney or heat disease.
4. Obese BMI over 30 kg/m^2 .

Collection of Blood Samples:

1.ten milliliters of venous blood was taken from each patient in the morning, provided they fast .through puncture the vein with injections designated to draw blood and dispose of it after use.

2.The blood was placed in special tubes called gel tubes, which have a yellow color.

3.The blood was left for (10-20) minutes at room temperature in order for the blood to start clotting .

4.The tube was placed in a centrifuge under a speed of 2000-3000 RPM for 20 minutes to separate the blood and obtain the blood serum.

5.The separated serum was divided into five parts and stored at a temperature of (-20 degrees Celsius) until the necessary tests are performed .

Calculation of BMI:

Body mass index (BMI) was obtained from measuring weight in kilograms & height in meters by using suitable scales and applied the following equation-:

BMI (Kg/m2) = Weight / Height

Waist Circumference Measurement

After the participant was kept in a standing position, a trained exam- iner would locate the belly button and then place a soft measuring tape around the waist at the navel level. Next, the participant would be asked to take a normal breath and hold a breath at the end of exhaling. Then, waist circumference was measured after the participant exhaled one normal breath. A detailed description of waist circumference measurement was provided in the CHARLS Biomarker Questionnaire Protocol(http://charls.pku.edu.cn/pages/data/2011-charls-wave1/zh-cn. html(3).

Methods:

Determination of Human Aldosterone concentration:

Assay Principle:

This kit is an Enzyme-Linked Immunosorbent Assay (ELISA). The plate has been pre-coated with human ALD antibody. ALD present in the sample is added and binds to antibodies coated on the wells. And then biotinylated human ALD Antibody is added and binds to ALD in the sample. Then Streptavidin-HRP is added and binds to the Biotinylated ALD antibody. After incubation unbound Streptavidin-HRP is washed away during a washing step. Substrate solution is then added and color develops in proportion to the amount of human ALD. The reaction is terminated by addition of acidic stop solution and absorbance is measured at 450 nm.

Assay Procedure

1. Prepare all reagents, standard solutions and samples as instructed. Bring all reagents to room temperature before use. The assay is performed at room temperature.

2. Determine the number of strips required for the assay. Insert the strips in the frames for use. The unused strips should be stored at 2-8°C.

3. Add 50µl standard to standard well. Note: Do not add antibody to standard well because the standard solution contains biotinylated antibody.

4. Add 40µl sample to sample wells and then add 10µl anti-ALD antibody to sample wells, then add 50µl streptavidin-HRP to sample wells and standard wells (Not blank control well). Mix well. Cover the plate with a sealer. Incubate 60 minutes at 37°C.

5. Remove the sealer and wash the plate 5 times with wash buffer. Soak wells with at least 0.35 ml wash buffer for 30 seconds to 1 minute for each wash. For automated washing, aspirate all wells and wash 5 times with wash buffer, overfilling wells with wash buffer. Blot the plate onto paper towels or

other absorbent material.

6. Add 50µl substrate solution A to each well and then add 50µl substrate solution B to each well. Incubate plate covered with a new sealer for 10 minutes at 37°C in the dark.

7. Add 50µl Stop Solution to each well, the blue color will change into yellow immediately. 8. Determine the optical density (OD value) of each well immediately using a micro plate reader set to 450 nm within 10 minutes after adding the stop solution.

Result and discussion:

Results

General Characteristics of the Population

From February 2022 to April 2022, a total of 120 patients were diagnosed with obesity-related hypertension without hypertension, without obesity with hypertension, and without obesity All subjects in the study were recruited from Al-Imamain Al-kadmayn Hospital in Baghdad Medical City, Iraq, and the project was approved by the Institutional Ethics Committee.

Analysis of Anthropometric and Physiological Variables

Anthropometric and physiological variables of hypertensive- and normotensive subjects are presented in Table 2. As expected, hypertensive patients were younger than normotensive subjects. Mean ages of normotensive - and hypertensive subjects were observed as 32.65 ± 10.33). and 39.10 ± 10.34). years, respectively. The difference between the mean ages of two groups was statistically significant t(118) = -3.42, p < .001. The mean values of body mass index (BMI) noticed in hypertensive- and normotensive subjects were 31.061± 7.5081 and 30.779± 5.6947 kg/m2, respectively and the difference in the mean values of BMI of both the groups was statistically not significant t(-0.231),p =.818 hypertensive subjects had higher Waist circumference 106.53 ± 13.32) than normotensive subjects 101.73 ± 16.56). but differences in Waist circumference *t*(-1.749), p= 0.083 were statistically non-significant

Higher mean value of systolic blood pressure (SBP) was observed in hypertensive subjects (147.22 ± 8.63) mmHg) as compared to normotensive (110.83 ± 9.26) mmHg) and the difference was statistically significant (p<0.001). Similarly, higher mean value of diastolic blood pressure (DBP) was noticed in hypertensive subjects (100.87 ± 5.60 mmHg) as compared to normotensive (70.75 ± 9.20 mmHg) the difference between the two groups was also statistically significant (p<0.001). Normotensive subjects took an edge over hypertensive subjects in the mean value of pulse pressure (PP). Its value was observed as 46.3577±8.4704 mmHg in hypertensive- and 40.0833±0.6455 mmHg in normotensive subjects. The difference in their mean values was statistically significant (p<0.001).

Similarly, it was observed that normotensive subjects had higher mean values of HbA1c 5.439± 0.3670% as compared to their hypertensive counterparts 5.173± 0.5063%, and the difference was statistically significant. (P=0.01)

It was observed that normotensive subjects had lower mean values of random blood glucose (RBG) (90.613± 9.3086 mm/dl) as compared to their hypertensive counterparts (91.290± 10.9466 mm/dl), although the difference was statistically non-significant. (p= 0.716)

Table 0-1 Anthropometric and Physiological variables of hypertensive and normotensive individuals

	HT	Normotensive	t	P*
	Mean± SD	Mean± SD		
Age	39.100± 10.3362	32.650± 10.3331	-3.418	< .001
Waist	106.533± 13.3245	101.733± 16.5620	-1.749	.083
BMI	31.061± 7.5081	30.779± 5.6947	-0.231	.818
DBP	100.867± 5.5980	70.750± 9.1977	-21.666	< .001
SBP	147.224± 8.6316	110.833± 9.2593	-22.268	< .001
PP	46.3577±8.4704	40.0833±0.6455	-5.721	< .001
FBS	91.290± 10.9466	90.613± 9.3086	-0.365	.716
HbA1c	5.173± 0.5063	5.439± 0.3670	3.294	.001

*Results for Testing the Mean Differences by Bp using *t*-Tests

Comparison of the Aldosterone, Renin, MDA and serum electrolytes by Bp

For Normotensive, Aldosterone had an average of 519.28 ± 177.59. For Hypertensive, Aldosterone had an average of 541.63 ± 208.18. For Normotensive, the Renine had an average of 140.53 ± 87.89. For Hypertensive, Renine had an average of 144.94 ± 104.88. For Normotensive, MDA had an average of 1.17 ± 0.91. For Hypertensive, MDA had an average of 1.32 ± 1.09. For Normotensive, Potassium had an average of 3.63 ± 0.92. For Hypertensive, Potassium had an average of 4.35 ± 0.81. For Normotensive, Sodium had an average of 144.40 ±41.98. For Hypertensive, Sodium had an average of 165.90 ± 32.65. The summary statistics can be found in Table 3-2.

Table 0-2 Results for Testing the Mean Differences by Bp using t-Tests

	Variable	**M± SD**	**n**	**t**	**p**
Aldosterone	Normotensive	519.277± 177.589	60	-0.633	0.528
	Hypertensive	541.631± 208.180	60		
Renine	Normotensive	140.531± 87.885	60	-0.250	0.803
	Hypertensive	144.944± 104.879	60		
MDA	Normotensive	1.168± 0.911	60	-0.823	0.412
	Hypertensive	1.319± 1.090	60		
Potassium	Normotensive	3.630± 0.918	60	-4.561	< .001
	Hypertensive	4.349± 0.805	60		

Sodium	Normotensive	144.405± 41.976	60	-3.131	0.002
	Hypertensive	165.898± 32.645	60		

Biochemical analysis

For Normotensive, Cholesterol had an average of 144.57± 21.50. which was statistically lower significantly than that for Hypertensive, 165.04± 20.29, P <0.001. triglyceride mean for Normotensive, 110.08± 25.00. was lower than that for Hypertensive, 114.78± 34.83but it was statistically not significant P= .397. HDL had an average of 45.84± 7.66 For Normotensive which was lower than that for Hypertensive, 47.05 ± 8.99 the difference was statistically not significant p= 0.430. For Normotensive, LDL had an average of 76.70± 21.30. which was significantly lower than that for Hypertensive, 95.01 ± 17.95, p <0.001. For Normotensive, VLDL had an average of 22.01 ± 5.00. which was comparatively similar Hypertensive VLDL average of 22.77± 7.02, P= 0.498.

For Normotensive, Urea had an average of 25.76 ± 6.87. For Hypertensive, Urea had an average of 30.33± 6.30. For Normotensive, Creatinine had an average of 0.95 ± 0.31. For Hypertensive, Creatinine had an average of 0.70 ± 0.21. For Normotensive, GPT had an average of 22.93 ± 5.67. For Hypertensive, GPT had an average of 17.62 ± 5.57. For Normotensive, GOT had an average of 25.73 ± 5.87. For Hypertensive, GOT had an average of 17.21 ± 6.35. The summary statistics can be found in Table 3-3.

Table 0-3 Results for Testing the Mean Differences of lipid profile and other biochemical parameters by Bp using t-Tests

	Variable	***M***	***SD***	***n***	***t***	***p***
Cholesterol	Normotensive	144.575	21.502	60	-5.362	< .001
	Hypertensive	165.040	20.286	60		
triglyceride	Normotensive	110.078	24.998	60	-0.850	.397
	Hypertensive	114.782	34.833	60		
HDL	Normotensive	45.836	7.660	60	-0.793	.430
	Hypertensive	47.045	8.995	60		
LDL	Normotensive	76.697	21.304	60	-5.091	< .001
	Hypertensive	95.010	17.955	60		
VLDL	Normotensive	22.013	4.998	60	-0.681	.498
	Hypertensive	22.770	7.022	60		
Urea	Normotensive	25.758	6.869	60	-3.799	< .001
	Hypertensive	30.330	6.302	60		
Creatinine	Normotensive	0.947	0.309	60	5.108	< .001
	Hypertensive	0.703	0.205	60		
GPT	Normotensive	22.935	5.670	60	5.181	< .001
	Hypertensive	17.618	5.571	60		
GOT	Normotensive	25.727	5.873	60	7.633	< .001
	Hypertensive	17.207	6.345	60		

Assessment of Obesity

In the present study, prevalence of obesity in normo- and Hypertensive patients was computed using WHO (2000) criteria of BMI. Subjects having BMI less than 30 kg/m2 were considered as non-obese and those BMI equal or above 30 kg/m2 as obese. The distribution of subjects according to BMI classification has been presented in Table 3-2

out of 120 subjects, 50% patients were non-obese and 50% were obese. Out of 60 (50%) normotensive subjects, 50% (30) were non-obese and 50% (30) were obese. Similarly, out of 60 (50%) hypertensive patients, 50% (30) patient were non-obese, whereas, 50% were obese, it is clear that the prevalence of obesity was equal and matched in hypertensive and normotensive individuals (x2=0.00, df=1, p= 1.00).

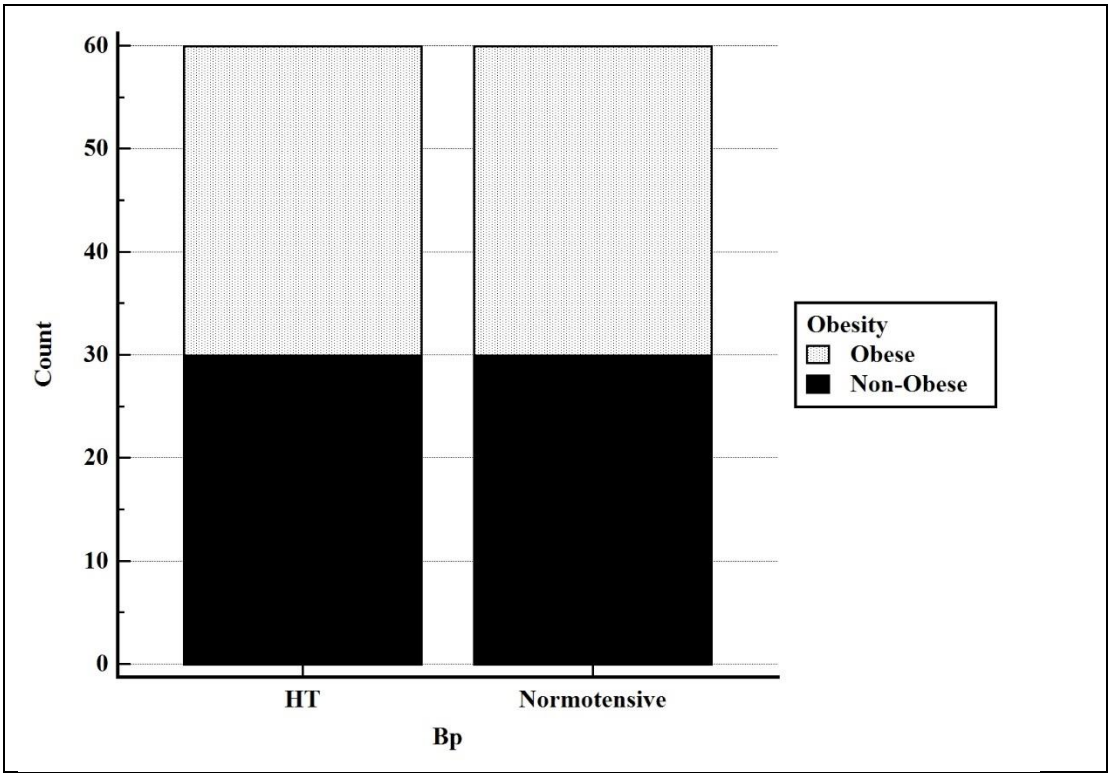


Figure 0-1 Percentage prevalence of Obesity in hypertensive and normotensive individuals

Table 0-4 Percentage prevalence of Obesity according to BMI criteria of WHO (2000) in Non-Obese and Obese hypertensive and normotensive individuals

Bp	Non-Obese	Obese	Total
HT	30 (50.0%	30 (50.0%)	60 (50.0%)
Normotensive	30 (50.0%)	30 (50.0%)	60 (50.0%)
Total	60 (50.0%)	60 (50.0%)	120
Chi-squared	0.000		
DF	1		
Significance level	P = 1.0000		

Assessment of Gender

Gender was also totally matched with male to female ratio 50:50 in both hypertensive and normotensive groups Table 3-3

Table 0-5 Gender distribution

Bp	female	male	Total
HT	25 (41.7%)	35 (58.3%)	60 (50.0%)
Normotensive	35 (58.3%)	25 (41.7%)	60 (50.0%)
Total	60 (50.0%)	60 (50.0%)	120
Chi-squared	3.306		
DF	1		
Significance level	P = 0.0690		

Comparison of BMI by Bp and Obesity

An analysis of variance (ANOVA) was conducted to determine whether there were significant differences in BMI by Bp and Obesity. The results of the ANOVA were significant, $F(3, 116) = 61.99$, $p < .001$, indicating there were significant differences in BMI among the levels of Bp and Obesity (Table 3). The interaction effect between Bp and Obesity was not significant, $F(1, 116) = 3.15$, $p = .079$, $\eta^2_p = 0.03$, indicating there were no significant differences for BMI for each factor level combination of Bp and Obesity. The main effect, Bp was not significant, $F(1, 116) = 0.14$, $p = .712$, indicating there were no significant differences of BMI by Bp levels. The main effect, Obesity was significant, $F(1, 116) = 182.69$, $p < .001$, $\eta_p^2 = 0.61$, indicating there were significant differences in BMI by Obesity levels. Paired *t*-tests between each pair of measurements revealed that the mean of BMI for Obese (36.06± 5.27) was significantly larger than for Non-Obese (25.78± 2.70), $p < .001$. The means and standard deviations are presented in Table 4

Table 0-6 Analysis of Variance Table for BMI by Bp and Obesity

Term	*SS*	*df*	*F*	*p*	η_p^2
Bp	2.37	1	0.14	.712	0.00
Obesity	3,171.17	1	182.69	< .001	0.61
Bp: Obesity	54.62	1	3.15	.079	0.03
Residuals	2,013.50	116			

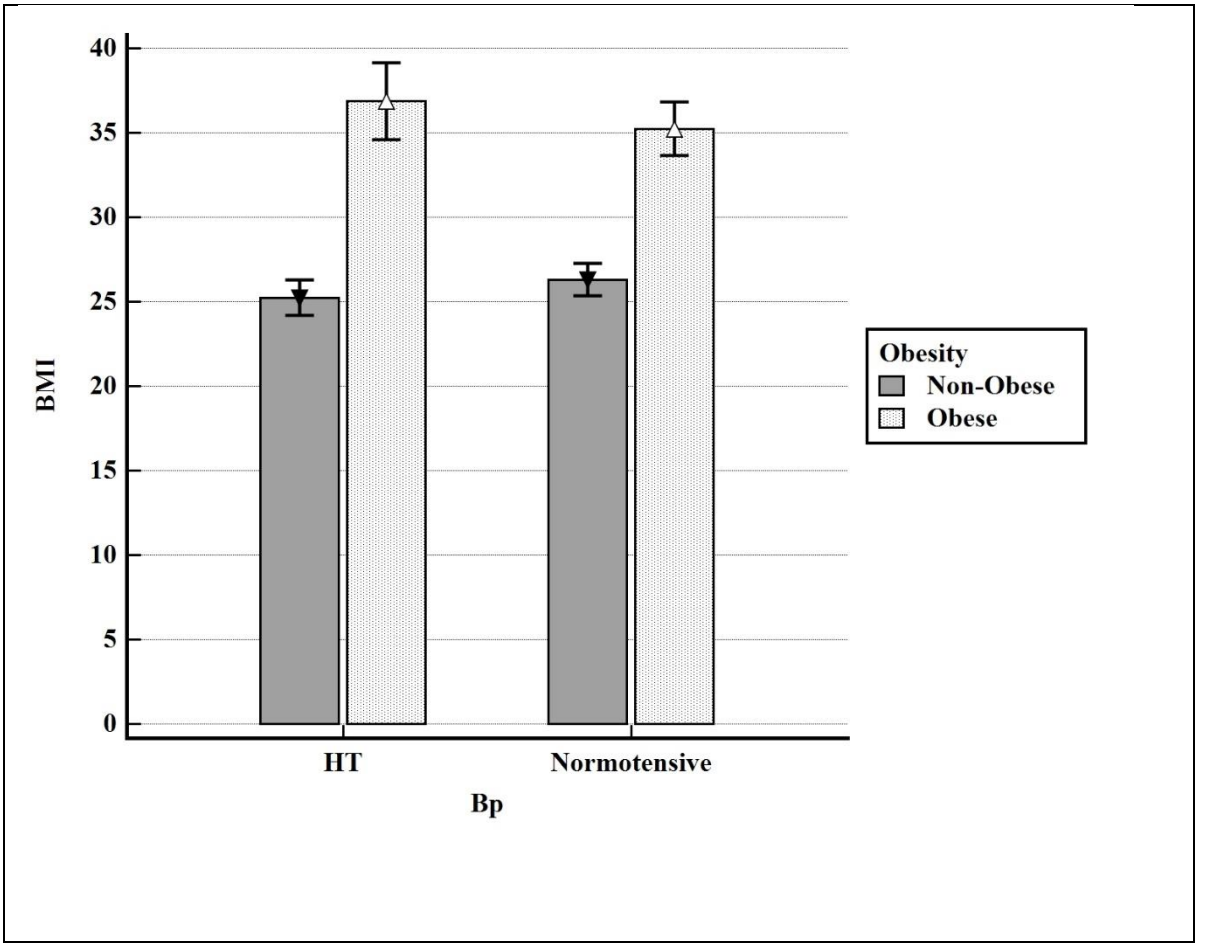
Table 0-7 Mean, Standard Deviation, and Sample Size for BMI by Bp and Obesity

Combination	M	SD	n
Normotensive: Obese	35.245	4.245	30
Hypertensive: Obese	36.876	6.093	30
Normotensive: Non-Obese	26.313	2.587	30
Hypertensive: Non-Obese	25.245	2.755	30

Table 0-8 Post-hoc Comparisons of BMI with Tukey's Honest Significant Difference Test:

Comparison	M	Lwr Limit	Upr Limit	p
Hypertensive-Normotensive	0.281	-1.225	1.788	0.712
Non-Obese-Obese	-10.281	-11.788	-8.775	2.109×10^{-15}
Hypertensive:Obese-Normotensive:Obese	1.631	-1.173	4.435	0.431
Normotensive:Non-Obese-Normotensive:Obese	-8.932	-11.736	-6.128	1.315×10^{-12}
Hypertensive:Non-Obese-Normotensive:Obese	-10.000	-12.804	-7.196	4.607×10^{-14}
Normotensive:Non-Obese-Hypertensive:Obese	-10.563	-13.367	-7.759	3.941×10^{-14}
Hypertensive:Non-Obese-Hypertensive:Obese	-11.631	-14.435	-8.827	2.864×10^{-14}
Hypertensive:Non-Obese-Normotensive:Non-Obese	-1.068	-3.872	1.736	0.754

Note. M is calculated on the differences between the groups in each comparison. Upper and lower limits for the means are calculated using a 95% confidence interval.



Comparison of waist circumference by Bp and Obesity

The ANOVA was examined based on an alpha value of .05. The results of the ANOVA were significant, $F(3, 116) = 22.86$, $p < .001$, indicating there were significant differences in Waist among the levels of Bp and Obesity (Table 11). The interaction effect between Bp and Obesity was not significant, $F(1, 116) = 0.00$, $p = .964$, $\eta^2_p = 0.00$, indicating there were no significant differences for Waist for each factor level combination of Bp and Obesity. The main effect, Bp was significant, $F(1, 116) = 4.66$, $p = .033$, $\eta_p^2 = 0.04$, indicating there were significant differences in Waist by Bp levels. The main effect, Obesity was significant, $F(1, 116) = 63.91$, $p < .001$, $\eta_p^2 = 0.36$, indicating there were significant differences in WAIST CIRCUMFERENCE by Obesity levels. Paired *t*-tests were calculated between each pair of measurements to further examine the differences among the variables based on an alpha of .05. The Tukey HSD p-value adjustment was used to correct for the effect of multiple comparisons on the family-wise error rate. For the main effect of Bp, the mean of waist circumference for Normotensive ($M = 101.73$, $SD = 16.56$) was significantly smaller than for Hypertensive ($M = 106.53$, $SD = 13.32$), $p = .033$. For the main effect of Obesity, the mean of WC for Obese ($M = 113.02$, $SD = 14.29$) was significantly larger than

for Non-Obese (M = 95.25, SD = 9.95), p < .001. The means and standard deviations are presented in Table 12.

Table 0-9 Analysis of Variance Table for waist circumference by Bp and Obesity

Term	*SS*	*df*	*F*	*p*	η_p^2
Bp	691.20	1	4.66	.033	0.04
Obesity	9,469.63	1	63.91	< .001	0.36
Bp:Obesity	0.30	1	0.00	.964	0.00
Residuals	17,188.73	116			

Table 0-10 Mean, Standard Deviation, and Sample Size for Waist circumference by Bp and Obesity

Combination	*M*	*SD*	*n*
Normotensive : Obese	110.67	17.27	30
Hypertensive : Obese	115.37	10.28	30
Normotensive : Non-Obese	92.80	9.73	30
Hypertensive : Non-Obese	97.70	9.70	30

Table 0-11 Post-hoc Comparisons of WC with Tukey's Honest Significant Difference Test:

Comparison	M	Lwr Limit	Upr Limit	p
Hypertensive-Normotensive	4.800	0.398	9.202	0.0328
Non-Obese-Obese	-17.767	-22.169	-13.365	1.100×10^{-12}
Hypertensive:Obese-Normotensive:Obese	4.700	-3.493	12.893	0.444
Normotensive:Non-Obese-Normotensive:Obese	-17.867	-26.059	-9.674	5.923×10^{-07}
Hypertensive:Non-Obese-Normotensive:Obese	-12.967	-21.159	-4.774	4.023×10^{-04}
Normotensive:Non-Obese-Hypertensive:Obese	-22.567	-30.759	-14.374	4.290×10^{-10}
Hypertensive:Non-Obese-Hypertensive:Obese	-17.667	-25.859	-9.474	7.913×10^{-07}
Hypertensive:Non-Obese-Normotensive:Non-Obese	4.900	-3.293	13.093	0.406

Note. M is calculated on the differences between the groups in each comparison. Upper and lower limits for the means are calculated using a 95% confidence interval.

Discussion

The results we demonstrated there showed that obese patients had lower aldosterone hormone increases than hypertensive obese patients, whereas non-obese and non-hypertensive patients had aldosterone hormone decreases. Previous studies have shown that high aldosterone is often associated with

hypertensive obesity, and low aldosterone is associated with hypotensive weight loss (Cat et al., 2016) (Kawarazaki & Fujita, 2016), and it has also been shown in studies that MDA doses in Blood pressure-related obesity cases do not increase unless it is a disease, as previous studies have shown that malondialdehyde is increased in high-fat obesity cases, malondialdehyde is the end product of peroxidized polyunsaturated acids, so Dialdehydes are high (Bhatt et al. 2020) (Savira et al. 2020). Increased oxidative stress (Zhou et al., 2021) (Ndisang et al., 2014), our study also showed that increased serum sodium in obese subjects increased hypertension compared with non-obese subjects with or without hypertension or without hypertension, in which aldosterone is increased and sodium is reabsorbed in the kidneys, compared to serum potassium in obesity-related stress, but not with the stress of renal excretion, it falls outside the body and blood levels drop (Kawarazaki & Fujita , 2016)

Reference

1. Kawarazaki W, Fujita T. The role of aldosterone in obesity-related hypertension. Am J Hypertens. 2016;29(4):415–23.
2. Purnell JQ. Definitions, Classification, and Epidemiology of Obesity. Endotext [Internet]. 2018;1–24. Available from: http://www.ncbi.nlm.nih.gov/pubmed/25905390
3. Sun JY, Ma YX, Liu HL, Qu Q, Cheng C, Kong XQ, et al. High waist circumference is a risk factor of new-onset hypertension: Evidence from the China Health and Retirement Longitudinal Study. J Clin Hypertens. 2022;24(3):320–8.