STUDY OF BMI, WAIST-HIP RATIO, LIPID PROFILE IN NORMOTENSIVE AND HYPERTENSIVE MALES.

M. Usha Rani, ¹ N. Sharmila², M. Padma Geetanjali³.

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ABSTRACT: This study was aimed at comparing the variables like Body mass index (BMI), Waist - Hip ratio (WHR) and Lipid profile in normotensive and hypertensive males. The age group of the subjects were between 35-65 years. The objectives of doing this study are as follows:

A) To measure the following parameters among the hypertensive and Normotensive males.
1) BMI (Kg / m2 ) : Body mass index 2) WHR: Waist-hip ratio 3) TC (mg / dl): Total serum Cholesterol level 4) TG (mg / dl): Total serum Triglyceride level 5) HDL-cholesterol (mg/dl): Serum High density lipoprotein level 6) LDL-cholesterol (mg/dl): Serum Low density lipoprotein level 7) VLDL-cholesterol (mg/dl): Serum Very low density lipoprotein Level 8) TC/ HDL-cholesterol ratio
B) To compare the values of the above mentioned parameters in hypertensive males with that of age matched normotensive males.
The present case-control study was carried out in 60 hypertensive males and 60 normotensive males. The subjects were selected from the general population of town area. Maximum males belonged to middle socioeconomic class. The subjects were between the age of 35-65 years. The mean duration of history of hypertension was 4.15 ± 1.30 years. All the subjects were free from cardiovascular diseases, cerebrovascular accidents, diabetes and any other illness or complications.

CONCLUSION:
1. There is increase in BMI in both groups suggestive of that obesity acts as a risk factor for hypertension.
2. There is increase in WHR in both groups suggestive of that central obesity in particular is associated with hypertension.
3. There is increase in Total cholesterol level, Triglyceride, LDL-cholesterol, VLDL-cholesterol levels, Total cholesterol to HDL-cholesterol ratio in hypertension suggestive of dyslipidemia is also a risk factor associated with hypertension
4. Effective control of Hypertension both by Non pharmacological and pharmacological method will reduce the incidence of cardiovascular, cerebrovascular and renal complications.
5) Hence for the prevention of hypertension and cardiovascular complications, health education regarding lifestyle modifications have to be done in the population.

KEY WORDS: BMI, Waist-Hip Ratio, Lipid Profile, Normotensive, Hypertensives.

INTRODUCTION: Hypertension is an important public health problem worldwide and is the most widely recognized modifiable risk factor for cardiovascular and cerebrovascular disease. The worldwide prevalence estimates for hypertension may be as much as 1 billion individuals per year and approximately 7.5 million deaths per year is attributed in the year 2004, i.e about 12.8% of the total global deaths. The estimated number of Indians with hypertension was 120 million in year 2000,
which is likely to increase to 200 million by 2025. The prevalence of hypertension in India is 59.9 and 35.5 per thousand males in the urban and rural population respectively. Obesity is another major public health problem worldwide including in the developing countries. According to world health organization (WHO) statistics 2012, approximately 2.8 million are obese. The worldwide prevalence of obesity (BMI>30kgm) almost doubled by 2008. The prevalence of obesity in India in men is 9.3%. The common diseases associated with obesity are hypertension, dyslipidemia, non-insulin dependent diabetes mellitus, atherosclerosis and certain types of cancer. According to data from the Framingham Cohort, obesity accounts for 78% of essential hypertension in men. Obesity and overweight are strong, independent risk factors for hypertension. It is estimated that 60% hypertensives are >20% overweight. Anthropometric measurements are universally applicable, simple, inexpensive and non-invasive techniques to measure obesity but still are underutilized tools for guiding public policy. Body mass index (BMI), Kg/m2, is a simple index of weight for height that is commonly used to classify adults as underweight, overweight and obese adults. A few individuals who are exceptionally muscular may be misclassified as overweight or obese. Therefore, obesity defined solely by BMI may be an imperfect approximation. The distribution of weight gain is of crucial importance. A predominantly "central" pattern of weight gain, is considered more ominous for cardiovascular and glycemic standpoints. It confers a far higher risk than that expected by BMI measurements. Central obesity is often referred as abdominal, android, or visceral obesity. Waist-hip ratio (WHR) is used to indicate abdominal fat accumulation. It gives a better viewpoint about the type of obesity as central or peripheral. Abdominal obesity is diagnosed clinically by a waist-to-hip ratio that is > 0.95 in men. Waist-hip ratio had strong relationship with type 2 diabetes, hypertension and dyslipidemia. Approximately 10% of the global population is affected by dyslipidemia. In the developed countries there are more than 240 million people with abnormal lipoprotein levels. Of these more than 55 million have low levels of HDL-cholesterol and / or high triglyceride levels. A 2006 study reveals a high incidence of dyslipidemia in India. Almost 40% of males had total cholesterol concentration above 200 mg/dl. High prevalence of dyslipidemia was seen in males than in females. Various cross sectional and prospective epidemiological studies have shown that hypertension increases significantly with higher BMI and Waist circumference.

Hypertension can be effectively controlled by secondary prevention (drugs) but primary prevention by means of life style modification, dietary approach to stop hypertension (DASH), should be top priorities for reducing the risk of cardiovascular and cerebrovascular mortalities. Therefore, the present study was undertaken to evaluate BMI, WHR and Lipid Profile in hypertensive and Normotensive males

**MATERIAL AND METHODS:** The present case-control study was carried out in 60 hypertensive males and 60 normotensive males. The subjects were selected from the general population of town area. Maximum males belonged to middle socioeconomic class. The subjects were between the age of 35-65 years. The mean duration of history of hypertension was 4.15 ± 1.30 years.

**SELECTION CRITERIA - 1) CASES-**
- Diagnosed cases of essential hypertension attending medicine OPD for regular check up.
Patients on antihypertensive drugs like Ramipril and Losartan and stamlo beta commonly.
Patients not suffering from any other disease or complication.

2) Controls -- Healthy and normotensive. The subjects with the following history were excluded from the study: --
- Those having cardiovascular illness, cerebrovascular accidents.
- Those having history of diabetes in the past or present.
- Those with the history of addiction to tobacco, alcohol, smoking.
- Those suffering from any other disease or complication.

In the present study each subject was made familiar with the procedure to alleviate any fear or apprehension. The physical examination of all the subjects before the start of the procedure was done with the help of proforma. The consent form was signed by the subjects before the procedure. All observations were made between 8.30 am and 9.30am.

Method - Apparatus -
- Mercury sphygmomanometer (Diamond)
- Measuring tape
- Human weighing machine (Kruups)
- Lipid profile Kit.

BLOOD PRESSURE MEASUREMENT: The subjects were initially made to rest for 15 mins. Then the blood pressure was measured two times in a sitting position during physical examination. Based on the circumference of the subject's arm, a regular adult cuff was chosen. The cuff was placed on the subject's right arm, at the heart level and blood pressure was measured by palpatory method. An interval of at least 2 minutes was kept between these two separate measurements, there after the mean of the two measurements was considered as the blood pressure. The systolic and diastolic blood pressure was then recorded by auscultatory method. The systolic blood pressure was defined as the appearance of the first sound (korotkoff phase I) and diastolic blood pressure as disappearance of sound (korotkoff phase 5) during deflation of cuff at a 2-3 mm per second decrement rate of mercury column. Two readings were taken at an interval of 2 minutes. Thereafter the mean of the two readings was considered as blood pressure.

The parameters selected for the present study are Body mass index (BMI), Waist hip ratio (WHR) and Lipid profile.

1) BODY MASS INDEX (BMI): For BMI the following anthropometric measurements were used -
- Weight (Kilograms) 15- Body weight was measured with human weighing machine (Kruups) in a standing position without shoes to the nearest 100 gm with minimal clothes. Height (meters) 15, 24-
- Height was measured with tape meter in standing position without shoes while the shoulders were in normal position, to the nearest 0.10 cm BMI was calculated as - 15 BMI = Weight in kilograms / (Height in meter)²

2) WAIST HIP RATIO (WHR): Waist circumference (centimeters) - 7.24 It was measured in centimeters at the midway point between the inferior margin of the last rib and the crest of ilium in a horizontal plane, while the person was standing with the abdomen relaxed at the end of the normal expiration. It was measured to the nearest 0.1 cm with the measuring tape. Hip circumference
It was measured in centimetres around the pelvis at the point of maximum protrusion of buttocks without compressing the skin. It was measured to the nearest 0.1 cm. WHR was calculated as -15'24 WHR = Waist circumference in cm Hip circumference in cm.

3) LIPID PROFILE: Collection of sample - The collection of blood samples has been done after overnight fasting from antecubital vein with all aseptic precautions. 2 ml blood was collected in plain bulb for estimation of serum lipids. Blood was allowed to clot at room temperature for half an hour and then centrifuged at 3000 rmp (revolutions per minute) for estimation of lipid profile. The tests were done on Mindray BS-300 Auto analyser. Estimation of serum Total Cholesterol - Enzymatic method. Methodology :- CHOD-PAP Method (Enzymatic colorimetric method).

PRINCIPLE: The estimation of cholesterol involves the following enzyme catalyzed reactions -

a) Cholesterol ester CE _____ Cholesterol + fatty acid
b) Cholesterol + 02 CHOP p Cholest-4-en-3-one +H2O2
c) 2H2O2 + 4 AAP + Phenol POD p 4 H2O + Quinoneimine
CE - Cholesterol esterase
CHOD - Cholesterol oxidase
AAP - Aminoantipyrene

The red Quinoneimine dye has maximum absorbance at 510 nm. The intensity of red colour is proportional to the concentration of cholesterol in solution.

REAGENTS:

REAGENT 1: Cholesterol reagent Active ingredient Concentration
  a) Cholesterol esterase > 200 IU / L
  b) Cholesterol oxidase > 150 IU/L
  c) Peroxidase 2000 IU / L
  d) Sodium phenolate 20 mmol / L
  e) 4 - Aminoantipyrine 0.5 m mol / L
  f) Phosphate buffer 68 mmol / L

REAGENT 2: Cholesterol standard
Cholesterol 200 mg / dl

REAGENT 3: (Aqua 4)
Doubled deionized, 0.2 micron, membrane filtered particle free water for reconstruction of reagent 1.

REAGENT RECONSTITUTION: Reagent 1 and reagent 3 was allowed to attain room temperature. Reagent 3 was added as indicated on the label, to contents of each vial of reagent 1. Then swirled to dissolve but not vigorously.

ASSAY PROCEDURE: SR.NO.

REAGENTS BLANK STANDARD SAMPLE:
1. Working reagent 1000uL
2. Distilled water 10uL
3. Standard — 10 uL
4. Sample — — 10UL
Mixed well and incubated at 37°C for 10 mins. The absorbance of standard and each sample was read at 510 nm.
Calculation: -
Absorbance of sample = Cholesterol (mg / dl)
-------------------- X Concentration of standard
Absorbance of standard (mg / dl)

**ESTIMATION OF SERUM TRIGLYCERIDES:**
**METHODODOGY: GLYCEROL PHOSPHATE OXIDASE (GPO) AND PEROXIDASE (POD)**

**PRINCIPLE:** lipase

\[ \text{Triglycerides} + \text{H}_2\text{O} \rightarrow \text{Glycerol + FFA} \]

\[ \text{Glycerol + ATP} \rightarrow \text{Glycerol -3-phosphate +ADP} \]

\[ \text{GPO} : \text{Glycerol} -\text{3-phosphate} + \text{O}_2 \rightarrow \text{DAP} + \text{H}_2\text{O}_2 \]

\[ \text{peroxidase} \]

\[ \text{H}_2\text{O}_2 + 4\text{ AAP} + 3,5\text{ DHBS} \rightarrow \text{Quinoneimine} + 2\text{H}_2\text{O} \]

\[ \text{GK} : \text{- Glycerol kinase} \]

\[ \text{GPO} : \text{- Glycerol phosphate oxidase} \]

\[ \text{DAP} : \text{- Dihydroxy acetone phosphate} \]

\[ \text{ATP} : \text{- Adenosine triphosphate} \]

\[ \text{DHBS} : \text{- 3,5 Dichloro- 2-hydroxybenzene sulfonate} \]

The intensity of Quinoneimine formed is proportional to the triglyceride concentration in the sample when measured at 510 nm.

**REAGENT COMPOSITION:** Reagent -1 (Triglycerides DES reagent) Active Ingredient Concentration

1) ATP 2.5 mmol / L
2) Mg2+ 2.5 mmol / L
3) 4-Aminoantipyrine 0.8 mmol / L
4) 3-5 DHBS 1 mmol/L
5) Peroxidase > 2000IU / L
6) Glycerol kinase > 550 IU / L
7) GPO > 8000 IU/L
8) Lipoprotein lipase > 3500 IU / L
9) Buffer 53 mmol / L

Also contains non-reactive fillers, stabilizers and surfactants.

**REAGENTS - 2 (TRIGLYCERIDE STANDARDS):** Triglyceride Standards 200 mg / dl Reagent bottle and Aqua-4 (supplied in the kit) is allowed to attain room temperature. Then Aqua-4 was added as indicated on the label to the contents of each vial The bottle was swirled to dissolve and then allowed to stand for 10 minutes at room temperature.

**ASSAY PROCEDURE:** Sr. No. Reagents Blank Standard Sample

1. Working reagent 1000 uL
2. Distilled water 10 uL
3. Standard ~ 10 uL ~
4. Sample — — 10 uL

Mixed well and incubated at 37°C for 10 mins. The absorbance of standard and each sample was read at 510 nm.

**CALCULATIONS:**
Absorbance of sample
Triglycerides (mg / dl) = ----------------------------- X Concentration of standard
Absorbance of standard (mg / dl)

**ESTIMATION OF SERUM HDL - CHOLESTEROL: (Phosphotungstic acid method) 45**

**Principle:** Chylomicron, LDL-C and VLDL-C are precipitated from serum phosphotungstate in presence of divalent cations such as mg2+. The HDL-C remains unaffected in the supernatant and is estimated using ERBA cholesterol reagent.

Phosphotungstate
Serum/plasma ----------------- ► HDL-C + (LDL-C + VLDL-C)
Mg

**REAGENT COMPOSITION:**
Reagent -1 (precipitating reagent)
Phosphoric acid 2.4 mmol / L
Magnesium chloride 40 mmol / L

**REAGENT - 2 (HDL-C STANDARD):** HDL-C standard 25 mg / dl

**PRECIPITATION OF LDL-C, VLDL-C AND CHYLOMICRONS:**
Pipette Volumes
Sample 250ul
Precipitate reagent 500ul

2 +
Mixed well and allowed the reaction mixture to stand for 10 minutes at room temperature .Centrifuged at 4000 rpm for 10 minutes to obtain a clear supernatant. The supernatant was used to determine the concentration of HDL-cholesterol in the sample.

**ASSAY PROCEDURE:**
Sr.No. Reagents Blank Standard Sample
1. Cholesterol Working reagent 1000 uL 1000 uL 1000 uL
2. Distilled water 50 uL ~
3. Standard — 50 uL —
4. Supernatant — — 50 uL

Mixed well, incubated at 37 C for 10 mins. The absorbance of standard and each sample was read at 510 nm.

**Calculations:**
Absorbance of sample
HDL-cholesterol (mg/dl) = ----------------------------- X Value of standard
Absorbance of standard Dilution Factor
QUANTITATIVE ESTIMATION OF LDL-CHOLESTEROL AND VLDL-CHOLESTEROL: (By Friedwald method) 36 Estimation of LDL-cholesterol and VLDL-cholesterol is based on indirect calculations made from total cholesterol, HDL-cholesterol and Triglycerides

1) VLDL-cholesterol = TG/5
2) LDL-cholesterol = TC - (HDL-C + TG / 5)
3) LDL-cholesterol = TC-(HDL-C + VLDL-C)

STATISTICAL METHOD: The results were Statistically analysed by Independent Sample t-test. P-value of < 0.05 was considered statistically significant.

\[
\text{S.E. (}\overline{X}_1 - \overline{X}_2) = \sqrt{\frac{(SD_1)^2}{n_1} + \frac{(SD_2)^2}{n_2}}
\]

S.E. (\(\overline{x}_1 - \overline{x}_2\)) - Standard error of difference between two means.
SDi - Standard deviation of means of first sample. SD2 - Standard deviation of means of second sample.
n1 - Number of subjects in first sample.
n2 - Number of subjects in second sample.
## RESULTS

### Group Statistics

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<tr>
<th>Group</th>
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## Independent Samples Test

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<th>Std. Error Difference</th>
<th>95% Confidence Interval of the Difference</th>
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<td>.000</td>
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<td>5.847</td>
<td>32.433 to 55.634</td>
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<td>6967</td>
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<td>6.567</td>
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<td>111641</td>
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<td>.77882 to 1.45400</td>
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Table no. 3 comparison of BMI between hypertensive and Normotensive males.

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<th>GROUPS</th>
<th>CASES</th>
<th>CONTROLS</th>
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<tbody>
<tr>
<td>NUMBER(n)</td>
<td>60</td>
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<tr>
<td>Mean</td>
<td>28.68</td>
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<td>S.D</td>
<td>4.2</td>
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<td>S.Error of mean</td>
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<td>0.15</td>
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<td>t-value</td>
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<td>p-value</td>
<td>3.508E-15 (&lt;0.01)</td>
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<td>SIGNIFICANCE</td>
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</table>

Result: There is a highly significant difference of mean BMI among Hypertensives as compared to BMI of Normotensive males.
Table no.4 Comparison of WHR between Hypertensive and normotensive men.

<table>
<thead>
<tr>
<th>GROUPS</th>
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<th>CONTROLS</th>
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<tr>
<td>NUMBER(n)</td>
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<tr>
<td>Mean</td>
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<td>S.D</td>
<td>0.08</td>
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<td>S.Error of mean</td>
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<td>t-value</td>
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<td>9.914</td>
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<tr>
<td>P-VALUE</td>
<td>9.546E-17 (&lt;0.01)</td>
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<td>SIGNIFICANCE</td>
<td>HIGHLY SIGNIFICANT</td>
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</table>

Result: There is a highly significant difference of mean WHR among hypertensive males as compared to WHR of Normotensive males.
Table no.5 Comparison of TC (mg/dl) between Hypertensive and normotensive males.

<table>
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<th>GROUPS</th>
<th>CASES</th>
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<tr>
<td>NUMBER(n)</td>
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<tr>
<td>Mean</td>
<td>203.92</td>
<td>151.48</td>
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<tr>
<td>S.D</td>
<td>42.16</td>
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<td>S.error of mean</td>
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<td>t-value</td>
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<td>P.VALUE</td>
<td>5.11E-13 (&lt;0.01)</td>
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</table>

**Result:** There is a highly significant difference in mean of TC among hypertensive males as compared to normotensive males.
Table no.6 Comparison of TG (mg /dl) between Hypertensive and normotensive males.

<table>
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<th>CONTROLS</th>
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<td>NUMBER(n)</td>
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</tr>
<tr>
<td>S.Error of mean</td>
<td>8.21</td>
<td>6.51</td>
</tr>
<tr>
<td>t-value</td>
<td>3.12</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td>0.0022418(&lt;0.01)</td>
<td></td>
</tr>
<tr>
<td>SIGNIFICANCE</td>
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<td></td>
</tr>
</tbody>
</table>

Result: There is a significant difference in mean of TG among hypertensive males as compared to normotensive males.
Table no.7 Comparison of HDL-cholesterol (mg/dl) between Hypertensive and normotensive males.

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>CASES</th>
<th>CONTROLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number(n)</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>Mean</td>
<td>41.17</td>
<td>39.17</td>
</tr>
<tr>
<td>S.D</td>
<td>5.61</td>
<td>3.07</td>
</tr>
<tr>
<td>S.Error of mean</td>
<td>0.72</td>
<td>0.39</td>
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<td>t-value</td>
<td>2.42</td>
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</tr>
<tr>
<td>p-value</td>
<td>0.017526(&lt;0.01)</td>
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</tr>
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</tbody>
</table>

Result: There is a significant difference in mean of HDL-cholesterol among hypertensive males as compared to normotensive males.
### Table no.8 Comparison of LDL-cholesterol (mg/dl) between Hypertensive and Normotensive males.

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>CASES</th>
<th>CONTROLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>NUMBER(n)</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>Mean</td>
<td>127.37</td>
<td>83.33</td>
</tr>
<tr>
<td>S.D</td>
<td>38.25</td>
<td>24.25</td>
</tr>
<tr>
<td>S.Error of mean</td>
<td>4.93</td>
<td>3.13</td>
</tr>
<tr>
<td>t-value</td>
<td>7.53</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td>2.28E-11(&lt;0.01)</td>
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<tr>
<td>SIGNIFICANCE</td>
<td>HIGHLY SIGNIFICANT</td>
<td></td>
</tr>
</tbody>
</table>

**Result:** There is a highly significant difference in mean of LDL-cholesterol among hypertensive males as compared to normotensive males.
Table no.9 Comparison of VLDL-cholesterol (mg/dl) between Hypertensive and Normotensive males.

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>CASES</th>
<th>CONTROLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>NUMBER(n)</td>
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<td>60</td>
</tr>
<tr>
<td>Mean</td>
<td>35.63</td>
<td>28.67</td>
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<tr>
<td>S.D</td>
<td>12.38</td>
<td>10.30</td>
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<tr>
<td>S.Error of mean</td>
<td>1.59</td>
<td>1.33</td>
</tr>
<tr>
<td>t-value</td>
<td>3.34</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td>0.0010993(&lt;0.01)</td>
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</tr>
<tr>
<td>SIGNIFICANCE</td>
<td>HIGHLY SIGNIFICANT</td>
<td></td>
</tr>
</tbody>
</table>

**Result:** There is a highly significant difference in mean of VLDL-cholesterol among hypertensive males as compared to normotensive males.
Table no. 10 Comparison of TC / HDL-cholesterol between Hypertensive and Normotensive males.

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>CASES</th>
<th>CONTROLS</th>
</tr>
</thead>
<tbody>
<tr>
<td>NUMBER(n)</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>Mean</td>
<td>4.99</td>
<td>3.88</td>
</tr>
<tr>
<td>S.D</td>
<td>1.14</td>
<td>0.64</td>
</tr>
<tr>
<td>S.Error of mean</td>
<td>0.14</td>
<td>0.08</td>
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<tr>
<td>t-value</td>
<td>6.56</td>
<td></td>
</tr>
<tr>
<td>p-value</td>
<td>2.908E-09 (&lt;0.01)</td>
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<tr>
<td>SIGNIFICANCE</td>
<td>HIGHLY SIGNIFICANT</td>
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</tr>
</tbody>
</table>

Result: There is a highly significant difference in mean of TC / HDL-cholesterol ratio among hypertensive males as compared to normotensive males.
RESULTS

1. Table No.1: shows group statistics of mean, standard deviation and standard error of mean for the above parameters in hypertensive and Normotensive males.

2. Table No. 2: shows Independent Sample Test comprising of Levine's test for equality of variances and t-test for equality of means for the above parameters.

3. Table No.3: shows a statistically highly significant increase in BMI among Hypertensive males as compared to Normotensive males.

4. Table No.4: shows a statistically highly significant increase in WHR among Hypertensive males as compared to Normotensive males.

5. Table No 5: shows a statistically highly significant increase in Total cholesterol level (TC) among hypertensive males as compared to normotensive males.

6. Table No.6: shows a statistically significant increase in Triglyceride level (TG) among hypertensive males as compared to normotensive males.

7. Table No 7: shows no significant change in the levels of High density lipoprotein (HDL-cholesterol) among hypertensive males as compared to Normotensive males.

8. Table No 8: shows a statistically highly significant increase in Low density lipoprotein (LDL-cholesterol) levels among hypertensive males as compared to normotensive males.
DISCUSSION: In the present study, the careful statistical analysis of observed values of Body mass index, Waist hip ratio and lipid profile (Total cholesterol, Triglyceride, HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol levels, Total cholesterol to HDL-cholesterol level ratio) are done between hypertensive males with age matched normotensive males. The mean blood pressure in our study was 121.33±7.18/79.7±6.35 and 147.2±9.99/93.13±6.49mmHg in normotensive and hypertensive males respectively.

COMPARISON OF BMI BETWEEN HYPERTENSIVE AND NORMOTENSIVE MALES: In the present study, it was observed that there is a high statistically significant difference in Body mass index (BMI) between hypertensive and normotensive males. The hypertensive males have high BMI values than normotensive (Table 3). According to the classification of W.H.O (World health organization) of BMI the hypertensive males found in the present study fall in the category of overweight individuals. Overweight > 25 is further classified as Pre-obese 25-29.9, Obese I 30-34.9, Obese II 35-39.9, obese III £ 40.2 In Asian Indians, the relative contribution of fat is more and muscle is decreased.


Sharma et al (2008) found that hypertension was significantly higher among persons with adverse BMI, WC, WHR. They also found that BMI was the most sensitive (27.4%) of the three indicators for detecting obesity but M.T. Guagnango et al (2001) reported that WC is the most important anthropometric factor associated with hypertensive risk and that BMI shows no relation to hypertensive risk.
Body mass index (BMI), Kg/m^2, has high correlation with body fat. The adipose tissue plays a causative role in the genesis of hypertension. Increase in BMI leads to hypertension by various mechanisms:

1. Increase in mass of fat causes insulin resistance. This results in hyperinsulinemia. Insulin predisposes to hypertension by the following mechanisms:
   - Stimulating renal sodium reabsorption.
   - Stimulation of sympathetic nervous activity.
   - Increased secretion of endothelin.
   - Impaired vasodilatation.

2. In obese persons, leptin levels increase due to adipocyte accumulation. It stimulates arcuate POMC (pro-opiomelanocortin) expression and a - MSH (melanocyte stimulating hormone), which would then act elsewhere in the hypothalamus on MC4 - R - expressing neurons (melanocortin receptors), causing decreased food intake and increased sympathetic activity. This may be a crucial role linking obesity and hypertension.

3. The obese subjects have elevated sympathetic activity that may contribute significantly to obesity related hypertension. The various mechanisms and mediators for elevated adrenergic activity are:
   - Elevated insulin levels with insulin resistance.
   - Elevated leptin levels.
   - Activation of renal afferents stimulated by increased intrarenal pressures, due to structural and functional alterations in the kidney.
   - Angiotensin II release
   - Potentiation of central chemoreceptors sensitivity.
   - Impaired baroreceptor sensitivity.

4. The Renin angiotensin aldosterone system seems to be activated in obesity, despite volume expansion and sodium retention. Reports suggest that a positive relationship between plasma angiotensin level, plasma rennin activity, and plasma angiotensin converting enzyme with BMI exits in humans. Moreover elevated serum aldosterone levels have been reported in obese.

5) Adipocytes act as a source of inflammatory cytokines, such as tumour necrosis factor - a, interleukin - 6, C- reactive protein and plasminogen activator inhibitor. Thus obesity acts as a low grade inflammatory condition. The progressive pro-inflammatory state resulting from increased obesity promotes insulin resistance and also perpetuates atherogenesis. The endothelial modulators such as vasoactive endothelial growth factor, plasminogen activator inhibitor - I, angiotensinogen, renin and angiotensin II are secreted by fat cells that contribute to vasomotor dysfunction and cause hypertension and endothelial injury.

6. Lawrence de konen et al (2007) suggested that WHR is strongly associated with cardiovascular risk factors. It was found that centrally obese men are more susceptible to high TG (Triglyceride), high TC (Total cholesterol) levels. Yildiran Hilal et al (2010)1found that there was significant association between anthropometric measurements and hypertension. They also concluded that levels of TC, LDL-C, and TG are significantly higher among hypertensive subjects.
Thus the above mentioned mechanisms explain the positive association found between Body mass index and Hypertension.

**COMPARISON OF WHR BETWEEN HYPERTENSIVE AND NORMOTENSIVE MALES:** In our study, the WHR when compared between hypertensive and normotensive males is statistically highly significant. The hypertensive males have high WHR as compared to normotensive males. (Table 4) Our study matches with the study of Larsson Bet al (1989), Phil E* Jurimae T. et al S (2001), M.T. Guagnango et al (2001) K-C Huang et al (2002), F.Aziz et al (2004), Lawrence de Koning et al (2007), A.Lattifah et al (2008), Sharma et al (2008)5. Dalton M.et al (2003), studied that WHR had the strongest positive association with hypertension as compared to BMI and WC. A.Esmailzadeh et al (2004) concluded that of all the anthropometric indices WHR had highest sensitivity, specificity and accuracy to predict cardiovascular risk factors. Al-Lawati J.A et al (2008) found that WHR was a better predictor of coronary heart disease followed by WC and then BMI. Central obesity or visceral obesity is often referred as abdominal obesity, which is diagnosed by WHR.

- Compared to subcutaneous adipose tissue, intraabdominal adipose tissue has more fat cells per unit mass, high blood flow, more androgen receptors and greater catecholamine induced lipolysis, Thus in subjects with visceral obesity free fatty acid load is delivered to the liver via the portal blood.
- This activates hepatic afferent pathways leading to sympathetic activation and contributes to insulin resistance. Thus abdominal obesity plays important role in development of insulin resistance leading to hypertension.
- Abdominal adipocytes are more sensitive to anti-lipolytic effects of insulin. Hence they become more hypertrophic when exposed to insulin.
- The free fatty acids may also enhance reflex vasoconstrictor responses in peripheral circulation.
- Moreover central adipose tissue in particular acts as a rich source of inflammatory cytokines which is increasingly important in the causation and progression of hypertension and atherosclerosis.

Thus central obesity, especially intra-abdominal fat accumulation, is known to be closely related to insulin resistance and its disorders including hypertension and dyslipidemia. Hence hypertensive subjects show higher WHR than normotensive males.

In the present study, it has been found that there is statistically high significant difference in TC, TG, LDL-cholesterol, VLDL-cholesterol levels and TC / HDL-cholesterol ratio in hypertensive and normotensive males.. The hypertensive males have high TC, TG, LDL-cholesterol, VLDL-cholesterol levels, TC/ HDL-cholesterol ratio where as there is no significant change in the levels of HDL-cholesterol levels as compared to normotensive males.

(Tables-5- 10) M. Solati et al (2004) also found that systolic and diastolic blood pressure were significantly higher in men with serum TG level > 1.8 mmol / L and with WC > 95 cm. They were also found to have high serum TC, LDL-cholesterol levels and low HDL- cholesterol level. A.L. Lattifah et al (2008) concluded that the TC level, TG level, LDL-cholesterol level were significantly higher among hypertensive subjects as compared to normotensive subjects.
David E Laaksonen et al (2008) proved that dyslipidemia predicts the development of hypertension during a seven year follow up. They observed that a 1 - standard deviation change in TG factor was associated with 1.5 - 1.6 fold increase in risk of hypertension. Disturbed metabolism of LDL-cholesterol was associated with a 2.3 - 2.6 fold increase risk of hypertension. Abnormal TG and LDL-cholesterol metabolism seemed to be most strongly associated with the development of hypertension. Hypertension may be a consequence of dyslipidemia or closely related metabolic abnormality.

Long term observational studies, such as the Framingham Heart Study in US, provided evidence about coronary heart disease relative risk which decreased by 2-3% with every 1 mg/dl increase in HDL-cholesterol while with every 1 mg/dl decrease in LDL-cholesterol the risk decreased by 1%.

The cardioprotective effect of HDL-cholesterol is mainly exerted by facilitating the ‘reverse cholesterol transport’. The binding of HDL-cholesterol receptor class BI (SR-B1) leads to activation of endothelial nitric oxide synthase (eNOS) and therefore enhances vasorelaxation. It also inhibits adhesion of monocytes to endothelium, and has antioxidant effect. The other adjuvant effects of HDL are, anti-inflammatory, anti-oxidant, anticoagulant and fibrinolysis.

LDL-cholesterol and Triglycerides may damage the epithelium, impair NO (Nitric oxide) release and cause endothelial dysfunction. Triglyceride rich lipoproteins and LDL-cholesterol have been shown to be toxic to endothelial cells, whereas HDL-cholesterol may be protective. Therefore long term damage to the endothelium may lead to increased peripheral vascular resistance and thus arterial hypertension.

The atherogenic lipoproteins are present not only in circulating bloodstream, but are also in arterial wall, where they accumulate and are prone to oxidative modification, Dyslipidemia thus also leads to arterial wall stiffness and decreased arterial compliance.

The mechanism remains speculative, but endothelial dysfunction is integral not only in pathogenesis of atherosclerosis, thrombosis and insulin resistance but also in hypertension.

VLDL-cholesterol is the precursor of IDL-cholesterol and LDL-cholesterol is a precursor of LDL-cholesterol. Thus one LDL particle is derived from one VLDL particle. Hence increase in VLDL leads to increase in LDL. Abnormal triglycerides and LDL-cholesterol metabolism seems to be most strongly associated with the development of hypertension. Hence prolonged elevated levels of VLDL-cholesterol, IDL-cholesterol and LDL-cholesterol in blood are often accompanied by premature or more severe atherosclerosis.

Hypercholesterolemia is also found to be associated with increased levels of circulating angiotensinogen and angiotensin peptides and all components of renin-angiotensin-aldosterone system including renin resulting into hypertension.

Thus dyslipidemia i.e. increase in TC, TG, LDL-cholesterol, VLDL-cholesterol and decrease in HDL-cholesterol is associated with hypertension. Moreover as there is increase in total cholesterol and decrease in HDL-cholesterol level in hypertension the increase in TC / HDL-cholesterol ratio is also associated with hypertension.

Hence in our study hypertensive males have increased BMI ( Body mass index), WHR ( Waist hip ratio ), and altered lipid profile i.e. increase in TC, TG, LDL-cholesterol, VLDL-cholesterol, TC to HDL-cholesterol ratio where as there is no significant change in the levels of HDL-cholesterol between two groups which is similar to the study done by Parinita Kataraki et al (2012).
CONCLUSION:

1. There is increase in BMI in both groups suggestive of that obesity acts as a risk factor for hypertension.
2. There is increase in WHR in both groups suggestive of that central obesity in particular is associated with hypertension.
3. There is increase in Total cholesterol level, Triglyceride, LDL-cholesterol, VLDL-cholesterol levels. Total cholesterol to HDL-cholesterol ratio in hypertension suggestive of dyslipidemia is also a risk factor associated with hypertension.
4. Effective control of Hypertension both by Non pharmacological and pharmacological method will reduce the incidence of cardiovascular, cerebrovascular and renal complications.
5. Hence for the prevention of hypertension and cardiovascular complications, health education regarding lifestyle modifications have to be done in the population:-

- Dietary Approaches to stop Hypertension (DASH) diet.
- A diet with increased fresh fruit and vegetables and reduced saturated fat content.
- Reduction of dietary sodium intake and increased dietary potassium intake.
- Regular Aerobic physical activity such as brisk walking (at least 30 mins/day most days of week) reduces systolic blood pressure by approximately 4-9mm/Hg.
- Aerobic exercise lowers blood pressure in previously sedentary individuals.
- Periodic check-up of lipid profile to avoid complications.
- Periodic check-up of Blood pressure to avoid complications.

BIBLIOGRAPHY:


**AUTHORS:**
1. M. Usha Rani
2. N. Sharmila
3. M. Padma Geetanjali.

**PARTICULARS OF CONTRIBUTORS:**
1. Associate Professor, Department of Physiology, Andhra Medical College, Visakhapatnam, Andhra Pradesh.
2. Assistant Professor, Department of Physiology, Andhra Medical College, Visakhapatnam, Andhra Pradesh.
3. Professor, Department of Physiology, Andhra Medical College, Visakhapatnam, Andhra Pradesh.

**NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:**
Dr. M. Usha Rani,
Associate Professor,
Andhra Medical College,
Visakhapatnam, Andhra Pradesh.
Email- ushprasad6@gmail.com.

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