A Study of Lipid Profile in Obese Hypertensive Subjects

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Abstract

Background: Obesity is a significant risk factor for metabolic syndrome in adults. Central fat distribution greatly alters the lipid profile and induces atherogenic dyslipidemia even in normoglycaemic, non-hypertensive subjects.

Aim and Objectives: Hence, the aim of the present study to assess lipid profile changes in non-obese hypertensive subjects. Obesity, hypertension and dyslipidemia are the three highly significant risk factor for the deranged lipid profile. Obesity can be defined as excess accumulation of body fat arising from a sustained or a periodic positive energy balance that when energy intake exceeds energy expenditure[1]. Indicators of overweight are useful in the diagnosis and management of obesity in both children and adults.

Material & Methods: This study was conducted on newly diagnosed cases of essential hypertension attending medical outdoor of M.G.M. Medical College, Kisangani. A complete clinical examination including laboratory investigation was done to exclude any systemic or other diseases which are likely to affect blood lipid levels directly or indirectly.

Results- The association between dyslipidaemia, obesity and hypertension is well establishedand all have been found to be major risk factor for the development of CAD, a leading cause of visits to physician and cause of death .

Conclusion: Our study was envisaged to know the effect of obesity on lipid profile profile only in hypertensive and not in general population, and the study found some definite but paradoxical effects. These are that in obesity on a background of hypertension, the total and LDL cholesterol as also the HDL cholesterol are decreased, but on use other hand, the value of VLDL cholesterol and triglycerides are grossly and significantly increased. These finding have two major Clinical implications in that obese hypertensives will be more prone to metabolic syndrome and type 2 diabetes mellitus, and steps should be taken to prevent them accordingly and also apart from statins one should treat the obese hypertensives with fibrates, fat restriction and physical exercise also.

Key words: *Obesity, Lipid profile,dyslipidaemia, Hypertension*.

Background

Obesity, hypertension and dyslipidaemia is these are the three highly significant risk factors for overall human health, life expectancy and easily morbidity, particularly in relation to cerebrovascular and cardiovascular disease profiles. These factors are so extremely well known that they don't need my references to note. Obesity can be defined as excess accumulation of body fat arising from a sustained or a periodic positive energy balance that is when energy intake exceeds energy expenditure[1]. The most common method of classifying overweight and obesity is based on Body Mass Index (BMI) . According to the World Health Organization (WHO) classifies individual with BMI 25-29.99 Kg/

m^2 as overweight while individuals with BMI> 30 Kg/ m^2 are termed obese. Obesity can also be measured by knowing the body fat contest using various methods like Waist circumference (WC), Waist-Hip ratio (WHR), Skin and subcutaneous fat thickness in various areas of the body and also by measuring the Bio-electrical impendence of the body which is grossly affected by the body fat. Obesity is strongly related with hypertension dyslipidaemia and metabolic syndrome. In our thesis we are particularly interested in established the relationship of obesity with hypertension and lipid profile status.[1] Hypertension is the most common cardiovascular morbidity seen in the primary care and leads various fatal or severely morbid conditions like myocardial infarction, cerebral haemorrhage, cerebral thrombosis, renal failure, heart failure and death, if not detected early and treated appropriately. As per INC 812) report hypertension is regarded and is treatable in all adults with a B.P.> 140/90 mm Hg and in person with diabetes, CKD or related systemic co-morbidities the same values are maintained to define hypertension. Hypertension is a common disorder affecting 20 % of adult population[2] and 12 % of all deaths are caused by hypertension and its complications. We all know that.

Obesity has been described as an epidemic in many places throughout the world and its prevalence is of great concern. The World Health Organization (WHO) has defined obesity as having a body mass index (BMI) over 30 (body mass/ ht2). BMI does not directly assess how much fat a person has but is an indirect assessment that assumes that a higher body mass is due to an increasing percentage of the body's mass being fat 1191 Body mass index (BMI), which was defined by the World Health Organization (WHO), has been used for many years as a global index for assessing obesity[3].

India is undergoing rapid economic transition. At this stage in the associated epidemiological transition, the country is facing the double burden of communicable and non -communicable diseases. As in all such transitions, nutrition plays a central part. Obesity, representing one extreme of the continuum, is a preventable risk factor for chronic degenerative diseases while chronic energy deficiency (CED), though less directly " preventable," is associated with impaired physical capacity, reduced economic productivity, increased mortality, and poorer reproductive outcomes.

Under all these variable finds in manifold studies, it is assumed that the Lipid profile should be studied in detail in our context, that is, in Bihar (Kishanganj) district, India, in as much as obese hypertensive have already two risk factors, and we should know whether in most of the obese Hypertensive cases, The lipid profile is truly adverse and renders a third dimension in the issue, or in all hypertensive, the lipid profile shows no significant difference irrespective o f whether the hypertensive person is obese or not. This is why we have ventured to proceed on to the present study whereby the lipid profile is evaluated and compared between, obese and non obese hypertensive patients.

Although hypertension and obesity are both closely associated, there is no universal anthropometric marker of this association. This is probably due to distinct population characteristics, and in the case of Brazil, the highly heterogeneous population.[4,5]

The current estimated frequency of hypertension in India is 10-15% in rural and 25-30% in urban population as shown in different epidemiological studies. Due to the stress and tension inherent within the altering life patterns there is a changing trend in the overall prevalence of hypertension.[6,7]

Hypertension is also directly responsible for 57 percent of all strokes deaths and 24 percent of coronary heart disease death in India and also remains to be the leading cause of blindness, renal failure and congestive heart failure. Previously thought as a disease of urban well-off population, and a rural infringement in nowadays increasingly being reported. The probability of escalating cardiovascular diseases in rural India is a public health concern and not much research had been done to know about the burden and risk factors in rural areas.[8,9]

Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease

deaths in India.[10]

The meta-analysis of eight studies carried out in urban areas gives a pooled prevalence rate of 164.18 per thousand and in rural areas was 15744 per thousand. 4 Pooling of epidemiological studies shows that hypertension is present in 25% urban and 100% rural subjects in India.[10]

Almost 30-65% of adult urban Indians are reported to be either overweight (BMI>=25) or obese (BMI>=30) or have central obesity.[10]

Apart from the age groups 25-34 and 65+, the mean BMI was significantly higher for women compared to men across age-groups The standardized prevalence of overweight ($25 Kg/m^2 \leq BMI < 30 Kg/m^2$) and obesity ($BMI \geq 30 Kg/m^2$) was respectively 23.89% and 11.19% for the study population, 23.79% and 7.59% for men, and 28.8% and 21.2% for women. Abdominal obesity was present in 14% of men and 59.5% of women.[11]

Methods

MATERIAL & DETAILS OF EXPERIMENTAL STUDY:

1. Human volunteers:

a. This study was conducted on newly diagnosed cases of essential hypertension attending medical outdoor of M.G.M. Medical College, Kishanganj.

b. A complete clinical examination including laboratory investigation was done to exclude any systemic or other diseases which are likely to affect blood lipid levels directly or indirectly.

2) Group formation:

It contain 25 obese male and female hypertensive subjects with (Systolic B.P- 140-200 mm of Hg ,Diastolic B.P- 90-110 mm of Hg, B.M.I-Less than 25%)

Selection Criteria :

Inclusion Criteria :

1. Only Hypertensive subjects will be included.
2. Age- All subjects will be from 35-60 yrs age group.
3. Sex - Both male and female will be selected for the study.
4. Body weight - Body weight will be in kilograms.
5. Body Height - It will be in cms.
6. Body Mass Index (B.M.I.) - Body weight in kg Body height in m

Exclusion Criteria:

1) Other than hypertensive subject.

2) Any other disease which affect lipid profile

Study Period :- From November 2012 To April 2014.

Blood Collection: Blood samples analysis was done for total cholesterol, triglyceride, HDL-cholesterol,VLDL cholesterol, LDL cholesterol by using Fried Ewald's equation.

Results

SIGNIFICANCE OF DEMOGRAPHIC VARIABLES BY GENDER OF OBESE PATIENT

TABLE NO 1:- STATISTICAL SIGNIFICANCE OF DEMOGRAPHIC VARIABLE BY GENDER OF OBESE PATIENTS

Gender	Female (n=25		Male (n=25)		t	p
D. variable	Mean	SD	Mean	SD		
Age	49.16	8.42	44.36	7.52	1.90	>0.05
Ht	149.00	8.47	170.12	6.11	9.04	<0.001
Wt	64.24	7.80	79.92	8.51	6.06	<0.001
BMI	28.64	1.82	27.50	1.43	2.20	<0.05

Here again the mean age in the two sexes is similar and within significant difference (F=49.168.42years; versesM=44.36±7.52 years; verses M=44.36±7.52 years; p>0.5) the mean height is females and male are149.0±8.47as and 170.12±6.11 and respectively. The males are definitely and significantly taller (p<0.001).The mean weight in the two sexes is F: M=64.24±7.80kg vs 79.92± 8.51kg(p<0.001),proving again, that obese males are significant heavier than obese females. The BMI of the obese group of females is 28.69± 1.82 and that is obese male is 5.27±1.43.This is a bit paradoxical average heavier than female but the mean BMI of female is significant greater than that of male p<0.05.In our study WC in all category of male hypertensive is 96.34 ± 4.78 and which is higher than desired value (<95cm) but the WHR is 1.043 ± 0.096, which is well neither normal nor desired range (0.85 to 1.15). However ,the mean WHR in all categories of female is 1.169±0.125 which is a bit higher than ideally what is should have been.

COMPARISON OF SBP, DBP, WC, HC AND WHR BY GENDER OF HYPERTENSIVE PATIENT WITH OBESITY(TABLE NO 2)

TABLE NO 2:- COMPARISON OF BP, WC, HC & WHR BY GENDER OF OBESE HYPERTENSIVE PATIENTS.

Gender	Female (n=25		Male (n=25)		t	p
Parameter	Mean	SD	Mean	SD		
SBP	163.84	9.69	170.32	9.89	2.09	<0.05
DBP	96.32	3.15	99.60	4.73	2.58	<0.01
WC	106.52	5.72	105.96	4.59	0.28	>0.05
HC	83.20	4.70	93.32	5.09	6.53	<0.001
WHR	1.279	0.079	1.132	0.044	7.27	<0.001

In obese patients with hypertension, the mean SBP in female and male patients, in our study were 163.84± 9.69 mm of Hg and 170.32±9.89 mm of Hg respectively. The DBP were 96.32± 3.15 (F) mm of Hg and 99.60± 4.73 (M) mm of Hg. The sex difference is both the parameters (SBP & DBP) is statistically significant (p<0.05and<0.01 respectively). The WC, HC and WHR in female were106.52±5.72 cm, 83.2+4.7 cm and 1.279z0.079 respectively. The same parameters in male in our study were 105.96±4.59 cm, 93.32±5.09 cm and 1.132±0.044 respectively. In obese parameters, WC was not statistically significant (p>0.05) in two sexes but the HC&WHR were both statistically significant between two sexes (p<0.001 in both). When we studied all these parameters in all hypertensive patients disregarding wither they were obese or not, then in females the mean value were for SBP 162.32±8.78 mm *of* Hg , DBP95.72± 3.02mm of Hg, WC 93.14z14.49 cm, HC 79.26z6.15 cm and WHR1.169± 0.125. The same parameters in male shows:- SBP 167.20± 10.64cm,DBP98.60± 5.60mmofHg,WC96.34± 11.78cm, HC 92.72± 4.99 and WHR 1.043 ± 0.096 respectively. All these parameters shows statistically significant differences (<0.05,<0.05,<0.001 and <0.001) respectively.

When hypertensive patients are compared to normal, one of major difference is an increased prevalence of obesity[12,13].Again conversely , weight again appears

to be a main determinant of the raise in BP that is commonly seen in aging.[14] It was estimated that excess body weight accounted for 26% of cases of hypertension is men and 28% in women[15]. Again in one study it was found that there is statistically significant relation between hypertensive and CRP[16]. While in another study it was found that CRP was strongly associated with BMI[17].In short obesity might cause an increase in CRP which then cause an increase in hypertension or the vice versecan help also. Whichever might be time , it is supported by our study also, that both systolic and diastolic blood pressure are slight significantly raised in obese person (both male and female) as compared to non obese individuals. The females have however a greater propensity of this size.

LIPIDPROFILE OF OBESE HYPERTENSIVE INDIVIDUALS :-(TABLE NO -3)

TABLE NO 3:- COMPARISON OF LIPID PROFILE BY GENDER OF OBESE HYPERTENSIVE PATIENTS.

Gender	Female (n=25)		Male (n=25)		t	P
D. variable	Mean	SD	Mean	SD		
CHOL T	239.36	14.92	235.96	26.73	0.49	>0.05
HDL	35.00	7.36	28.60	3.77	3.46	<0.001
LDL	152.93	21.86	154.00	27.91	0.13	>0.05
VLDL	51.03	6.73	54.05	6.81	1.41	>0.05
TG	255.20	33.59	271.24	34.07	1.50	>0.05

In case of comparison of lipid profile by gender of obese hypertensive patients, the mean total cholesterol, HDL, LDL, VLDL and triglycerides in female are 239.36±14.92 mg/dl, 35.00±7.36 mg/dl, 152.93±21.86 mg/dl, 51.03±6.73 mg/dl and 255.20±33.5 mg/dl, respectively. The same value in case of male obese hypertensive in our study are 235.96±26.73 mg/dl,28.60±3.77 mg/dl,154.00±27.91 mg/dl,54.05±6.81 mg/dl and 171.24±34.07, respectively. The sexual differences in all these value are statically significant (p<0.05,<0.001<0.05<0.05, and <0.05 respectively). However the value are higher in case of male in regard to total cholesterol, LDL-Cholesterol, VLDL-cholesterol and triglycerides but lower only in case of HDL-cholesterol than females. While comparing the lipid profile of hypertensive patients between two gender, irrespective of whether they were found, it was found that the mean values of total cholesterol, HDL, LDL, VLDL and triglyceride in a case of female are 239.34±14.03 mg/dl,35.46±6.87mg/dl,161.26±20.94 mg/dl, 42.22±10.23 mg/dl and 211.10±51.15 mg/dl respectively. The same value in order in case of male hypertensive (both obese and non obese together) are: 249.08±24.84 mg/dl, 29.82±3.65 mg/dl, 175.90±30.57 mg/dl, 43.30±12.15 mg/dl and 219.02±61.11 mg/dl, respectively.

These value are statistically significant (p value being <0.05<0.001<0.01) in case of total cholesterol, HDL-cholesterol and LDL-cholesterol, male value being

greater in case of total and LDL-cholesterol but lesser in case of HDL-cholesterol, than the corresponding female values. However, the value of VLDL-cholesterol and Triglycerides are though greater in male than the corresponding female values, yet this difference are not statistically significant (the p value being >0.05).

A study conducted by Castelli WP and Anderson KA[18] in 1986 had supported that blood pressure and serum cholesterol are correlated with "r" factor of 0.12 suggesting that those with higher blood pressure values tend to have higher serum cholesterol in their famous Framingham heart study

Kaare & Bonna[19] in 1991 supported the view that in both sexes non - HDL cholesterol levels increased significantly with increasing systolic or diastolic blood pressure and total cholesterol increase with age in women but decreased with age in men. Smoking, physical activity and alcohol consumption had little influence on the association between blood pressure and lipid profile. However, no mention was made regarding the effect of adiposity on lipid profile in hypertensive's.

Chen Y.D I, Wayne and Arthur[20] in 1991 in their study found that mildly hypertensive patients appear to have faster catabolic rate of APO - AI / HDL and hence lower HDL - C values.

Rost and Devis (1996) in the systolic hypertension in the elder program (S HEP study)[21] found that total plasma cholesterol, LDL - C and ratios of LDL - C / DHL - C as well as TC - HDL - C were significantly high in men and women with hypertension and (AI).

In a study from Calcutta[22] it is found that total cholesterol in other hypertensive was 231.20 mg / dl (compared to ours 237.66 mg / dl) and LDL - C (our values 163.46 mg / dl) and HDL - C= 30.90 mg / dl (compared to our value = 31.88 mg / dl). These values very similar to our compared to the values obtained in Mangalore which may be due to lower geographical proximity racial, cultural and dietary resemblances between Bengal and Bihar compared to there of Mangalore (which is in extremes South India).

In short, our studies have shown that despite the well established fact that lipid profile shows a deterioration both hypertension and obesity (deterioration means ↑TC, LDL - C, VLDL - C & TGL and HDL - C), but when both obesity and hypertension are concomitantly present, then the lipid profile assumes a typical pattern (i.e. ↑ Triglycerides) compared to non-obese hypertensive individuals. Our net - reaches have not revealed the existence of any study so far matching the goals and methodology envisaged as in ours, that is no further deterioration due to obesity in hypertensive in ours, that is no further deterioration due to obesity in hypertensive in the values of TC & LDL - rather than a slight betterment of those, and vis- a-vis a significant and notable deterioration (i.e. Increase) in the values of VLDL - C and triglycerides.

The cause behind these peculiar changes are matters of further and broader research and can only be hypothesized in the present stage, viz

(a) The presentation of increased free fatty acids to liver as a function of obesity in primarily responsible for over production of VLDL as well as triglycerides.[23]

(b) Increased fat levels in obesity increased insulin level which is turn increases VLDL and triglycerides.[24,25]

(c) LDL in obesity mean decrease HDL - C and increase VLDL- C and Triglycerides[26]

(d) Obesity increased inflammatory marker (eg hscp) which increase VLDL - C and reduce HDL- C

The findings of our study have also important clinical implications. The association between dyslipidmia, obesity and hypertension is well established[27,28], and all have been found to be major risk factor for the development of eVD, a leading cause of visits to physician[29] and cause of death[30]. The peculiar lipid profile pattern in obese hypertensive compared to non - obese hypertensive will definitely change our way of looking at these complications will cause a paradigm shift in the prevention, treatment and deeper understanding of these morbid states.

Summary & Conclusion

Our study was envisaged to know the effect of obesity on lipid profile profile only in hypertensives and not in general population, and the study found some definite but paradoxical effects. These are that in obesity on a background of hypertension, the total and LDL cholesterol as also the HDL cholesterol are decreased, but on use other hand, the value of VLDL cholesterol and triglycerides are grossly and Significantly increased. These finding have two major Clinical implications in that obese hypertensives will be more prone to metabolic syndrome and type 2 diabetes mellitus, and steps should be taken to prevent them accordingly and also apart from statins one should treat the obese hypertensives with fibrates, fat restriction and physical exercise also. Our studies have been done in Kishanganj, Bihar which comprises a semi urban and rural population. So, a more elaborate and multicentric study covering all categories of population is required to be done to establish the findings more definitely and conclusively.

Ethical Clearence- Taken from institutional ethical committee, Department of Physiology PMCH , Patna

Source of Funding- Self

Conflict of Intrest- No

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