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RESEARCH ARTICLE

COMPARATIVE STUDY OF THE PRESENCE OF SYSTEMIC HYPERTENSION, DIABETES MELLITUS, DYSLIPIDEMIA AND OTHER CHRONIC DISEASES IN ASYMPTOMATIC OBESE AND NON- OBESE ADULTS-STUDY FROM NORTH INDIA.

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Manuscript Info

Abstract

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Introduction:-

Obesity is now well recognized as a disease in its own right. It is a major risk factor of many non-communicable diseases such as cardiovascular diseases (CVD), hypertension and diabetes mellitus (DM). The prevalence of overweight and obesity is increasing, and obesity is now estimated to be the second leading cause of preventable death after cigarette smoking in US¹.

In India, obesity is emerging as an important health problem particularly in urban areas, paradoxically coexisting with undernutrition². The rising prevalence of obesity in India has a direct correlation with the increasing prevalence of obesity related co-morbidities; hypertension, the metabolic syndrome, dyslipidemia, DM-2 and cardiovascular disease^{3,4}.

BMI is calculated as weight in kg divided by square of height in meters. BMI >25 kg/m² is taken as measure of generalized obesity as per WHO Asia Pacific guidelines⁵. In India, new diagnostic cut off for the BMI is 23kg/m² as opposed to 25kg/m² globally. A person with BMI of 23kg/m² will now be considered overweight and below as normal weight, unlike the cut off limit of 25kg/m².

Though BMI, WC, or waist hip ratio (WHR) correlate well with each other, it is also believed that combined use of these parameters of generalized and abdominal obesity may be better in identifying people at risk of CVD than either of them alone⁶⁻⁸.

According to consensus statement⁷,

- BMI is the most researched measure of generalized obesity and should continue to be used using Asian Indian specific cut offs as described.
- WC should be used as a measure of abdominal obesity.
- Both BMI and WC should be used together (with equal importance) for population and clinic based metabolic and cardiovascular risk stratification⁹.

High prevalence of insulin resistance is due to excess body fat and abdominal adiposity. Although exact reasons are still not clear, certain unique clinical and biochemical characteristics of this ethnic group collectively called as "the Asian Indian phenotype" is considered a major factor contributing to the increased predilection for diabetes^{11,12}. Moreover they have lower levels of protective adipokines, adiponectin and have increased levels of

adipose tissue metabolites¹³. Asian Indians have more total abdominal and visceral fat for any given BMI and for any given body fat, they have increased insulin resistance^{14,15}.

High blood pressure is an unidentified physiological disturbance which leads ultimately to elevation of diastolic blood pressure (DBP) and systolic blood pressure (SBP), anatomic change in vascular free and functional impairment of involved tissue. Most epidemiological studies show that obesity is a strong risk factor for hypertension^{16,17}. As early as 1987 while studying hypertension precursors in young adults, Framingham researches demonstrated that 78% of hypertension cases in males and 65% of hypertension cases in females could be a direct result of obesity¹⁸.

The most characteristic lipid disorders in obesity are elevated total cholesterol (TC) and triglycerides (TG), high low density lipoprotein cholesterol (LDL-C) and low high density lipoprotein cholesterol (HDL-C)¹⁹. Previous studies have demonstrated that an increased BMI is associated with reduction in LDL particle size^{20,21}.

Material and Methods:-

The proposed study was carried out through camps held in various urban and sub-urban areas of Amritsar city. The research scholars explained the purpose of study to the individuals and assured that the information was used only for the study and will be kept strictly confidential. Informed consent was taken from the individuals before his or her inclusion in the study. A total of 1000 asymptomatic adults between the age group 35 to 55 years were studied. They were divided in two groups:

Group I : 500 asymptomatic obese adults.

Group II : 500 asymptomatic non-obese adults.

Exclusion criteria:

- a) Known diabetics
- b) Known hypertensives
- c) Coronary artery disease
- d) Renal failure
- e) Cirrhosis and other liver disorders
- f) Endocrine disorder or any other significant illness
- g) The patients on treatment with antihypertensives, antiobesity and hypolipidemic drugs

A detailed questionnaire using suggestions laid down by WHO, was used and details of history and physical findings were recorded on a performa. All individuals between 35-55 years furnished the information and were investigated as per performa. The questionnaire included general questions on health, diet, smoking, drinking habits, lifestyle.

Following obesity parameters were recorded:

1. Weight in kilograms on standard weighing scale.
2. Height in meters using standard measuring tape.
3. Waist circumference in centimeters.
4. Hip circumference in centimeters.

After an overnight fast (12 hours) using an indwelling cannula inserted in the antecubital vein, blood was withdrawn for fasting plasma glucose and lipid profile.

BMI was calculated as weight in kg divided by square of height in meters. $BMI \geq 23 \text{ kg/m}^2$ was taken as measure of generalized obesity as per WHO Asia Pacific guidelines for Indians⁵ (new guidelines for Indians) In accordance with BMI, classification is as under:

Classification	WHO Asia Pacific guidelines	WHO Asia Pacific guidelines for Indians
Underweight	<18.5	<18.5
Normal range	18.5-24.9	18.5-23.0
Overweight	>25.0	>23.0
Preobese	25.0-29.9	23.0-24.9
Obese Class I	30.0-34.9	25.0-30.0
Class II	35.0-39.9	>30
Class III	>40.0	

Waist and hip ratio:

Waist and hip circumferences was measured in the standing position. Waist was measured as the smallest horizontal girth between the costal margin and the iliac crest. The hip circumference was taken at the level of greater trochanter. The ratio more than 1.0 in males and 0.85 in females indicated abdominal fat accumulation²². The waist circumference more than 90 cm in males and 80 cm in females was taken as abnormal as per WHO Asia Pacific guidelines⁵.

Investigations :

All selected subjects underwent an overnight fast of approximately 10 - 12 hours. In the morning fasting blood samples were taken for :

- plasma glucose
- lipidogram (TC, HDL and TG)

Serum Lipid estimation:

The abnormalities were recorded as by National Cholesterol Education Programme²³.

Glucose estimation (Glucose oxidase method):

Plasma glucose was measured on the same day of sample collection by glucose oxidase method.

Statistical Analysis:-

Data generated from the study was analysed according to standard statistical methods. Results were tabulated in the form of mean + S.D. and analysed using students' 't' test and the level of significance was determined as its p value with $p < 0.05$ taken as statistically significant and $p < 0.001$ taken as highly significant. $P > 0.05$ was taken as statistically not significant.

Multiple comparisons were done by post Hoc tests using bonferroni to find prevalence of hypertension, diabetes, dyslipidemia among asymptomatic obese male, obese female, non-obese male and non-obese females of Amritsar city and using Tukey HSD to find the trend according to BMI categories.

Results:-

The present study was carried out in Amritsar city, enrolling 1000 asymptomatic adults, aged 35-55 years, 500 obese and 500 non-obese. The population was studied for association of systemic hypertension, diabetes mellitus and dyslipidemia with obesity, using BMI, in particular. Asymptomatic non-obese adults were also compared for the presence of systemic hypertension, diabetes mellitus and dyslipidemia. The analysis was performed separately for men and women.

I. DISTRIBUTION OF POPULATION ACCORDING TO AGE

In our study population, out of 500 obese, 283 were in age group 35-45 (56.6%) and 217 were in age group 46-55 yrs (43.4%). Out of 500 non-obese, 271 (54.2%) were in age group 35-45 yrs and 229 (45.8%) in age group 46-55 yrs, with mean age in non-obese group 44.70 ± 5.932 yrs and obese group 44.73 ± 5.748 yrs (Chi square 0.445).

II. DISTRIBUTION OF POPULATION ACCORDING TO SEX

In our study population, out of 500 obese, 331 (66.2%) were males and 169 (33.8%) females. Out of 500 non-obese, 324 (64.8%) were males and 176 (35.2%) females (Chi square 0.641).

On comparing obese and non-obese group, significant linear relationship of obesity with weight ($p < 0.001$), BMI ($p < 0.001$), TC ($p < 0.001$), TG ($p < 0.001$), LDL-C ($p = 0.003$), HDL-C ($p = 0.000$), VLDL-

C(p<0.001), non-HDL-C(p<0.001), FBS(p<0.001), avSBP(p<0.001), avDBP(p<0.001) was observed, but not with WC(p=0.5500, HC(p=0.517), WHR(p=0.992), as depicted in table 3 and figure 3:

Parameters	Non-Obese (n=500)		Obese (n=500)		P-Value
	Mean	SD	Mean	SD	
Height	163.46	8.798	162.41	8.63	0.055
Weight	60.96	8.29	76.44	10.8	<0.001*
Body mass index(BMI)	22.53	2.08	28.92	3.41	<0.001*
Waist circumference(WC)	87.42	10.2	87.03	10.33	0.550
Hip circumference(HC)	91.25	11.177	90.80	10.83	0.517
Waist hip ratio(WHR)	0.9602	0.0566	0.9602	0.05874	0.992
Total cholesterol(TC)	222.69	42.718	231.76	35.97	<0.001*
Triglycerides(TG)	156.94	34.63	167.34	29.73	<0.001*
LDL- C	141.19	36.87	147.78	31.72	0.003*
HDL – C	41.25	10.17	38.85	10.55	0.000*
VLDL- C	31.21	6.74	33.54	5.97	<0.001*
Non-HDL-C	181.44	35.978	192.91	25.42	<0.001*
Fasting blood sugar(FBS)	99.75	28.19	107.69	33.64	<0.001*
AvSBP	121.83	10.58	140.65	15.97	<0.001*
AvDBP	81.53	6.08	88.69	2.85	<0.001*

Table 3:- Comparison of different parameters between obese and non-obese group

These obese and non-obese groups were further divided according to gender into 4 subgroups:- Non-obese male, Non-obese female, Obese male, Obese female. We observed that similar relations were observed between different parameters among obese males and females.

While on comparing non-obese males and non-obese females, significant difference of height (p<0.001), weight(p<0.001), LDL-C(p=0.005), non-HDL-C(p=0.05) in males as compared to females was observed. Females showed significantly higher HDL-C.

On evaluating our study population according to BMI, significant linear relationship of BMI with weight (p=0.000), WC (p=0.000), HC(p=0.000) was observed, but not with WHR(p=0.174), as depicted in table 4 and figure 4:

Body mass index	Sex		Age	Height	Weight	Body mass index	Waist circumference	Height circumference	Waist hip ratio
	Male	Female							
<18.5	23	10	45.61± 5.396	164.7± 7.75	47.35±-5.16	17.41± 0.93	88.94± 10.4	92.43± 11.34	0.96± 0.05
18.5-23.0	308	159	44.64± 5.968	164.27± 8.42	61.9± 7.63	22.88± 1.60	87.31± 10.1	91.16± 11.17	0.96±-0.05
23.0-24.9	224	129	44.80± 5.676	163.23± 8.92	72.56± 8.76	27.16± 1.39	77.82± 24.9	77.43± 29.31	0.95± 0.05
25.0-30.0	69	26	44.60± 5.819	161.18± 8.19	80.94± 8.93	31.06± 0.96	95± 81.18	74.50± 27.77	0.96± 0.05
30.0-39.9	27	18	44.76± 6.0	158.29± 6.044	91.26± 8.24	36.31± 1.54	77.36± 23	76.92± 25.44	0.98± 0.06
>40	4	3	42.71± 7.25	156.57± 5.76	99.67± 6.37	40.55± 0.414	76.15± 29.48	66.35± 38.75	0.95± 0.04
p-value	0.56		0.879	0.543	0.000*	0.000*	0.000*	0.000*	0.174

Table 4:- Population demographics based on BMI categories.

V. PREVALENCE OF RISK FACTORS BASED ON BMI CATEGORIES

On evaluating various risk factors according to BMI, significant linear relationship of BMI with calorie intake ($p=0.000$), TC ($p=0.000$), TG ($p=0.000$), LDL-C ($p=0.000$), HDL-C ($p=0.000$), VLDL-C ($p=0.000$), non-HDL-C ($p=0.000$), FBS ($p=0.004$), avSBP ($p=0.000$), avDBP ($p=0.000$) was observed, as depicted in table 5 :

Body mass index	Calorie intake	TC	TG	LDL-C	HDL-C	VLDL-C	Non-HDL-C	FBS	avSBP	avDBP
<18.5	1061.0±2 62.0	250.6± 34.0	176.03±2 5.58	149.70±3 4.3	40.21± 9.79	35.20± 5.11	200.02± 5.11	100.52±4 8.5	117.73±6 9.0	79.67± 4.18
18.5-23.0	1594.9±3 20.0	227.0± 42.6	155.59±3 4.8	140.59±3 7.0	40.43± 9.86	30.92± 6.67	186.57± 6.67	99.70± 26.2	122.13±1 0.7	81.67± 6.818
23-24.9	1621.49± 364.0	233.08±3 4.77	167.56±2 7.8	146.39±3 3.0	40.33± 11.02	33.53± 5.57	199.08± 5.57	108.10±3 4.9	139.46±1 4.98	88.42± 8.4
25.0-30.0	1619.65± 380.0	227.6± 37.3	165.39±2 7.8	147.78±3 3.9	38.72± 10.6	33.31± 5.67	188.88± 5.67	106.18±2 9.2	142.69±1 6.26	89.27± 9.47
30.0-39.9	2189.0± 241.0	234.13±3 9.0	171.6± 42.0	139.47±3 6.5	39.13± 11.4	34.46± 81.0	199.67± 36.5	109.36±3 4.8	148.84±1 7.0	88.87± 9.18
>40	2437.0± 0.00	205.71±4 4.31	155.0± 42.2	146.00±3 9.14	34.57± 9.09	31.11± 9.8	200.61± 9.8	96.8± 7.3	143.86±1 7.62	92.14± 14.33
p-value	0.000*	0.000*	0.000*	0.003	0.000	0.000*	0.000*	0.004*	0.000*	0.000*

Table 5:- Prevalence of risk factors based on BMI categories.

VI. PREVALENCE OF ALL THE THREE CONDITIONS IN OBESE AND NON-OBESE GROUP (DIABETES, HYPERTENSION, DYSLIPIDEMIA)

In our study population, out of 500 obese, 28 (5.6%) had all three conditions and out of 500 non-obese, 1 (0.2%) had all three conditions, with total prevalence 2.9%. Thus a significant correlation of all three conditions was found with obesity ($p=0.001$), as depicted in table 6 :

ALL THE THREE CONDITIONS	Group				Total	
	Obese		Non-obese			
	No.	%age	No.	%age	No.	%age
No	472	94.4	499	99.8	971	97.1
Yes	28	5.6	1	0.2	29	2.9
Total	500	100.0	500	100.0	1000	100.0

$p=0.001$

Table 6:- Prevalence of all the three conditions in obese and non-obese group (diabetes, hypertension, dyslipidemia)

VII. PREVALENCE OF ALL THE THREE CONDITIONS IN RELATION TO BMI CATEGORIES

While according to BMI, a similar correlation was found between all three conditions and increasing BMI ($p=0.001$), as depicted in table 7 :

			Body Mass Index (kg/m ²)						Total
			<18.5	18.5 - 23.0	23.0 - 24.9	25.0 - 30.0	30 - 39.9	>=40	
ALL THE THREE CONDITIONS	No	Count	33	466	340	87	40	5	971
		% within Body Mass Index (kg/m ²)	100	99.8	96.4	91.6	88.9	71.4	97.1
	yes	Count	0	1	13	8	5	2	29
		% within Body Mass Index (kg/m ²)	0	0.2	3.6	8.4	11.1	28.6	2.9
Total		Count	33	467	353	95	45	7	1000

$p=0.001$

Table 7:- Prevalence of all the three conditions in relation to BMI categories**VIII. RELATION OF ANY OTHER DISEASE WITH OBESITY**

In our study population, out of 500 obese, 292 (58.4%) had any other disease and out of 500 non-obese, 98 (19.6%) had any other disease, with total prevalence 39.0%. Thus a significant correlation of any other disease was found with obesity ($p < 0.001$). Also a significant correlation was found between any other disease and increasing BMI ($p < 0.001$).

IX. RELATION OF SMOKING WITH OBESITY

In our study population, out of 500 obese, 81 (16.2%) smoked and out of 500 non-obese, 68 (13.6%) smoked, with total prevalence 14.9%. (p value 0.248).

X. RELATION OF ALCOHOL INTAKE WITH OBESITY

In our study population, out of 500 obese, 126 (25.2%) took alcohol and out of 500 non-obese, 91 (18.2%) took alcohol, with total prevalence 21.7%. (p value 0.007).

XI. RELATION OF PHYSICAL ACTIVITY WITH OBESITY

Our study population was divided according to their physical activity into sedentary, moderate and heavy workers. Prevalence of obesity increased significantly with decreased physical activity i.e., in sedentary workers (P value < 0.001).

XII. RELATION OF DIET (VEGETARIAN/NON VEGETARIAN) WITH OBESITY.

In our study population, out of 500 obese, 208 (41.6%) were vegetarian and 292 (58.4%) non vegetarian and out of 500 non-obese, 402 (80.4%) were vegetarian and 98 (19.6) non vegetarian with total prevalence 39% of vegetarian and 61% of non vegetarian. (p value < 0.001).

Discussion:-

The present study was carried out in Amritsar city to find out association of obesity, as assessed by anthropometric index of BMI, with systemic hypertension, diabetes mellitus and dyslipidemia.

The prevalence of dyslipidemia was 44% in obese group as compared to 20% in non-obese group. Its prevalence increased with increasing BMI, from 21.4% with BMI 18.5-23.0 to 44.4% with BMI > 40 , with overall prevalence 32% (p value 0.001), TC, TG, LDL-C, VLDL-C, non HDL-C increased with increasing BMI and HDL-C decreased (p=0.000). These results were similar to those of SHIELD survey²⁴, which showed prevalence of dyslipidemia from 17.9% at BMI 18.5-24.9 to 35.9% with BMI > 40 , overall prevalence 25.8%. In a study by Lawati JA²⁵, low HDL-C was the most common abnormalities in the study population with 75.4% of individuals and hypertriglyceridemia was 20%.

In our study, serum total cholesterol levels in obese were 231.76 ± 35.79 mg/dl as compared to non obese, 222.69 ± 42.718 mg/dl. (p < 0.001). Serum TC levels were significantly higher in obese females (232.44 ± 33.91 mg/dl) as compared to obese males (225.39 ± 37.09 mg/dl) with p value 0.05. Similar results were seen in a study by Brown, et al²⁶ where mean serum TC levels increased with increasing BMI ranging from (193-211 mg/dl) among men and in women between (217-219.5 mg/dl). Among men, the prevalence of high TC ranged from 13% to 22% while in women, the prevalence of high TC ranged from 13% to 27%. The minor differences in the average values of lipid variables could be accounted for by the differences in ethnicity, lifestyles, diet patterns and social environment of the population under study.

In our study, HDL-C significantly decreased from 40.06 ± 10.98 mg/dl in non obese group more so in non obese males (p=0.000) as compared to 38.85 ± 10.55 mg/dl in obese group (p value 0.000). In obese males HDL-C levels were significantly lower i.e., 37.85 ± 9.975 mg/dl in obese males as compared to 40.06 ± 10.98 mg/dl in obese females. (p=0.023). These results were similar to study done by Brown, et al³¹ which showed mean levels of HDL-C ~ 10 mg/dl higher in women than in men and they decreased with increasing BMI in both sexes.

In our study, serum triglyceride levels increased from 156.94 ± 34.63 mg/dl in non obese group to 167.34 ± 29.73 mg/dl in obese group which was significantly higher (p < 0.001). Increase in TG levels was significantly higher in obese males (170.99 ± 32.29 mg/dl) as compared to obese females (167.17 ± 21.159 mg/dl) (p= 0.05). While a study by Cercato C, et al²⁷ higher levels of TG and reduced HDL-C levels were associated

with obesity. With increasing BMI, TG levels increased from 120.9 ± 78.7 mg/dl at BMI 18.5-24.9, with odds ratio to 158.8 ± 96.9 mg/dl at BMI >40 ($p < 0.02$) levels, with odds ratio 2.6. Levels of HDL-C decreased from 52.6 ± 13.3 mg/dl at BMI 18.5-24.9 to 45.1 ± 10.1 at BMI >40 . However, the study did not reveal any association between elevated levels of TC or LDL-C and obesity. The contrast in results in our study and the study done by Cercato C, et al²⁷ could be attributed to ethnicity, lifestyles, diet patterns and social environment of the population under study. Moreover, Asian Indians are noted to have adverse lipid profile, having higher TG, lower TC, LDL-C, HDL-C compared to others. The mean levels of LDL-C in obese group (147.78 ± 31.72 mg/dl) was significantly higher than non obese group (141.19 ± 31.72 mg/dl) ($p = 0.003$), more so in non obese females in non obese group.

The prevalence of high TC, LDL-C and TG and a high TC/HDL-C ratio and low HDL-C increased steadily in line with blood pressure, BMI, waist circumference, waist hip ratio and fasting blood glucose ($p < 0.001$). In a study done by Humayun, et al²⁸ in Peshawar, in females, dyslipidemia showed a gradual increase with age for all BMI categories.

In our study, non HDL-C levels increased with increasing BMI ($p = < 0.001$). It was significantly higher in obese males (193.54 ± 27.12 mg/dl) as compared to obese females (192.38 ± 22.93 mg/dl) (p value = 0.04).

In our study, 34.8% of obese and 19.2% of non obese were diagnosed as hypertensives. Average SBP of obese females was 141 ± 16.51 mmHg as compared to non obese females 121.2 ± 10.39 mmHg. Average SBP of obese males was 140.96 ± 15.29 mmHg as compared to non obese males 122.16 ± 10.68 mmHg. Average DBP of obese females was 82.16 ± 8.906 mmHg as compared to non obese females 80.31 ± 5.97 mmHg. Average DBP of obese males was 89.23 ± 8.68 mmHg as compared to non obese males 82.16 ± 6.05 mmHg. Thus both SBP and DBP were significantly higher in obese group ($p < 0.001$). Also prevalence of high blood pressure increased progressively with increasing BMI of 15.2% with BMI < 18.5 to 42.9% with BMI > 40 . ($p < 0.001$). Overall prevalence was 27%. These results were similar to those by SHIELD survey²⁴ which showed overall prevalence 23.4%, and 13.2% with BMI 18.5-24.9 to 40.5% with BMI > 40 .

In our study, a positive correlation was found between DM and obesity. Out of total 500 non obese, 74 were detected diabetic i.e., 14.8% as compared to 108 obese i.e. 21.6% ($p < 0.005$). Prevalence of DM also increased with increasing BMI with 15% prevalence at BMI 18.5-23.0 to 42.9% with BMI > 40 (p value 0.005). Overall prevalence was 18.2%. This was higher than found by SHIELD survey²⁴, where it was 8.2%, prevalence of DM increased from 3.5% with BMI 18.5-24.9 to 25.1% with BMI > 40 . This may be explained as prevalence of DM is much more in India as compared to West. Moreover, the Asian Indian phenotype is considered the major factor contributing to the increased predilection for diabetes in India^{11,12}. This was similar to results seen in study done by Cercato C, et al²⁷ in Brazilian population which showed positive correlation of BMI with DM ($p < 0.001$) with odd's ratio 3.8, 5.8 and 9.2 with increasing BMI.

In our study, FBS increased with increasing age, with 103.68 mg/dl in age group 35-45 years and 103.78 mg/dl, in age group 55-65 years. Similar results were shown by Shmulewitz, et al²⁹. Individuals more than 50 years of age had a 35-fold increased risk of DM compared to those 20-34 years of age and a 3.1 fold increased risk compared to 35-45 years old ($p < 0.001$).

In our study, impaired FBS was more in males (110.56 ± 35.58 mg/dl) as compared to females (109.78 ± 37.82 mg/dl) which was similar to study done in Kerala by Vijay kumar G, et al³⁰, which showed proportions of newly detected diabetics among men, 2.7% and among women, 2.5% respectively. The minor differences in the mean values of FBS could be accounted for by the differences in ethnicity and diet pattern our study group.

Also there was correlation of hypertension, hypercholesterolemia, low physical activity, non vegetarian diet with DM in our study similar to seen in study by Vijay Kumar G, et al³⁰. In our study, overall prevalence of all the three conditions was 2.9%. The prevalence of all the three conditions in obese group was 5.6% as compared to non obese group where it was 0.2%. Also statistically positive correlation was seen in prevalence of all the three conditions and increasing BMI, it was 0.2% with BMI 18.5-23.0 to 28.6% with BMI > 40 . (p value 0.001). These results were similar to SHIELD survey²⁴ from 1999-2002, prevalence of all the three conditions increased from 0.9% with BMI 18.5-24.9 to 11.6% with BMI

>40. Overall prevalence of all the three conditions was 3.3%. Tao T, et al³⁵, also showed obesity was significantly associated with a history of DM 18% vs 7%, $p<0.05$; hypertension 48% vs 28%, $p<0.05$; hypertriglyceridemia 2.67 ± 1.95 mmol/L vs 1.86 ± 0.95 mmol/L, $p<0.05$.

In retrospective study by Cercato C, et al²⁷ on Brazilian population, the prevalence of systemic hypertension, DM, hypertriglyceridemia and low HDL-C increased along with weight, but the prevalence of hypercholesterolemia did not. The odds ratio adjusted for gender and age, according to grade and obesity compared with patients with normal weight were respectively 5.9, 8.6 and 14.8 for systemic hypertension, 3.8, 5.8 and 9.2 for DM and 1.2, 1.3 and 2.6 for hypertriglyceridemia.

Overall mean lipid levels of TC, LDL-C showed a statistically significant association with hypertension. No significant association was noticed in HDL-C level. These results were similar to those found in study by Pramila Devi R, et al³¹.

In our study, there was positive correlation of abnormal glucose with elevated TG and low HDL-C. Thus non HDL-C was higher in patients with abnormal glucose metabolism as compared to normal glucose metabolism. These were associated with the risk of cardiovascular disease. These results were in time with those in Hoorn study³². Hypertension is a common problem among diabetes patients, accelerating progression of vascular diabetic complications. Thus diabetes and hypertension frequently co-exists. These results are in time with study done by Mancia G³³ and Simonson DC³⁴.

In our study, prevalence of any other disease in obese group was 58.4% as compared to non obese group of 19.6%. Thus its prevalence increased with increasing BMI, 20.4% with BMI 18.5 to 24.9 to 66.7% with BMI >40. Overall prevalence was 39%. These results were similar to those in SHIELD survey²⁴, which showed prevalence of any other disease to be 26.2% with BMI 18.5-24.9 to 62% with BMI >40, with overall prevalence 38.9%. ($p=0.001$).

In our study, smoking did not correlate with obesity. Out of 500 obese population, 81 smoked, with prevalence 16.2% as compared to non obese, 68 smoked with prevalence 13.6% ($p=0.248$). These results are consistent with study done by Shmulewitz, et al²⁹ which showed smoking was associated with a decreased risk of obesity and diabetes, consistent with previous studies showing that the effect of smoking on obesity is the basis of the decreased risk of diabetes in smokers.

In our study, alcohol intake significantly correlated with obesity ($p=0.007$). In obese, out of 500 adults 25.2% took alcohol compared to non obese in whom 18.2% took alcohol. These results were similar to those in study by Tao T, et al³⁵.

In our study, out of 500 obese, 65.2% of obese were sedentary workers, 33.8% moderate and 1% heavy workers as compared to non obese in whom 62.8% were sedentary, 31.8% moderate and 5.4% heavy worker ($p<0.001$). Thus more physical inactivity was present in obese. The results were similar to those seen in study by Tao T, et al³⁵. In our study, higher caloric intake with non vegetarian diet was associated with increasing body mass index ($p<0.001$). These results were similar to those of study by Tao T, et al³⁵. Change in fat intake was positively associated with change in body mass index in men.

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