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Original Article

EVALUATION OF LIPID PROFILE AND LIVER FUNCTION PARAMETERS IN OBESE AND NON-OBESE HYPERTENSIVE INDIVIDUALS ATTENDED TERTIARY CARE TEACHING HOSPITAL

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ABSTRACT

Objective: In this study, lipid profile parameters and liver function parameters were measured and correlated in hypertensive obese and non-obese patients who were attended a tertiary care teaching hospital.

Methods: This study is a retrospective observational, cross-sectional study over the course of a year in a tertiary care hospital. All adult patients with hypertension, both obese and non-obese, are included in the study population. The study involved 150 participants in total, including obese and non-obese people (71 men and 79 women). Standard techniques were used to assess the serum levels of TG, TC, LDL, HDL, and liver enzymes such SGOT, SGPT, bilirubin, and ALP. Moreover, BMI was calculated for both study groups. The association between elevated lipid profile markers and liver enzyme abnormalities was assessed by correlation analysis.

Results: Out of 150 samples size comprises 75 obese and 75 non-obese individuals, 71 males and 79 females were reported in the current study. Our study gives very good association between lipid profiles to almost all LFT and the highest is shown between HDL, LDL and VLDL to all LFT. There was a high significant difference was noticed between the obese and non-obese individuals in relation to bilirubin (D) concentration, SGOT and SGPT between the two groups $P<0.0001$. The levels of albumin were lowered and the significant variations were noticed between the two groups. The mean values of cholesterol, LDL and triglycerides were very high in obese individuals than non-obese individuals and the HDL was lower in obese individuals.

Conclusion: The study found that liver function variables and abnormal lipid profiles were highly prevalent in obese hypertension patients. In participants with dyslipidemia, increased liver enzymes were seen more frequently.

Keywords: Dyslipidemia, Lipid profile, Hypertension, Obese, Non-obese

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INTRODUCTION

Obesity is becoming more and more prevalent, approaching epidemic levels at an alarming rate in both developed and underdeveloped countries worldwide. The majority of research investigations have revealed that the prevalence of overweight and obesity among urban residents ranges between 30% and 65%. Obesity and overweight are primarily brought on by unhealthy eating patterns, less physical activity, and genetic hereditary. May be the most common form of malnutrition is obesity [1, 2]. Body mass index (BMI) is a commonly used categorization metric. For overweight persons the BMI will be 25 kg/m^2 and for obese individuals BMI is 30 kg/m^2 in men and women. In most clinical settings, BMI is quite useful and has a good correlation with laboratory-based measurements of adiposity for population studies. Because obesity is not a single disease and the results of long-term follow-up on a broad scale are not yet accessible in the literature, studying obesity is challenging. The knowledge that is currently available merely represents a cross-section of the population. According to recent statistics, 650 million persons worldwide are fat and nearly 1.9 billion are overweight. Obesity-related fatalities have been estimated to reach 2.8 million in number. More than 135 million people in India suffer from obesity. Age, gender, location, socioeconomic position, and other factors affect the prevalence of obesity in India [3]. The developing world faces a significant risk of obesity and its negative effects due to the intake of energy-dense food (i.e., poor eating habits), a sedentary lifestyle, a lack of health care facilities, and financial constraints (i.e. diabetes, hypertension, dyslipidemia, ischemic heart disease, etc). The majority of individuals also have metabolic disorder like fatty liver [4-6]. A reversible, acquired metabolic disease known as fatty liver causes the activation of lipolysis, which leads to a buildup of triglycerides inside the hepatocytes. The metabolic syndrome is known to have a significant component called fatty liver, and its significance has lately grown. Pregnancy, total parenteral hyper alimentation, severe hepatitis, glycogen storage disease, jejunoileal bypass procedures for obesity, cystic fibrosis, congenital generalized lipodystrophy, excessive alcohol intake, poorly controlled hyperlipidemia, diabetes, excess exogenous or endogenous corticosteroids, and toxins like carbon tetrachloride and yellow phosphorus. It is now understood that in some people, fatty infiltration of the liver is the forerunner to serious chronic illness and hepatocellular cancer. It is known that an increase in triglyceride stores is associated with a linear increase in the production of cholesterol, which in turn is associated with an increase in cholesterol secretion in bile and an increased risk of gallstone formation and the emergence of gall bladder diseases. However, the precise biochemical mechanisms underlying the association between obesity and the aforementioned diseases have not yet been fully elucidated. Similar to this, higher levels of circulating triacylglycerol in obesity are linked to lower levels of high-density lipoprotein, which may indicate the obese people are at a greater risk for cardiovascular disease and heart attacks [7-9]. The study aims to measure and correlate the values of lipid profile parameters and liver function parameters in hypertensive obese and non-obese individuals attended tertiary care teaching hospital.

MATERIALS AND METHODS

The present study is a retrospective observational, cross sectional study conducted for a period of one year in a tertiary care teaching hospital. The study population includes all the adult Obese and non-obese patients suffering from hypertension. Cochran's formula

which considering 95% level of significance at 5% precision level and prevalence of dyslipidaemia is 30% used for sample size calculation. The sample size is 150 comprises 75 obese and 75 non-obese individuals.

Inclusion criteria

- Patients of age>18 y with hypertension.
- Patients who gave informed written consent.

Exclusion criteria

- Liver cirrhosis and other liver diseases patients
- Patients taking lipid lowering drugs.
- Pregnant individuals

Methodology

BMI was calculated as weight (kg)/height (m^2). Diazo transfer reaction was used for the measurement of bilirubin in patient serum samples in the present study. Bilirubin reacts with diazotized sulfanilic acid in acidic medium to form azobilirubin, a pink colored complex whose absorbance is proportional to bilirubin concentration. Direct bilirubin, being water soluble is allowed to react with diazotized sulfanilic acid in the absence of an activator, while for total bilirubin (Direct and Indirect) the diazotization is carried out in the presence of an activator. The ranges of direct bilirubin is 0.0-0.2 mg % and total bilirubin is 0.2-1.0 mg %. SGOT (Serum glutamic oxaloacetic transaminase) and SGPT (Serum glutamate pyruvate transaminase) are two of the most common enzymes produced by the liver. Kinetic UV method is used for determination. The normal value of SGPT is<40 U/l and SGOT is<37 U/l. The procedure involves the addition of working reagent 1 ml to 0.1 ml of sample. Mix and after 1 min incubation, measure the change of optical density per minute. Alkaline phosphatase (ALP) activity has been measured by many methods differing in substrate, buffer type, buffer concentration, time and temperature of incubation and unit of measurement. ALP Kit is based on the recommendations of the International Federation of Clinical Chemistry (I. F. C. C.). This method utilizes p-nitrophenylphosphate (p-NPP) as the substrate and the 2-amino-2-methyl-1-propanol buffer (AMP) ensure a high catalytic activity of ALP. Serum ALP hydrolyzes p-NPP into yellow colored p-nitrophenol (p-NP) at an alkaline pH. The rate of p-NP formation is directly proportional to the ALP activity and is measured in terms of change in absorbance at 405 nm. The normal value in adults is 40-120 U/l. Albumin is measured by photocolorimetric method. Albumin in a buffered medium binds with bromocresol green (BCG) and produce a green color whose absorbance at 630 nm is proportional to the albumin. Estimation of Lipid profile parameters (total cholesterol, HDL-cholesterol, triglyceride, LDL, VLDL) were analyzed using Cobas analyzer. The Cholesterol High Performance reagent is used to measure cholesterol enzymatically. Using reagents from the same manufacturer, triglycerides and cholesterol are both measured enzymatically in parallel. The same reagent is used to test triglycerides in blank samples, but lipase is not present. The Roche Diagnostics direct HDL-cholesterol reagent is used to measure cholesterol and triglycerides simultaneously. Similar analyses were carried out to determine LDL and VLDL levels. Laboratory variables outside of the normal parameters were defined as: total cholesterol (>200 mg/dl), HDL cholesterol (<40 mg/dl), LDL cholesterol (>130 mg/dl), triglycerides (>150 mg/dl) and VLDL (5 to 40 mg/dl).

Statistical analysis

To determine the significance between the two groups obese and non-obese, a student paired t-test will be utilized. All of the various variables were examined for relationship using Pearson's correlation. P values <0.05 were regarded as significant.

RESULTS

Out of 150 samples size comprises 75 obese and 75 non-obese individuals, 71 males and 79 females were reported in the current study. The number of females was more than males (fig. 1). There was no significant difference between genders in the observed associations. Table 1 depicts the mean values and significant variations in the mean values of lipid function and liver function variables between the obese and non-obese individuals. Hypertension is generally prone to elevation in altered lipid metabolism and it is handled by liver, a relationship between lipid profile and LFT must exist. Our study gives very good association between lipid profiles to almost all LFT and the highest is shown between HDL, LDL and VLDL to all LFT. When bilirubin (T) concentration between the obese and non-obese were compared the results showed insignificant variations P=0.6 (table 2). There was a high significant difference was noticed between the obese and non-obese individuals in relation to bilirubin (D) concentration, SGOT and SGPT between the two groups P<0.0001 (table 2). When alkaline phosphatase concentration between the obese and non-obese were compared the results showed insignificant variations P=0.9 (table 2). The levels of albumin were lowered and the significant variations were noticed between the two groups. The concentrations of SGOT, SGPT, total bilirubin, alkaline phosphatase were significantly high between the obese and non-obese individuals were identified in the present study (table 2).

Further dyslipidemia is also linked to LFT and our study findings are supported by previous observations. The mean values of cholesterol, LDL and triglycerides were very high in obese individuals than non-obese individuals and the HDL was lower in obese individuals (table 2). All the lipid parameters were significantly P<0.0001 among the hypertensive obese individuals comparatively than non-obese individuals. This indicates that they are the key influential variables in the elevation of hypertension. However the pearson's correlation showed insignificant influence of lipid parameters and liver function variables between the obese and non-obese hypertensive individuals (table 3).

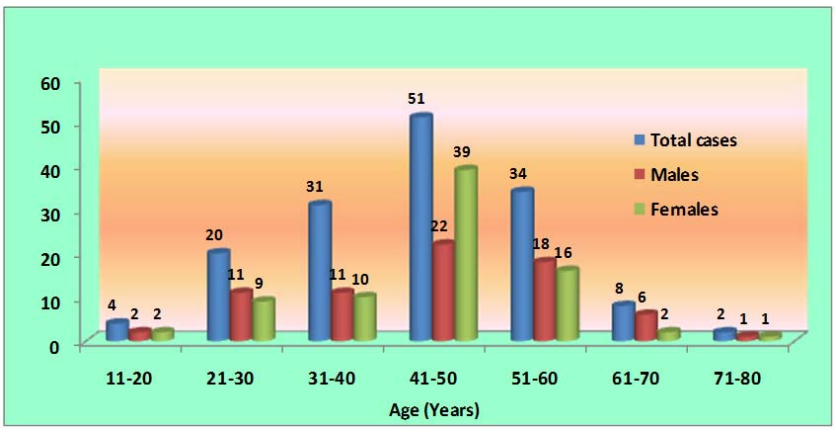

Fig. 1: Age and gender-wise distribution of cases

Table 1: Variations of liver function and lipid profile variables in obese and non-obese hypertensive individuals

Study variables		Mean	Std. deviation	Std. error mean
Bilirubin (Total)	Control (non-obese individuals)	.7547	.92799	.10715
	Test (obese individuals)	.7000	.68042	.07857
Bilirubin (Direct)	Control (non-obese individuals)	.3000	.59729	.06897
	Test (obese individuals)	7.8	12.03	0.97
SGOT	Control (non-obese individuals)	31.2307	38.46285	4.44131
	Test (obese individuals)	75.9733	44.94440	5.18973
SGPT	Control (non-obese individuals)	23.9040	19.55903	2.25848
	Test (obese individuals)	16.4787	32.37280	3.73809
ALP	Control (non-obese individuals)	96.2800	38.74398	4.47377
	Test (obese individuals)	95.7733	51.37319	5.93207
Cholesterol	Control (non-obese individuals)	146.1467	35.43540	4.09173
	Test (obese individuals)	247.5067	68.84616	7.94967
HDL	Control (non-obese individuals)	42.9867	18.92195	2.18492
	Test (obese individuals)	35.4667	13.58291	1.56842
LDL	Control (non-obese individuals)	85.3200	27.41826	3.16599
	Test (obese individuals)	153.9733	64.48821	7.44646
Triglycerides	Control (non-obese individuals)	119.9200	35.71008	4.12344
	Test (obese individuals)	208.1600	84.53370	9.76111
Albumin	Control (non-obese individuals)	4.2400	.53902	.06224
	Test (obese individuals)	4.4693	.77389	.08936
BMI	Control (non-obese individuals)	20.7387	2.23582	.25817
	Test (obese individuals)	24.4333	4.28528	.49482

Table 2: Paired t-test variations of liver function and lipid profile variables in obese and non-obese hypertensive individuals

Variables		Paired differences					t-value	Paired t-test 'p' value
		Mean	Std. deviation	Std. error mean	95% confidence interval of the difference			
					Lower	Upper		
Bilirubin (Total)	Non-obese and obese	0.05	1.100	.127	-.198	.307	.430	.668
Bilirubin (Direct)	Non-obese and obese	7.5	24.6	3.11	1.92	2.54	2.6	.000
SGPT	Non-obese and obese	-14.6	13.37	1.544	-17.76	-11.60	-9.507	.000
SGOT	Non-obese and obese	-44.7	64.09	7.400	-59.48	-29.99	-6.046	.000
ALP	Non-obese and obese	.506	58.9	6.80	-13.05	14.07	.074	.941
Cholesterol	Non-obese and obese	-101.36	82.405	9.51	-120.31	-82.40	-10.652	.000
HDL	Non-obese and obese	7.52	24.42	2.81	1.901	13.13	2.667	.009
LDL	Non-obese and obese	-68.65	66.40	7.66	-83.93	-53.37	-8.953	.000
TGs	Non-obese and obese	-88.24	92.925	10.73	-109.62	-66.85	-8.224	.000
Albumin	Non-obese and obese	-.229	.86	.100	-.429	-.029	-2.284	.025
BMI	Non-obese and obese	-3.69	4.39	.507	-4.70	-2.68	-7.285	.000

Table 3: Correlation analysis of study variables

Study variables		Correlation	Sig. 'p' value
Bilirubin (T)	Non-obese and Obese	.089	.445
Bilirubin (D)	Non-obese and Obese	-.080	.496
SGOT	Non-obese and Obese	-.176	.131
SGPT	Non-obese and Obese	-.075	.525
ALP	Non-obese and Obese	.167	.152
Cholesterol	Non-obese and Obese	-.163	.162
HDL	Non-obese and Obese	-.105	.371
LDL	Non-obese and Obese	.142	.226
TGs	Non-obese and Obese	-.035	.763
Albumin	Non-obese and Obese	.159	.172
BMI	Non-obese and Obese	.212	.067

DISCUSSION

In this study, outpatient hypertension subjects, both obese and non-obese, and their serum lipid parameters (LDL-C, HDL-C, TC, and TG) and liver function parameters (bilirubin, SGOT, SGPT, ALP, and albumin levels) were assessed. In the study population, it was found that significantly more patients with obesity and hypertension had higher levels of TC, TG, LDL-C, bilirubin, SGOT, SGPT, ALP, and albumin. Anthropometric data, such as BMI, were also higher in hypertensive obese patients than the hypertensive non-obese individuals. Compared to non-obese individuals, obese had mean cholesterol, LDL, and triglyceride values that were extremely high, and their HDL levels were lower. In comparison to non-obese persons, all lipid markers were considerably $P<0.0001$ higher in hypertension obese people. This suggests that they are the major contributing factors to the rise in blood pressure. These higher mean levels of TC, TG, and LDL-C in hypertensive patients are in agreement with the results of other related studies which are conducted in different parts of the world [9-14]. In line with the severity of hypertension, an increasing trend was seen for the prevalence of lipid abnormalities, as well as for blood levels of TG, TC, and LDL-C and a declining trend for HDL-C, indicating that these conditions are linked to hypertension in obese people. These findings are consistent with a Nayak *et al.* study [15]. The most

frequent abnormality in the blood lipid profile among hypertension patients was abnormally high LDL-C values, which were followed by abnormally high TC and TG levels. Yet, it was found that the most uncommon lipid anomaly in hypertension patients was low HDL-C. But rather than happening alone, the irregularities frequently happened in groups. The study has showed that the serum level of HDL-C was found to be lower and the results were in agreement with the prior studies when the results for serum TC, TG, and LDL-C were compared with other studies conducted in other regions of the world. Compared to the non-obese control group, hypertension groups had a greater prevalence of dyslipidemia and abnormal liver enzymes. In few earlier research [16–20], the prevalence of dyslipidemia and liver function test indicators was also discovered. In our study, patients who were dyslipidemic had a very high prevalence of increased liver enzymes. This result is partially in line with a study in other populations, where abnormal liver function test indicators were clearly recognised in people who were dyslipidemic, which likely matched the occurrence of dyslipidemia in instances linked with NAFLD.

CONCLUSION

The study found that liver function variables and aberrant lipid profiles were highly prevalent in obese hypertension patients. In participants with dyslipidemia, increased liver enzymes were seen more frequently. This study's findings suggest that people with dyslipidemia may have a higher risk of getting liver disease than people without the condition, and that this risk may be particularly significant in obese people. To comprehend the fundamental processes of lipid-induced hepatic impairment in the target group, a sizable prospective investigation is required.

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Nil

AUTHORS CONTRIBUTIONS

All the authors have contributed equally.

CONFLICT OF INTERESTS

Declared none

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