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To assess the level of lipid parameters and liver enzymes in lipid induced dyslipidaemia

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ABSTRACT

Background: Dyslipidaemia, a major contributor to cardiovascular diseases, is rapidly increasing in Asian countries including India. In addition to the cardiovascular system, abnormal lipid levels are also known to cause complications in renal and hepatic systems. The data regarding dyslipidaemia and its relationship with liver enzymes has threatened. Therefore, this study was conducted to evaluate liver enzyme and dyslipidaemia in lipid induced hepatic diseases.

Methodology: A total of 150 participants (100 males and 50 females) were enrolled in the study. Serum levels of TG, TC, LDL, HDL and liver enzymes including ALT, AST, GGT and ALP were analysed using standard methods. Dyslipidaemia and liver function tests abnormalities were defined according to the international standard guidelines.

Results: Dyslipidaemia and liver enzyme abnormalities were higher in hypertensive participants than in the healthy participants. About 61% of participants with dyslipidaemia had at least one or more elevated liver enzymes. In regression analysis, an independent association was observed between serum GGT and all lipid components.

Conclusion: Subjects with dyslipidaemia often have a higher chance of having liver diseases than subjects with no dyslipidaemia

Keywords: Dyslipidaemia; Liver enzymes; Hypertension; Cardiovascular Diseases.

Introduction:

One of the main causes of cardiovascular diseases (CVDs) and a serious global public health concern is dyslipidemia. The general population now has a higher frequency of dyslipidemia, according to research conducted in the last few decades. Furthermore, compared to industrialized countries, an epidemiological shift in dyslipidemia has been seen in emerging countries. Research from South Asian nations has indicated a growing trend in dyslipidemia among the local populations. (1)

Globally, cardiovascular illnesses caused by dyslipidaemia constitute the primary cause of death. The incidence of cardiovascular diseases is rising quickly among Bangladeshis. Complications in the hepatic, renal, central neurological, and endocrine systems have also been linked to dyslipidaemias. Despite affecting all of the major organ systems, liver damage is thought to be particularly severe because of its strong relationship to lipid metabolism. By producing, storing, and transferring lipid metabolites, the liver contributes significantly to lipid metabolism. Thus, alterations in liver metabolism and hepatic tissue injury might result from abnormalities in lipid levels. Non-alcoholic fatty liver disease (NAFLD) is the most prevalent

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chronic liver disease, characterized by an excessive buildup of lipids in the liver. In industrialized countries, it is currently the most prevalent type of hepatic condition, and in developing countries, it is expected to be the same in the coming decades. Fatty liver disease is also on the rise in India. [2,3,4]

The liver health can be evaluated by measuring the liver enzymes aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and γ-glutamyl transferase (GGT). Serum GGT is present in maximum cell surface concentration and is highly active in the kidney, liver, and pancreas, whereas serum ALT is mostly detected in the liver and is thought to be a particular marker for hepatic injury. Serum GGT is thought to be a biomarker for both liver damage and alcohol use. Moreover, GGT is believed to be associated with inflammation and oxidative stress since it facilitates glutathione uptake. Serum ALT and GGT levels over normal are linked to a number of risk factors, including obesity, dyslipidaemia, hyperglycaemia, and elevated blood pressure, that are linked to metabolic syndrome, diabetes, and cardiovascular illnesses. Elevated levels of ALT and GGT have also been found to be associated with NAFLD.

The liver dysfunction markers ALT and AST have been linked to increased lipid profiles in the general USA population, according to a study. There are no precise statistics on the incidence of dyslipidaemia and its effect on liver function in the Indian population, despite the high prevalence of lipid-induced liver impairment. [7,8] A few epidemiological studies revealed the prevalence of dyslipidaemia in adult Indians, although the majority of them only looked at that specific group and ignored other possible confounders like diabetes and hypertension. Furthermore, only few studies have focused on the connection between liver enzyme levels and adult lipid profile [8]. Consequently, the goal of the current study is to assess the dyslipidaemia and hepatic enzyme status in dyslipidaemia in comparison with hypertensive group.

Materials & Methods

This year-long study, which ran from July 2013 to June 2014, was conducted cross-sectionally and analytically in the Department of Biochemistry at MGM Medical College in Aurangabad, Maharashtra, India, in cooperation with the Department of Medicine at the same institution. Patients from the MGM Medical College Aurangabad, Maharashtra, India, outpatient department served as the subjects. All participants have given their written and verbal consent. The Institutional Ethical Committee gave the study its approval.

For this investigation, a total of 150 individuals, aged 20 to 50, whose clinical histories included demographic data as well as information on the quantity and duration of alcohol usage, were collected. Out of which 75 were hypertensive and 75 were lipid induced dyslipidaemia.

A person with any clotting, bleeding disorders, autoimmune diseases, hepatitis, liver injury, alcohol-related disorders, renal diseases, lipid storage diseases and corticosteroid use were excluded from the study. A blood sample was drawn and proper aseptic procedures were followed in order to evaluate the lipid characteristics. Samples of 3 milliliters of blood were drawn into a red vacutainer. The blood sample was centrifuged for 15 minutes at 3000 RPM in order to extract the serum sample. then kept for further examination of liver enzymes and lipid markers at -20ºC. Serum triglyceride, total cholesterol, and HDL cholesterol were measured using the EM-200 Batch Analyzer to analyze the fasting serum lipid profile based on ERBA

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Enzymatic estimate. Friedewald's equation was used to determine LDL cholesterol: LDL-C= Total cholesterol-(Triglycerides/5)-HDLC. The formula for VLDL cholesterol was serum triglyceride/5.

Diagnostic Criteria:

Dyslipidaemia was defined as the presence of one or more values of TC = 240 mg/dL, TG = 150 mg/dL, LDL = 160 mg/dL, and HDL = 40 mg/dL according to the National Cholesterol Education Program Adult Treatment Panel III. At least one measurement of the ALT level (>45 U/L in men and >34 U/L in women), the AST level (>35 U/L in men and >31 U/L in women), the GGT level (>55 U/L in men and >38 U/L in women, and the ALP level (>129 U/L in men and >104 U/L in women) were indicative of abnormal or elevated liver enzymes. Sustained high blood pressure (SBP ≥ 140 mm Hg and/or DBP ≥ 90 mm Hg) or the use of anti-hypertensive drugs by the subject was considered hypertension. [9,10]

Statistical analysis:

The mean and standard deviation were used to present the quantitative data that had been gathered. Moreover, frequency (%) was used to illustrate the intensity of alcohol drinking. For multiple comparison, used ANOVA (Analysis of Variance). Using SPSS version 20, an analysis was conducted for the significant test. A P value of less than 0.005 is regarded as statistically significant.

Results:

Variables	Male (n = 100)	Female (n = 50)	Total (n = 150)	*P*-value
Age (years)	45.5 ± 7.9	43.6 ± 12.45	43.5 ± 10.9	0.74
Weight (kg)	68.1 ± 8.0	48.3 ± 5.7	64.2 ± 10.2	< 0.001
Height (cm)	168.4 ± 5.9	151.7 ± 7.9	160.7 ± 6.3	< 0.001
BMI (kg/m^2)	24.5 ± 3.4	26.1 ± 4.1	24.6 ± 7.9	0.280
SBP (mmHg)	125.9 ± 5.8	126.2 ± 4.9	126.7 ± 5.7	0.616
DBP (mmHg)	83.6 ± 9.9	83.2 ± 10.1	82.5 ± 2.4	0.539
TG (mg/dL)	199.6 ± 109.54	163.2 ± 87.89	185.7 ± 111.76	0.001
TC (mg/dL)	203.3 ± 98.23	220.3 ± 74.26	210.7 ± 67.34	0.021
LDL (mg/dL)	133.6 ± 62.3	162.7 ± 89.8	140.9 ± 70.0	0.001
HDL (mg/dL)	30.8 ± 9.8	38.9 ± 8.7	36.4 ± 5.9	0.016
ALT (U/L)	36.8 ± 6.9	30.8 ± 15.9	34.9 ± 11.0	0.350
AST (U/L)	28.0 ± 10.0	26.0 ± 11.1	28.4 ± 1.2	0.404
GGT (U/L)	30.8 ± 27.5	28.5 ± 26.8	32.1 ± 29.8	0.380
ALP (U/L)	101.6 ± 38.9	88.9 ± 27.90	97.6 ± 39.5	0.023
Hypertensive (%)	41.6	46.0	42.6	0.470
Physical activity (%)				
Low	22	20	23	0.235
Medium	70	70	70	
Adequate	11.3	3.9	7.7	

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Smoking status (%)				
No	71.9	99	76	< 0.001
Yes	28.9	2	22	

Table-1: Baseline characteristics of the study participants. Data are presented as mean ± SD or %. *P*-values are obtained from independent sample t-test or Chi-square test.

Variables	Total (n = 175)	Dyslipidemia (n = 75)	Hypertensive (n = 75)	*P*-value
TG (mg/dL)	195.7 ± 99.9	210.9± 78.5	203.3 ± 116.3	0.005
TC (mg/dL)	210.7 ± 68.8	230.5 ± 52.0	235.9 ± 91.5	< 0.001
LDL (mg/dL)	138.7± 69.7	148.5 ± 39.4	157.1 ± 80.9	< 0.001
HDL (mg/dL)	34.5 ± 11.3	25.7± 8.0	35.3 ± 14.4	0.002
ALT (U/L)	37.9 ± 23.4	75.4 ± 13.9	36.6 ± 23.0	0.014
AST (U/L)	28.9 ± 11.7	60.9± 11.0	30.5 ± 15.7	0.003
GGT (U/L)	35.6 ± 18.8	40.7 ± 11.9	41.0 ± 37.3	< 0.001
ALP (U/L)	98.9 ± 24.8	94.6 ± 29.8	100.3 ± 41.0	0.505

Table-2: levels of lipid markers and liver enzymes in various groups. Data are presented as mean ± SD. *P* value[a] is the difference between healthy and hypertensive group.

Discussion:

The Vedic period is where the history of alcohol usage begins, particularly in Indian civilization. It is a well-established fact that regular alcohol use might have an impact on lipid components. Drinking alcohol on a daily basis is linked to higher triglyceride and fatty acid synthesis as well as decreased VLDL generation, which causes lipid droplets inside the hepatocyte to aggregate. This intensifies the inflammation, which leads to the development of steatohepatitis and raises the risk of cirrhosis and fibrosis, both of which can advance and cause serious health problems. Particularly in situations of steatohepatitis, a range of clinical diseases and morphological alterations have been noted. (11)

There was a high frequency of dyslipidemia among the study participants in this investigation. Serum GGT was the only liver enzyme to exhibit an independent correlation with every lipid measure. This is the first data that we are aware of about the relationship between liver enzymes and lipid profile markers for the Maharashtrians population. variances in the study population, study areas, age groups, socioeconomic status, genetic predisposition, individual lifestyle, and the use of different cut-off values in identifying dyslipidemia within the study population may all contribute to the variances in the prevalence. A number of research indicate that lipid profile abnormalities are clinically linked to hypertension and diabetes. (12)

The current study's prevalence of low HDL, which was is consistent with earlier research that found HDL cholesterol to be the most often impacted lipid parameter. (13) According to certain research, Asians may be more affected by low HDL cholesterol than by other increased lipid levels. Male and female participants in the sex group had different levels of increased lipids and liver enzymes. Male participants had considerably higher blood TG and low HDL levels

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than female participants, while more female participants had raised TC and LDL levels than male participants.

The hypertension group had a greater prevalence of dyslipidemia and abnormal liver enzyme levels than the healthy control group. The individuals with hypertension experienced an even higher frequency of these anomalies. These results suggest that hypertension and diabetes may be major risk factors for dyslipidemia and liver impairment. This result is partially consistent with another study conducted in other demographics, where dyslipidemic people had clearly identifiable aberrant liver function test indicators, which most likely reflected the presence of dyslipidemia in cases linked to NAFLD. (14)

NAFLD has been linked to high levels of TG, TC, LDL, and low HDL, and people with dyslipidemia are more likely to develop fatty liver disease. Although it is hypothesized that elevated liver function test markers are indicative of excess fat deposition in the liver and ongoing lipid assault on the liver while it performs regular liver functions adds more workload on hepatic cells and potentially changes their physiology, the precise causes of NAFLD pathogenesis are still unknown. Most significantly, the deposited lipid particles may promote hepatic tissue inflammation by causing internal free radical production. (15)

The liver tissue then experiences fibrosis or cell death as a result of these free radicals. Because of the increased stretch caused by the deposited lipid remains, the injured hepatic cell can thin and release more hepatic enzymes into the environment. Once more, it has been proposed that cellular GGT could be linked to the production of reactive oxygen species, a sign of the liver's reduced antioxidant capability as a result of the high lipid levels. Nevertheless, more research is necessary to determine the underlying mechanism of liver enzyme abnormalities in dyslipidaemia. (16,17)

Conclusions:

- Among the Maharashtrians population, dyslipidemia and abnormal liver enzyme levels were noted; they were more prevalent in persons with diabetes and hypertension than in those in good health.
- The blood's raised lipid and GGT levels may serve as a more reliable gauge of the development and severity of lipid-induced hepatic dysfunctions and their related consequences.
- According to this study's findings, those with dyslipidemia may be more likely than people without the condition to develop liver disease.
- To comprehend the fundamental pathways of lipid-induced hepatic impairment in the Maharashtrians population, a large-scale prospective investigation is required.

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