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Original Research Article

ASSOCIATION BETWEEN LIPID PROFILE AND LIVER ENZYMES WITH SERUM ELECTROLYTES AND KIDNEY FUNCTION IN MALE PATIENTS WITH CORONARY ARTERY DISEASE: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: In 2025, cardiovascular disease (CVD) remains a leading global health concern with a significant increase in CVD deaths. Deranged liver and kidney function can significantly impact individuals with coronary artery disease (CAD), increasing the risk of cardiovascular events and mortality, and vice versa. **Aim:** The main aim of the study was i) to explore the associations between liver enzymes and CAD severity and ii) to determine the correlation of liver enzymes with kidney function tests (KFT) and Serum electrolyte in CAD male patients. **Materials and Methods:** A total of 70 male subjects with CAD were enrolled and divided into two sub-groups based on the median Gensini scoring system after observing the angiography findings. The two subgroups were as follows: Group I (Gensini score ≤31, n=38) and Group II (Gensini score >31, n=32). Linear regression analyses were utilised to know the association of liver enzymes with the severity of CAD. p value of <0.05 was considered statistically significant. **Result:** The main findings were as follows: (1) Significantly higher AST, ALP, urea and creatinine were found in patients with higher Gensini score ($p<0.05$); (2) AST ($\beta=0.243$, $p=0.047$) and ALP ($\beta=0.369$, $p=0.001$) were found to be independently associated with the severity of CAD; (3) serum creatinine and sodium were positively correlated with GGT($p<0.05$). **Conclusion**: The present study demonstrated AST and ALP as an independent biomarker for severity of CAD. As liver function test is a readily available assay, validating these data in a larger study and translating the findings into clinical practice would be clinically helpful.

INTRODUCTION

In 2025, cardiovascular disease (CVD) remains a leading global health concern with a significant increase in CVD deaths. With a higher age-standardized CVD death rate (272 per 100,000 population) than the global average (235 per 100,000), India is expected to have a high burden of CVD.[1] The most prevalent major behavioural risk factors for heart disease and stroke among older persons in India include unhealthy eating habits, sedentary lifestyles, tobacco use, and excessive alcohol intake.[2] CVDs are also caused by a number of factors, including stress, poverty, population aging, urbanization, globalization, and genetics.

Lipid dysfunctions including high levels of total cholesterol (TC), low density lipoprotein (LDL), very LDL (VLDL) cholesterol, and triglycerides (TGs), along with low levels of high-density lipoprotein cholesterol (HDL-C), have a major impact on the development of CHD. Specifically, there is a clear correlation between the development and progression of coronary artery disease (CAD) and elevated LDL cholesterol.3In both population-based studies and patients with coronary artery disease, liver function tests (LFTs), such as gamma-glutamyl transferase (GGT), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and aspartate aminotransferase (AST), have become indicators of CVD risk over the past ten years, regardless of their association with metabolic syndrome or non-alcoholic steatohepatitis

(NASH). Numerous mechanisms, such as the direct or indirect contribution of GGT activity within atherosclerotic lesions, vascular calcification, pro-inflammatory activities, endothelial dysfunction, oxidative stress, and impaired haemostasis, have been implicated in the association of LFTs with CVD risk.[4-6] Increased liver enzymes—even those within the normal range—have garnered significant attention as possible new indicators of cardiovascular risk. Atherosclerosis and an elevated risk of cardiovascular disease are closely linked to non-alcoholic fatty liver disease (NAFLD), a prevalent liver disease marked by hepatic fat accumulation.[7] Deranged liver and kidney function can significantly impact individuals with CAD, increasing the risk of cardiovascular events and mortality, and vice versa.[8] Coupled with the incidence of large-vessel coronary disease, arteriosclerosis, microvascular disease, left ventricular hypertrophy and myocardial fibrosis, the prevalence of clinical symptoms of CAD rises as glomerular filtration rate (GFR) falls.[9] The relationship between liver enzymes and severity of CAD has not been thoroughly investigated, despite the fact that abnormal liver enzymes have been suggested as independent risk markers for cardiovascular and all-cause mortality in a number of patient populations. Hence, the main aim of the study was i) to explore the associations between liver enzymes and CAD severity and ii) to determine the correlation of liver enzymes with kidney function tests (KFT) and serum electrolytes in male patients with CAD.

MATERIALS AND METHODS

This was a hospital based cross-sectional study conducted in the Department of Biochemistry in collaboration with the Department of Cardiology, Regional Institute of Medical Sciences, Imphal, from January 2021 to October 2022. Ethical clearance was obtained from the Research Ethics Board, Institutional Ethics Committee (IEC), Regional Institute of Medical Sciences (RIMS), Imphal (Ref no. – A/206/REB-Comm (SP)/RIMS/2015/676/17/2020). A total of 70 male subjects with CAD were enrolled and divided into two sub-groups based on median Gensini scoring system after observing the angiography findings. The two subgroups were as follows: Group I (Gensini score ≤31, n=38) and Group II (Gensini score >31, n=32).

Inclusion Criteria: Only male CAD patients aged 40 years and above were included in present study.

Exclusion Criteria: Patients were excluded from the study if, they had a previous history of revascularisation procedures, malignancy, chronic kidney disease, chronic liver disease, inflammatory diseases, or if, they had used lipid-lowering drugs.

Blood sample collection and laboratory methods

5ml venous blood sample after overnight fasting of at-least 12 hours were collected in plain vial and serum was collected after centrifugation for 10 minutes at 2000-3000 rpm and stored at -20°C till analysis. Serum electrolytes, liver enzymes and KFT parameters were analysed using Beckman DC 700 AU. Serum sodium, potassium and chloride were measured with ion selective electrodes method. Serum creatinine was measured with a kinetic colorimetric assay according to the uncompensated Jaffe method. Serum urea was measured with Kinetic UV test for the quantitative determination of urea. Serum total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL)-cholesterol and high-density lipoprotein (HDL)-cholesterol were measured using homogeneous enzymatic colorimetric assays. Liver enzymes like aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were estimated with kinetic-UV test while Gamma glutamyl transferase (GGT) and alkaline phosphatase (ALP) were measured with kinetic colour test. Following appropriate standardization and quality control of the analyser and using the same lots of reagents, the processed sera were tested in a single batch.

The American Heart Association (AHA)/American College of Dentistry (ACC) 2012 recommendations classified CAD as ≥50% stenosis of at least one epicardial major coronary artery (>2 mm in diameter), as determined by coronary angiography.[10] Diabetes mellitus was defined as a minimum fasting blood glucose (FBG) level of 126 mg/dL or receiving antidiabetic treatments.[11] Hypertension was defined as a blood pressure of at least 140/90 mmHg or taking antihypertensive agents.[12] Hyperlipidaemia was defined as fasting triglyceride (TG) levels >150 mg/dL or total cholesterol (TC) levels >200 mg/dL or receiving lipid-lowering treatments.[13] Normal liver enzyme levels, measured in units per litre (U/L), typically fall within these ranges: ALT (0-45 U/L), AST (0-35 U/L), ALP (30-120 U/L) and GGT (0-30 U/L).[14-16]

Coronary angiography assessment

Coronary angiography was carried out and analysed by an experienced cardiologist. Thereafter, the Gensini score was determined by adding up the severity scores that were given depending on the extent of angiographic luminal stenosis in each coronary artery segment based on visual inspection. The total of each lesion score is the final Gensini score.[17] Taking the median Gensini score, the two subgroups were as follows: Group I (Gensini score <31) and Group II (Gensini score ≥31).

Statistical Analysis

Data analyses were performed using the IBM SPSS Statistics (version 26.0). Continuous (scale) variables with and without normal distributions were expressed as mean ± standard deviations. Linear regression analyses were utilised to know the association of liver enzymes with the severity of CAD. Pearson correlation coefficient was performed to determine the association of liver enzymes and lipid parameters with the kidney function and serum electrolytes. p

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value of <0.05 was considered statistically significant.

RESULTS

Table 1: The baseline characteristics of the study subjects

Variables	The study subgroups		
	CAD groups with Gensini score ≤ 31 (n= 38)	**CAD groups with Gensini score > 31 (n= 32)**	**p-value**
Age (years)	**56.9 ± 9.8**	**71.0 ± 9.2**	**0.001**
BMI (kg/m^2)	28.6± 3.7	29.7± 3.5	0.147
SBP (mmHg)	122 ± 14	121 ± 14	0.678
DBP (mmHg)	90± 8	90 ± 10	0.949
Cholesterol (mg/dl)	**156 ± 32**	**189 ± 32**	**0.001**
Triglycerides (mg/dl)	107 ± 30	119 ± 21	0.73
LDL Cholesterol (mg/dl)	**102± 35**	**123± 35**	**0.016**
HDL cholesterol (mg/dl)	**34 ± 5.3**	**31± 4.1**	**0.006**
ALT (U/L)	33.6 ±12.4	38.1 ± 14.8	0.181
AST (U/L)	**45.4± 18.3**	**75.3± 46.3**	**0.001**
ALP (U/L)	**79.7 ± 42.4**	**112.7± 35.2**	**0.001**
GGT (U/L)	48.0 ± 21.8	57.4 ± 19.8	0.065
Sodium (mEq/L)	132.4± 4.6	132.6± 6.1	0.88
Potassium (mEq/L)	3.9 ± 0.57	3.7 ± 0.71	0.21
Chloride (mEq/L)	97.7±3.56	96.6 ± 5.7	0.32
Urea (mg/dl)	**26.6± 10.1**	**44.8± 20.0**	**0.001**
Creatinine (mg/dl)	**0.69±0.13**	**0.98 ± 0.35**	**0.001**

The baseline and laboratory characteristics of the study subjects were summarised in Table 1. There was a significant difference among the two groups investigated regarding the age distribution (56.9 ± 9.8 vs 71.0 ± 9.2, p=0.001), TC (156.0± 32.0 vs 189.0 ± 32.0, p=0.001), LDL (102.0 ± 35.0 vs 123.0 ± 35.0, p=0.016), HDL (34.0 ± 5.3 vs 31.0 ± 4.1, p=0.006), AST (45.4 ± 18.3 vs 75.3 ± 46.3, p=0.001), ALP (79.7 ± 42.4 vs 112.7 ± 35.2, p<0.001), Urea (26.6 ± 10.1 vs 44.8 ± 20.0, p=0.001), and creatinine (0.69± 0.13 vs 0.98 ± 0.35, p=0.001).

Table 2: Linear regression analysis of liver enzymes

Variables	β-coefficient	95% confidence interval for β		p-value
		Lower limit	Upper limit	
AST	**0.243**	0.001	0.162	**0.047**
ALT	0.092	-0.126	0.293	0.430
GGT	0.183	-0.015	0.228	0.085
ALP	**0.369**	0.046	0.170	**0.001**

Table 2 showed that AST (β=0.243, p=0.047) and ALP (β=0.369, p=0.001) were found to be independently associated with the severity of CAD.

Table 3: Correlation between serum electrolytes and kidney function with lipid profile among study groups

Study groups	Variables	Variables									
		Urea		Creatinine		Na		K		Cl	
		r	p	r	p	r	p	r	p	p	r
CAD groups with Gensini score ≤ 31(n=380 (n= 38)	TC	0.04	0.81	-0.27	0.11	-0.05	0.75	-0.03	0.84	0.12	0.46
	TG	0.11	0.52	0.10	0.49	-0.11	0.49	0.11	0.49	-0.07	0.67
	LDL	0.04	0.79	0.21	0.23	0.14	0.39	0.05	0.78	**0.42**	**0.01**
	HDL	-0.03	0.86	-.05	0.79	-0.06	0.74	0.03	0.88	-0.78	0.08
CAD groups with Gensini score >31 (n= 32)	TC	0.28	0.88	0.26	0.15	0.03	0.88	0.02	0.93	0.26	0.15
	TG	-0.33	0.07	-0.23	0.20	-0.28	0.12	0.15	0.41	-0.25	0.16
	LDL	0.18	0.53	0.33	0.07	0.04	0.81	-0.01	0.97	0.27	0.09
	HDL	-0.03	0.88	-0.09	0.09	0.60	0.26	-0.02	0.90	-0.07	0.71
Combination of the two groups	TC	**0.31**	**0.01**	**0.38**	**0.001**	0.14	0.25	0.06	0.62	0.12	0.34
	TG	0.17	0.16	0.21	0.08	-0.12	0.32	-0.17	0.15	-0.21	0.09
	LDL	**0.29**	**0.02**	**0.16**	**0.002**	0.18	0.13	0.12	0.32	0.17	0.14
	HDL	-0.17	0.16	**-0.19**	**-0.04**	-0.08	0.48	-0.12	0.31	-0.05	0.71

Table 3 demonstrated that serum urea and creatinine were positively correlated with TC (r=0.31, p=0.01 and r=0.38, p=0.001 respectively) and LDL (r=0.29, p=0.02 and r=0.16, p=0.002 respectively). Also, serum creatinine is negatively correlated with HDL (r=-0.19, p=-0.04).

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Table 4: Correlation between serum electrolytes and kidney function with liver enzymes among study groups

Study groups	Variables	Variables									
		Urea		Creatinine		Na		K		Cl	
		r	p	r	p	r	p	r	p	p	r
CAD groups with Gensini score ≤ 31 (n= 38)	AST	0.04	0.80	0.01	0.94	-0.03	0.84	0.08	0.62	0.04	0.82
	ALT	-0.22	0.18	**0.43**	**0.01**	-0.22	0.18	0.04	0.82	-0.04	0.82
	ALP	-0.21	0.20	-0.28	0.10	-0.12	0.47	0.13	0.46	0.42	0.15
	GGT	-0.14	0.38	0.26	0.12	0.16	0.35	0.05	0.78	0.24	0.14
CAD groups with Gensini score >31 (n= 32)	AST	-0.25	0.17	-0.30	0.09	0.06	0.73	-0.13	0.48	-0.30	0.66
	ALT	-0.38	0.03	-0.33	0.06	0.08	0.66	-0.14	0.45	0.23	0.09
	ALP	0.16	0.39	-0.21	0.25	-0.04	0.83	0.18	0.32	0.24	0.21
	GGT	0.24	0.42	**-0.35**	**0.02**	**0.37**	**0.04**	0.12	0.51	-0.06	0.19
Combination of the two groups	AST	0.06	0.58	-0.01	0.98	0.09	0.46	-0.13	0.28	-0.11	0.38
	ALT	-0.19	0.11	-0.19	0.11	-0.03	0.82	-0.09	0.46	-0.17	0.15
	ALP	0.08	0.51	0.03	0.81	-0.05	0.67	-0.08	0.48	-0.03	0.84
	GGT	0.17	0.15	**0.36**	**0.002**	**0.19**	**0.01**	0.93	0.44	0.21	0.09

Table 4 showed that there is significant positive correlation of serum creatinine (r=0.36, p=0.002) and sodium (r=0.19, p=0.04) with serum GGT.

DISCUSSION

The present study demonstrated the associations between liver enzymes and CAD severity and the correlation of liver enzymes with kidney function tests (KFT) and Serum electrolyte in male patients with CAD. The main findings were as follows: (1) Significantly higher age distribution, TC, LDL-C, AST, ALP, urea and creatinine and lower HDL-C were found in patients with higher Gensini score; (2) among the liver enzymes, AST and ALP were found to be independently associated with the severity of CAD; (3) serum creatinine and urea were positively correlated with TC and TG and serum creatinine was also negatively correlated with HDL-C; (4) serum creatinine and sodium were positively correlated with GGT.

The present study showed higher age distribution in more severe CAD group when compared with less severe CAD patients. Forman D E et al stated that that by the age of 75 and 85 years, respectively, most adults have developed significant aging vascular alterations to cause changes in physiology, organ function, and reserve.[18] Mitochondrial dysfunction is another consequence of oxidative stress, which includes the generation of excess ROS that develops with cardiac aging.[19] Loss of mitochondrial function is a critical factor in the development of cardiac dysfunction in older population because cardiac aerobic metabolism is heavily reliant on mitochondrial ATP generation.[20]

The present study demonstrated higher TC and LDL-C and lower HDL-C in patients with more severe CAD. These findings were consistent with the study conducted by Li Y et al, which included 452 patients and showed that TC and LDL-C were notably higher in patients with higher Gensini score while HDL-C were significantly lowered.[21] Vascular stenosis and atherosclerosis are known to be patho-physiologically linked to dyslipidemia.[22] Our findings showed that AST and ALP were significantly higher in severe CAD groups and independently associated with the severity of CAD. Yu T et al demonstrated ALP as an independent predictor of in-hospital mortality in patients with acute coronary syndrome.[23] Liver transaminases are frequently utilized and evaluated in a clinical environment. Various studies had shown that elevated AST is independently associated with cardiovascular events and atherosclerosis.[24-25] Oh PC et al demonstrated that increased serum ALP were independently associated with a higher risk of adverse cardiac events or cerebrovascular events after acute myocardial infarction.[26]

Bagheri B et al,[27] revealed significant negative correlation of serum creatinine with TC, TG and HDL-cholesterol while our study demonstrated significant positive correlation of serum creatinine and urea with TC and LDL-cholesterol and negative correlation of creatinine with HDL-cholesterol. Reduced creatinine clearance or an elevated serum creatinine level are reliable markers of increased death rates due to CVD.[28] However, Rasouli M et al stated creatinine as a marker for kidney function and an index for body water, but not an indicator for inflammation.[29] The present study showed that serum creatinine and sodium were positively correlated with GGT. In a prospective study by Singh KK et al, which included 200 patients with CAD, serum GGT levels were significantly higher in patients with double/triple vessel CAD as compared to those with single-vessel disease, highlighting the association of GGT with increasing severity of obstructive CAD.[30] Till date we could not find any studies which showed any direct correlation of serum sodium and creatinine with GGT in CAD patients. However, on the other hand, GGT is more directly linked to the presence and severity of CAD, particularly in relation to vascular obstruction and oxidative stress.[30]

Increased liver function test is suggesting a sign of excessive hepatic fat accumulation and ongoing lipid attacks on the liver, which increases the strain on hepatic cells and may alter the physiology of the organ. Most significantly, by producing free radicals within the liver, the accumulated lipid particles may promote inflammation of the liver tissue.[31] The liver tissue subsequently experiences fibrosis or cell death as a result of these free radicals. The additional

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stretch from the deposited lipid remnants can lead the injured hepatic cell to shrink and become more permeable, allowing it to release more hepatic enzymes outside. Yet again, it has been proposed that cellular GGT might be linked to the production of reactive oxygen species, which might be a sign of the liver's diminished antioxidant ability as a result of the high lipid levels.[32]

The study has some limitations. First, confounding factors were not considered. Second, the cross-sectional nature of the data may affect the relationship between lipid profile markers, KFT and electrolytes. Moreover, the sample size was relatively small and it was a single centred study, hence the findings should be generalised with caution. Further larger studies are required to confirm our findings.

CONCLUSION

Our study reinforced the evidence that AST and ALP, as an inexpensive and routinely marker, can be used as an independent indicator for the severity of CAD. The interaction of lipid profiles, liver enzymes, kidney function, and electrolyte levels are essential to comprehending and treating CAD as proactive management of these variables can help minimize cardiovascular risk. Also, liver function test is a readily available assay, validating these data in a larger study and translating the findings into clinical practice would be clinically helpful.

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