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Study of Lipid Profile of Patients with Chronic Coronary Syndromes at Sohag University Hospital

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ABSTRACT

Background: Coronary artery disease (CAD) has been proven to be causally associated to genetically defined and metabolically produced changes in lipid metabolism, as seen in numerous kinds of dyslipidemia.

Objective: The aim of the present study was to assess the lipid profile in patients with chronic coronary syndrome.

Patients and Methods: A hospital based cross-sectional study was conducted at Sohag University Hospital and included 100 patients with chronic coronary syndrome. The examined if our patients could achieve the target lipid profile level of their group according to 2019 ESC/EAS Guidelines for the management of dyslipidaemia. History of CAD and family history were taken. Clinical presentation and body mass index (BMI) were assessed. Finally, lifestyle, drug history (lipid lowering drug, anti HTN drug and anti-platelets drugs), dietary intake assessment and physical exercise were evaluated.

Results: Patients had a mean HDL-C level of 39.4 91mg/dL, a mean total cholesterol level of 188.91mg/dL, a mean LDL-C level of 119.67mg/dL and a mean triglyceride level of 149.15mg/dL. HDL-C was significantly lower for non-statin therapy group compared to statin therapy group (P<0.001), while triglycerides and vLDL were significantly higher non-statin therapy group compared to statin therapy group (P<0.001). Only 8% of patients achieved target LDL-C according to 2019 ESC/EAS guidelines.

Conclusion: Most of patients at Sohag University Hospital are not reaching the target LDL-C. Thus, more strict application of guidelines and investigating the predisposing factors for uncontrolled lipid profile, especially in patients with CAD, are urgently needed.

Keywords: Lipid Profile, Chronic Coronary Syndromes, LDL-C.

INTRODUCTION

From 1970 to 2015, it was predicted that cardiovascular disease deaths will decrease in wealthy nations while nearly doubling in underdeveloped ones. According to the Global Burden of Disease research, cardiovascular illnesses were responsible for 2.3 million fatalities, or 25%, of the 9.4 million total deaths recorded worldwide in 1990. There will likely be a 111% increase in cardiovascular fatalities in India by 2020, according to predictions. This surge exceeds China's 77%, other Asian nations' 106%, and economically developed nations' 15% increases [(1)].

Any community's relative changes in biological features such serum lipids, blood pressure, blood glucose, insulin, and thrombogenic variables are related to the prevalence of coronary heart disease in that group. This theory is based on Key's postulation of ill individuals and sick groups as well as Pickering's observation that sick people are just the extreme of a continuous distribution. Changes in lifestyle, such as quitting smoking, getting more exercise, drinking alcohol, and eating a rich diet, as well as psychological factors that accompany economic change, are the cause of these adjustments [(2)].

A significant biochemical change in the arteries occurs during dyslipidemia as a result of an accumulation of lipids, either in the form of free cholesterol or its ester, which results in the development of plaques in the inner wall of the artery. No new plaques will develop if the total cholesterol level is less than 150 mg/dl. When the plaques with thin fibrous caps break, an acute coronary event is anticipated. The commencement of an acute coronary event is determined by the kind of plaque, not the degree of coronary artery constriction. Dyslipidemia is reported to raise fibrinogen levels, platelet activation inhibitor, and platelet aggregation. Increased LDL oxidation (free radical damage), increased platelet aggregation (clumping), increased plasma fibrinogen, coagulation factors, hypertension, changes in glucose metabolism, smoking, hereditary and environmental variables are only a few of the causes linked to CAD [(3)].

It is generally established that statins help people with coronary artery disease or who are at high risk of developing cardiovascular disease by lowering cardiovascular events and death. Statins have pleiotropic benefits that include better endothelial function, decreased inflammation, and decreased thrombus formation in addition to decreasing low-density lipoprotein (LDL) cholesterol. Numerous recent studies have shown that statins are effective for treating coronary artery disease in people [(4)].

Current recommendations emphasize using statins to reduce low-density lipoprotein cholesterol (LDL-C) for both primary and secondary prevention. Low levels of high-density lipoprotein cholesterol (HDL-C) and increased triglycerides are two additional significant lipid abnormalities that both independently indicate a high residual risk for future cardiovascular events [(5)].

There is no discernible link between serum HDL-C and the prevalence of CVD. According to a systematic review and meta-analysis, higher HDL-C levels were not linked to a lower risk of CVD or CVD death [(6)].

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The aim of the present study was to assess the lipid profile in patients with chronic coronary syndrome.

In this study, we aimed to Study lipid profile in patient with chronic coronary syndrome and determine the lipid level of patients on anti-hyperlipidemia therapy to show whom of these patients are reaching he goal of therapy which is an LDL-C reduction of >_50% from baseline and an LDL-C goal of <1.4 mmol/L (<55 mg/dL) are recommended.

PATIENTS AND METHODS

A hospital based cross-sectional study was conducted at Sohag University Hospital and included 100 patients with chronic coronary syndrome.Patients were classified into 2 main groups: A) Patient on statin therapy; B) Patient not on statin therapy.

To determine if patients in Sohag University Hospital could achieve the target lipid profile level of their group 2019 ESC/EAS Guidelines for the management of dyslipidaemia were used [(7)].

The lipid level of patients on anti-hyperlipidemia therapy was accepted as reaching the goal of therapy when there was LDL-C reduction of >50% from baseline and LDL-C <1.4 mmol/L (<55 mg/dL).

Inclusion criteria: All patients have chronic coronary disease at Sohag University Hospital.

Exclusion Criteria: Patients with first attack of acute coronary syndrome, young patients <18 years, and old patients >75 years.

Method:

Medical records of the included patients were reviewed for:

a) History of coronary artery disease and family history.
b) Clinical presentation, body mass index (BMI), history of HTN, history of DM).
c) Socioeconomic characteristics (Age, sex, marriage state, education).
d) Life style (smoking: current smoker, ex- smoker, and non-smoker).
e) Drug history: (lipid lowering drug, anti HTN drug and anti-platelets drugs).
f) Dietary intake assessment, physical exercise.

Ethical consideration:

This study was ethically approved by the Institutional Review Board of the Faculty of Medicine, Sohag University. Written informed consent was obtained from all participants. This study was executed according to the code of ethics of the World Medical Association (Declaration of Helsinki) for studies on humans.

Statistical Analysis

SPSS Programme (Statistical Package for Social Sciences) software version 26.0, Microsoft Excel 2016, and the MedCalc Programme software were used to tabulate and statistically analyze the collected data. For numerical parametric data, descriptive statistics were performed using the mean, standard deviation (SD), minimum and maximum of the range; for numerical non-parametric data, the median and first and third interquartile ranges were used. For categorical data, the number and percentage were performed using.

For quantitative variables, inferential analyses were performed using the independent t-test when there were two independent groups and parametric data, and the Mann Whitney U when there were two independent groups and non-parametric data.

Chi-square test for independent groups was used for inferential analysis of qualitative data. P values equals or less than 0.05 were used to determine significance.

RESULTS

Table 1 illustrates distribution of studied cases as per demographic characteristics and clinical history.

Table (1): Demographic characteristics and clinical history of the studied cases.

Parameters		Studied cases (n= 100)	
		N	%
Age (years)	Mean ± SD	52.57 ± 8.54	
	Median	51.5	
	Range	35 – 71	
Sex	Male	71	71%
	Female	29	29%
Dietary habits	Negative	54	54%
	Positive	46	46%
Smoking	Not smoker	41	41%
	Smoker	59	59%
BMI	Normal	46	46%
	Obese	43	43%
	Morbid obesity	11	11%
Hypertension	Not hypertensive	46	46%
	Controlled hypertensive	41	41%
	Not controlled hypertensive	13	13%
DM	Not diabetic	42	42%
	Controlled diabetic	43	43%
	Not controlled diabetic	15	15%

SD: standard deviation, n: number, %: percentage.

Table 2 summarizes lipid profile in the studied cases.

Table (2): Descriptive analysis of studied cases as per lipid profile.

Parameters	Studied cases (n= 100)	
	Mean	SD
HDL-C (mg/dl)	39.40	8.10
Triglyceride (mg/dl)	149.15	36.31
Total cholesterol (mg/dl)	188.91	46.32
LDL-C (mg/dl)	119.67	28.45
vLDL-C (mg/dl)	30.04	7.31

SD: standard deviation, n: number, %: percentage.

Table 3 shows distribution of statin therapy treated ACS patients as per Target LDL-C attainment (% at goal).

Table (3): Distribution of studied cases as per Target LDL-C, according to ESC 2019.

Target	Studied cases (n= 100)	
	Mean	SD
<55 (mg/dl)	119.67	28.45

SD: standard deviation, n: number, %: percentage.

Table 4 shows distribution of lipid profile controlled cases among ACS patients. It was noticed that there were 8% controlled cases while there were 92% uncontrolled cases.

Table (4): Distribution of studied cases as per control.

Parameters		Studied cases (n= 100)	
		N	%
Controlled cases	No	92	92%
	Yes	8	8%

N: number, %: percentage.

The studied cases were divided into two groups according to anti hyperlipedimic therapy (statin therapy) intake: Group A: 63 patients on statin therapy, and Group B: 37 patients not on statin therapy. Table 5 shows comparison between the 2 studied groups regarding demographic and clinical data.

The comparison between mean age in statin therapy group and non-statin therapy group was not statistically significant (P=0.361). There were no statistically significant differences between the two groups regarding sex, dietary habits, smoking, BMI, hypertension (P>0.05). Only, there was a statistically significant difference between the two groups regarding DM (P=0.027).

Table (5): Comparison between the studied groups regarding demographic and clinical data.

Variable		Statin therapy group (No. = 63)		No statin therapy group (No. = 37)		Test value	P-value
		No.	%	No.	%		
Age (years)	Mean ± SD	51.97 ± 8.09		53.59 ± 9.29		T= 0.918	0.361
	Median	51.0		54.0			
	Range	35.0 - 71.0		37.0 - 70.0			
Sex	Male	43	68.3%	28	75.7%	X^2= 0.624	0.430
	Female	20	31.7%	9	24.3%		
Dietary habits	Negative	36	57.1%	18	48.6%	X^2= 0.677	0.411
	Positive	27	42.9%	19	51.4%		
Smoking	Not smoker	28	44.4%	13	35.1%	X^2= 0.835	0.361
	Smoker	35	55.6%	24	64.9%		
BMI	Normal	31	49.2%	15	40.5%	X^2= 0.836	0.658
	Obese	26	41.3%	17	45.9%		
	Morbid obesity	6	9.5%	5	13.5%		
Hypertension	Not hypertensive	28	44.4%	18	48.6%	X^2= 1.05	0.592
	Controlled hypertensive	28	44.4%	13	35.1%		
	Not controlled hypertensive	7	11.1%	6	16.2%		
DM	Not diabetic	27	42.9%	15	40.5%	X^2= 7.22	**0.027**
	Controlled diabetic	31	49.2%	12	32.4%		
	Not controlled diabetic	5	7.9%	10	27.0%		

P≤0.05 is considered statistically significant, P≤0.01 is considered high statistically significant, SD: standard deviation,
* Chi-square test and Student's t test.

Table 6 shows comparison between the two studied groups regarding lipid profile. HDL-C was significantly lower for non-statin therapy group compared to statin therapy group (P<0.001), while triglycerides and vLDL were significantly higher in non-statin therapy group compared to statin therapy group (P<0.001). Differences in mean LDL-C and TC levels, with higher values seen for non-statin therapy group, but did not reach/ statistical significance.

Table (6): Comparison between the two studied groups regarding lipid profile.

Variable		Statin therapy group (No. = 63)	No statin therapy group (No. = 37)	Test value*	P-value
HDL-C (mg/dl)	**Mean ± SD**	41.57 ± 8.29	35.70 ± 6.32	Z_{MWU}= 3.625	**<0.001**
Triglyceride (mg/dl)	**Mean ± SD**	137.97 ± 32.52	168.19 ± 41.28	Z_{MWU}= 2.868	**0.004**
Total cholesterol (mg/dl)	**Mean ± SD**	183.4 ± 44.3	205.27 ± 14.31	Z_{MWU}= 1.917	0.055
LDL-C (mg/dl)	**Mean ± SD**	116.57 ± 27.34	122.49 ± 28.23	Z_{MWU}= 0.757	0.449
vLDL-C (mg/dl)	**Mean ± SD**	27.6 ± 6.44	34.21 ± 7.62	Z_{MWU}= 3.186	**0.001**

P≤0.05 is considered statistically significant, P≤0.01 is considered high statistically significant, SD: standard deviation, * Mann-Whitney U test.

Table 7 shows that there was no relation between receiving statin therapy and lipid profile control (P>0.05).

Table (7): Association between receiving statin therapy and lipid profile control.

Variable		Receiving statin therapy				Test value	P-value
		No		Yes			
		No.	%	`No.	%		
Control	**No**	36	97.3%	56	88.9%	X^2= 2.24	0.135
	Yes	1	2.7%	7	11.1%		

P≤0.05 is considered statistically significant, P≤0.01 is considered high statistically significant, SD: standard deviation, * Chi-Square test.

DISCUSSION

This cross-sectional study was conducted on 100 patients with chronic coronary syndrome. Participants were divided into two main groups: Group A: 63 patients on statin therapy, and Group B: 37 patients not receiving statin therapy. The age of cases ranged from 35 to 71 years with mean age of 52.57 years. There were 71 males and 29 females.

Mohsen Ibrahim *et al.* [8] in their study with similar objective to the current study included 1000 consecutive CAD patients fulfilling the inclusion criteria. The outcomes were contrasted with those of a control group consisting of 716 normotensive and 1404 hypertensive participants without coronary disease. The patients' ages varied from 19 to 90, with a mean of 54 years old. They were mostly male (77.6%), had stable CAD (angina-AP), and 457 had a history of myocardial infarction (MI). A total of 56.7% of patients had hypertension, 25.8% had obesity (BMI 30 kg/m2), and 34.4% had diabetes mellitus.

A total of 717 patients in were investigated by **Huang *et al.*** [9]. For this study, 328 individuals made up the group with CAD and chronic kidney disease (CKD). There were 242 and 147 patients in the CAD-only and CKD-only groups, respectively. About 57.5% of the individuals with CAD had CKD. The oldest patients belonged to the CAD and CKD group. In the CKD group, female gender predominated more frequently. Notably, all patients in the CKD-only group had renal illness that was stage 5. The CAD and CKD group had higher rates of hypertension and myocardial infarction, whereas the CAD group had higher rates of smoking and angina pectoris. The group with CAD and CKD had the lowest ejection fraction. Concomitant drugs including calcium-channel blockers, angiotensin-converting enzyme inhibitors (ACEI), angiotensin-receptor blockers (ARB), beta-blockers, and aspirin were significantly different amongst the three groups. More than 90% of CAD patients with and without CKD received statin medication, compared to just 46.3% of CKD-only patients, which was another notable difference. The outcomes of the baseline and follow-up laboratory tests varied amongst the three groups as well.

Regarding lipid profile before admission in the studied cases, the mean total cholesterol and HDL-c level were 221.64 (SD 52.30) mg/dl and 27.47 (SD 6.72) mg/dl, respectively. The mean LDL-c and vLDL-c level were 138.70 (SD 33.34) mg/dl and 42.31 (SD 10.23) mg/dl, respectively. The mean triglycerides level was 183.93 (SD 43.32) mg/dl.

Gibson *et al.* [10] reported that at the time of randomization, the median serum LDL-C concentration was 106 mg/dl in both groups, which is consistent with our findings. In the 40 mg pravastatin/PCI group (n=1,425) and the 80 mg atorvastatin/PCI group (n=1,442), the median LDL-C concentration at 30 days was 89 mg/dl and 56.5 mg/dl, respectively (P<0.001). Similar to this, the attained median LDL-C values at 30 days were 87 mg/dl in the group receiving 40 mg of pravastatin and no PCI (n=637) and 58 mg/dl in the group receiving 80 mg of atorvastatin and no PCI (n=657) (P<0.001). In the PCI group, the median serum CRP level was 13.2 mg/l, whereas in the no PCI group, it was 9.4 mg/l. In the 40 mg pravastatin/PCI group, the CRP concentration was 2.14 mg/l at 30 days, 1.55 mg/l at 80 mg atorvastatin/PCI (P<0.001), 2.63 mg/l at 40 mg pravastatin/no PCI, and 1.88 mg/l at 80 mg atorvastatin/no PCI (P<0.001).

The mean values (mg/dl) for LDL-C, HDL-C, TC, and triglycerides (TG) in **Mohsen Ibrahim *et al.*** [8]

were 140.6, 41.5, 217, and 160.9, respectively. About 49.2% of CAD patients had low HDL-C (40 mg/dl), 30.2% had elevated LDL-C (>160 mg/dl), and 45% had elevated triglycerides (>150 mg/dl). According to gender and CAD type, different lipid abnormality patterns were observed. Low HDL-C was the most common anomaly in men (55.4%). The most prevalent anomaly in females was elevated LDL-C (41.1%).

Regarding lipid profile before and after admission in the studied cases, there was significant decrease in levels of total cholesterol, triglycerides, LDL-c and vLDL (P<0.001) while there was significant increase in HDL-c level post admission when compared to levels before admission.

In **Huang *et al.*** [9] study, they compared how statins affected the clinical outcomes of the three patient groups. According to the statistics, CAD and CKD patients who received long-term statin medication fared better than CAD-only and CKD-only patients. Long-term statin use had no appreciable impact on the MACE rate in CAD-only patients, but it did reduce the MACE rate by 20.5% in CAD and CKD patients using long-term statins. Long-term statin therapy showed substantial impacts on mortality rates for both cardiac and all-cause deaths in patients with CAD alone (approximately 11% decrease), although this difference was less pronounced in patients with CAD plus chronic kidney disease (around 28% reduction). On the other hand, long-term statin medication had no appreciable impact on the clinical outcomes of individuals with CKD alone.

Regarding distribution of statin therapy treated ACS patients as per Target LDL-C attainment (% at goal) by risk level. The statin-treated patients were divided according to pre-admission risk level, 34.9% of those at high risk, 20.6% of those at moderate risk and 44.4% of those at low risk were at their respective LDL-C target.

Of the 38,775 outpatients with obstructive CAD in **Arnold *et al.*** [11] study, 30,160 (77.8%) received statin treatment, 2042 (5.3%) received just non-statin lipid-lowering medicine, 6441 (16.6%) received both statin and non-statin lipid-lowering treatment, and 6573 (17.0%) received no treatment. Patients not receiving statin therapy (n=8615; 22.2%) had a higher likelihood of being younger, female, and history-free of previous myocardial infarction, coronary artery bypass grafting, or atrial fibrillation. Statin-untreated patients also had lower odds of having undergone past PCIs and having peripheral artery disease, heart failure, diabetes mellitus, hypertension, and concomitant hypertension (P<0.001 for all).

Regarding distribution of controlled cases among ACS patients, it was noticed that there were 8% controlled cases while there were 92% uncontrolled cases.

In comparison between the two studied groups regarding demographic and clinical data, the mean age in statin therapy group and non-statin therapy group was respectively with no statistically significant difference between the two groups (P=0.361). There were no statistically significant differences between the two groups regarding sex, dietary habits, smoking, BMI, hypertension (P>0.05). There were statistically significant differences between the two groups regarding DM (P=0.027).

In comparison between the two studied groups regarding lipid profile, patients in statin and non-statin therapy groups had a mean HDL-C level of 41.57 (SD 8.29) and 35.70 (SD 6.32) mg/dl, a mean TC level of 183.40 (SD 47.3) and 205.27 (SD 62.02) mg/dL, a mean LDL-C level of 116.57 (SD 50.05) and 122.49 (SD 48.04) mg/dL and a mean triglyceride level of 137.97 (SD 57.31) and 168.19 (SD 60.28) mg/dL.

HDL-C was significantly lower for non-statin therapy group compared to statin therapy group (P<0.001), while triglycerides and vLDL were significantly higher non-statin therapy group compared to statin therapy group (P<0.001). Differences in mean LDL-C and TC levels, with higher values seen for non-statin therapy group, did not reach statistical significance. There was no relation between receiving statin therapy and control (P>0.05).

Ray and Cannon [12] demonstrated, in contrast to our findings, that intensive statin therapy with 80 mg of atorvastatin to achieve a low-density lipoprotein cholesterol of 62 mg/dl resulted in a 3.9% absolute and a 16% relative risk reduction in death or major cardiovascular events up to 2 years, compared to 40 mg of pravastatin, in patients with ACS. The benefits of intensive statin treatment were shown very quickly (within 30 days), and they occurred against a backdrop of high secondary prevention drug use and percutaneous coronary intervention (PCI) (69%) for the index hospitalization. The C-reactive protein sub-study of the Myocardial Ischemia Reduction with Aggressive Cholesterol Lowering (MIRACL) study and the Pravastatin or Atorvastatin Evaluation and Infection Therapy study both found that atorvastatin (80 mg) resulted in a significant reduction in inflammation markers, while the Reversal of Atherosclerosis with Aggressive Lipid Lowering (REVERSAL) study found that intensive statin therapy was associated with

According to **Gibson *et al.*** [10] the clinical advantage of statins in secondary prevention is at least partially attributed to their ability to reduce LDL-C. The advantages of statin treatment in ACS patients, however, may also be attributable to pleiotropic processes, which are effects that don't reduce LDL-C. The amount and timing of reductions in cardiovascular events observed in certain clinical studies may not be fully explained by LDL-C lowering alone. Pleiotropic effects have been hypothesized to include advantages in

the reduction of inflammation, plaque stability, and endothelial function.

LDL-C readings were available for 3365 individuals (51.2%) in **Arnold *et al.* [11]** among patients who were not receiving any lipid-lowering treatment (n=6573; 17%). 1794 (53.3%) of individuals having LDL-C findings had values under 100 mg/dL, and 644 (19.1%) had levels under 70 mg/dL. Additional 1571 patients (46.7%) with LDL-C values under 100 mg/dL were not receiving any lipid-lowering therapy.

CONCLUSION

Most of patients at Sohag University Hospital are not reaching the target LDL-C. Thus, more strict application of guidelines and investigating the predisposing factors for uncontrolled lipid profile, especially in patients with CAD, are urgently needed.

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