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Original Research Article

Prognostic significance of high levels of lipoprotein (a) and C-reactive protein in coronary artery disease

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ABSTRACT

Background: Inflammatory markers have been associated with clinical outcome in patients with coronary artery disease (CAD). Atherosclerotic cardiovascular disease is the leading cause of mortality, accounting for more than 15 million deaths worldwide every year. The present study was done to evaluate the role of serum C-reactive protein (CRP) and serum lipoprotein (S. Lp) (a) in CAD.
Methods: Sixty patients admitted in Guru Nanak Dev Hospital, Amritsar and sixty healthy subjects were included, and they were followed up for 180 days for any major cardiovascular events after the treatment. Clinical parameters like S. Lp (a) and S. CRP levels were evaluated as risk factors for coronary artery disease and as predictors of major cardiovascular events (such as the occurrence of cardiac death, ischemic stroke or myocardial infarction and mortality.
Results: Patients were further categorized into three groups based on serum Lp (a) and serum CRP levels: group A: S. Lp (a) levels >30 mg/dl and S. CRP >5 mg/l, group B: S. Lp (a) levels >30 mg/dl and S. CRP levels were normal, group C: S. Lp (a) levels <30 mg/dl and S. CRP >3 mg/l. Patients with S. Lp (a) >30 mg/dl and S. CRP levels >5 mg/l had a significantly lower survival than the other two groups. When compared with normal healthy subjects S. Lp (a) and S. CRP levels were statistically higher in patients of ACS.
Conclusion: The adverse effects of elevated S. Lp (a) levels and S. CRP levels on cardiovascular system have been associated with endothelial dysfunction, inflammatory cells migration and infiltration, oxidative stress, and fibrinolysis inhibition. These inflammatory processes together lead to cardiovascular events and high levels of these inflammatory markers leads to high mortality rates.

Keywords: Coronary artery disease, Lipoprotein (a), CRP, Inflammatory process, Mortality rate

INTRODUCTION

Cardiovascular disease is the most common cause of death worldwide accounting for 17.9 million deaths annually.[1] Indians are suffering from more severe and diffuse acute coronary artery disease with a predisposition in younger age, having serious complications. Coronary artery disease (CAD) represents a common clinical manifestation of cardiovascular disease (CVD), which results in a large number of hospital admissions and emergency department consultations around the world.[2] Despite of advances in treatment options like reperfusion strategies and pharmacological treatments, people with acute coronary syndrome still experience subsequent cardiovascular events and mortality rates.[2] The most common risk factors are hyperlipidaemia, hypertension, diabetes mellitus, and cigarette smoking. Other relevant risk factors include insulin resistance syndrome, metabolic syndrome X, lipoprotein (Lp) (a) and some emerging risk factors include homocysteine, tissue plasminogen activator (tPA), plasminogen activator inhibitor 1 (PAI-1), fibrinogen, and factor VII. These all risk factors guide the physician about the patient's prognosis, risk assessment, and treatment

results.[3] For clinicians, risk evaluation plays an important role by, improving their clinical judgment processes, informing them about appropriate pharmacological treatments, helping them to determine when to discharge the patient, and helping them to schedule follow-up care after discharge.[4]

Coronary artery disease is divided two broad categories that is patients with chronic coronary artery disease who most commonly present with stable angina and patients with acute coronary syndromes (ACS). ACS is divided into ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA).[5] They are common in older adults, but now-a-days the incidence has increased in younger people.[6]

Atherosclerosis is the most common pathological multifactorial inflammatory process that leads to CAD. Inflammation in the vessel wall causes initiation, progression, plaque destabilization and eventually plaque rupture.[7] Inflammatory mediators, like activated T cells and mast cells attach themselves to the endothelium. Activation of macrophages, T lymphocytes, and SMCs leads to the release of additional mediators, like adhesion molecules, cytokines, chemokines, and growth factors. All of these inflammatory cells eventually lead to the formation of the atheromatous lesion, which consists of a lipid pool protected by a fibrous cap.[8] With time this plaque attracts deposits of calcium which leads to calcification of the arteries. Finally, endothelium-derived NO, is reduced at the site of vascular injury.[9,10] Decreased NO production is responsible for the clinical course.

The inflammatory aetiology of atherosclerosis has resulted in the search of biomarkers of inflammation that predict risk for coronary artery disease and its sequelae. In ACS, inflammatory biomarkers may provide independent information regarding pathophysiology, prognosis, and optimal therapeutic strategies. The goal of the present study was to evaluate levels of serum CRP and serum Lp (a) and risk of CAD and to find out the association between the inflammatory marker CRP and Lp (a) at the time of hospitalization for coronary artery disease and late cardiovascular outcomes (patients were followed up till 180 days).

CRP is an ‘‘acute-phase reactant’’ that increased in serum of patients suffering from inflammatory stimuli. CRP is involved in all stages of atherosclerosis. CRP activates the classical pathway of the complement system which directly amplifies and facilitates innate immunity, that is associated with initiation and progression of CHD.[11] CRP increases LDL uptake into macrophages and enhances the ability of macrophages to form foam cells. It also binds the phosphocholine of oxidized LDL. CRP inhibits endothelial NO synthase expression in ECs. NO has important anti-atherogenic effects, including decreased platelet aggregation, vasoconstriction, and smooth muscle cell proliferation.[12] CRP activates macrophages to secrete tissue factor, which can lead to disseminated intravascular coagulation and ultimately leads to thrombosis. CRP increases the expression of adhesion molecules in ECs that can attract monocytes to the site of injury.[13] CRP increases PAI-1 expression and activity. PAI-1 is a protease inhibitor that regulates fibrinolysis by inhibiting tissue plasminogen activator. Increased PAI-1 indicates lowered fibrinolysis and thus leads to atherogenesis.[14]

CRP elevations during admission for ACS also predict long-term risk of mortality. Patients with unstable angina and CRP >3 mg/l at discharge are more likely to be readmitted for recurrent cardiovascular instability or MI within 1 year.[15] In a prospective study of patients who underwent early invasive therapy for non–ST-elevation ACS, CRP 10 mg/l during admission remained associated with increased risk of death over a mean follow-up of 20 months.[16]

Lp (a) is a low-density lipoprotein (LDL)–like particle. It is composed of triglycerides, cholesteryl esters, oxidized phospholipids, and a molecule of apo lipoprotein B-100 (apo B), that is bound to the surface by a disulphide bridge to apo lipoprotein (a) (apo (a)).[17] Lp (a) has been associated with pro-inflammatory, pro-atherosclerotic, and pro-thrombotic effects, which promote the development and progression of several cardiovascular diseases. Lp (a) particles can cross the endothelial barrier and promote atherosclerotic plaque growth. Oxidized phospholipids carried by Lp (a) trigger macrophage apoptosis and may promote atherosclerotic lesion transformation into “unstable” plaques. Lp (a) seems to contribute to arterial vessel wall inflammation by promoting monocyte cell extravasation and endothelial cell activation.[18] Mac-1 leads to nuclear factor kB (NFkB) activation and this further leads to the production of molecules that mediate the adhesion of monocytes to the endothelium and subsequent arterial wall invasion.[19] The binding of Lp (a) to fibrin prevents plasminogen activation and results in impaired clot degradation.[20] The 2019 ESC/EAS guidelines for dyslipidaemia management advice for at least one Lp (a) level assessment for every adult during their lifetime.[21] The determination of Lp (a) levels may aid in identifying patients necessitating more intensive therapeutic interventions in CAD.

METHODS

An open, prospective, observational, comparative study was conducted in Department of Biochemistry in collaboration with Department of Medicine, Guru Nanak Dev Hospital, Amritsar after taking approval from institutional ethical committee. The study group included 60 clinically diagnosed patients of coronary artery disease, attending OPD and admitted in Department of Medicine, Guru Nanak Dev Hospital, Amritsar and 60 normal healthy subjects, between age group 30-80. All patients were enrolled after taking informed consent from patients/ relatives in their vernacular language. The patients were subjected to detailed systemic examination and all subjects

included in the study were investigated for: CRP was estimated by turbidimetric immunoassay, and serum Lp (a) was also estimated by turbidimetric immunoassay.[22,23]

Lipid profile (total cholesterol was estimated by a kit based on Trinder's method.[24] Triglycerides were estimated by GPO-POD method.[25] HDL was estimated by phosphotungstic acid method, as described by Burstein et al.[26] LDL was estimated by Friedwald's and Fredrickson's Formula.[27] VLDL-cholesterol was estimated by using formula (VLDL=TG/5) based on the average ratio of triglycerides to cholesterol in VLDL.

FBS was estimated by GOD-POD method.[28] Electrocardiography (ECG) and ECHO were also estimated.

Patients were followed up for 7-10 days in hospital and then for 180 days, on as domiciliary basis. This also included modified Bruce protocol treadmill test for all cases at 1 month, to exclude the patients having severe coronary artery disease.

Inclusion criteria

The diagnosis of ACS was done on basis of clinical history or definitely abnormal electrocardiogram or abnormal echocardiography.

Family history was also noted. Detailed history of previous treatment was also noted.

Exclusion criteria

Patients who were more than 80 years and less than 30 years of age were excluded.

Patients with previous history of diabetes mellitus, infarction, collagen diseases, and uraemia or hepatic disorders were also excluded.

RESULTS

The present study included 60 clinically diagnosed cases of coronary artery disease along with equally age and sex matched controls. All the participants i.e. controls and patients were evaluated for lipid profile complete Lp (a) and CRP. The observations thus made are as follows.

Table 1 depicts comparison of lipid profile among patients of CAD and healthy subjects under study. It is observed that mean total cholesterol, TG, LDL levels, TC/HDL ratio and LDL/HDL ratio, were statistically highly significant and mean HDL levels were statistically low among patients of CAD when compared with healthy subjects.

Table 2 depicts comparison of mean levels of serum Lp (a) and serum CRP levels among patients of ACS and healthy subjects under study. It is observed that mean levels of serum Lp (a) and serum CRP were statistically highly significant among patients of CAD when compared with healthy subjects.

Table 1: Comparison of mean of values of lipid profile among patients of CAD and healthy subjects.

Parameters	Healthy subjects	Patients	P value
Total cholesterol (mmol/l)	4.46	5.28	<0.001
HDL (mmol/l)	1.06	1.01	<0.05
LDL (mmol/l)	2.81	3.49	<0.001
TG (mmol/l)	1.34	1.58	<0.05
TC/HDL ratio	4.4	5.2	<0.05
LDL/HDL ratio	2.9	3.4	<0.001

Table 2: Comparison of mean levels of serum Lp (a) and serum CRP among patients of CAD and healthy subjects.

Variables	S. Lp (a) (mg/dl)	S. CRP (mg/l)
Healthy subjects	22.9	1.26
Patients	61.8*	6.98*

*P value <0.001 when compared with normal individuals

Mean levels of S. Lp (a) and S. CRP in different groups of patients

Patients were further categorized into three groups based on serum Lp (a) and S. CRP levels: group A: S. Lp (a) levels >30 mg/dl and S. CRP >5 mg/l, group B: S. Lp (a) levels >30 mg/dl and S. CRP levels were normal, group C: S. Lp (a) levels <30 mg/dl and S. CRP >3 mg/l.

Figure 1 shows comparison of S. CRP and S. Lp (a) levels among three groups of patients under study. It is observed that mean S. Lp (a) and S. CRP levels among group A patients were significantly higher as compared to other two groups. In group B patients only S. Lp (a) levels were high and in group C patients only S. CRP levels were high.

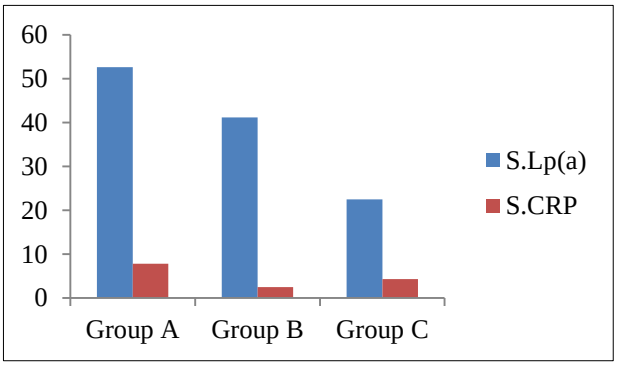

Figure 1: Mean levels of serum Lp (a) and S. CRP in different groups of patients.

Table 3 shows mean of % of hospitalization among three groups of patients of ACS under study. It is observed % of

hospitalization among group A patients were significantly higher as compared to other two groups, only 1.2% of patients need hospitalization in group B and in group C only 0.6% patients required hospitalization.

Table 3: Mean of % of hospitalization in different groups of patients of CAD.

Groups	% of hospitalization
Group A	8.2
Group B	1.2
Group C	0.6

Mean mortality rate after 180 days in different groups of patients of CAD

Figure 2 shows comparison of mean mortality rate among three groups of patients of CAD under study. It is observed that mean mortality rate among group A patients was significantly higher as compared to other two groups. In group B patients' mortality rate was 8.7 only and in group C patients it was 2.3.

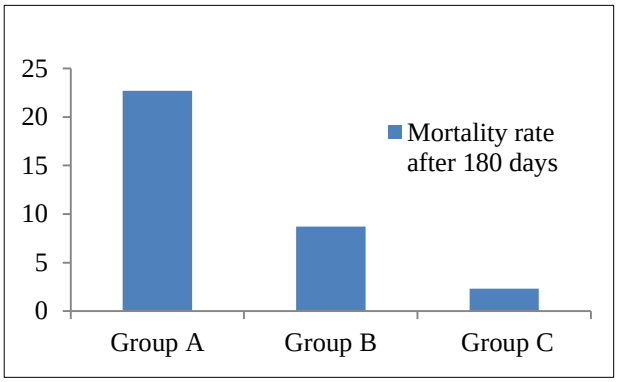

Figure 2: Mean of mortality rate after 180 days in different groups of patients of CAD.

DISCUSSION

CAD stands as a predominant contributor to global morbidity and mortality, acting as a primary catalyst for coronary heart disease-associated hospitalizations and fatalities, so causing a substantial disease burden.[29] Current study is done to evaluate the relationship between serum Lp (a) and CRP levels in coronary artery disease and their prognostic role in assessing mortality after 180 days of treatment. In this study mean values of total cholesterol, LDL, TG, TC/HDL ratio, LDL/HDL ratio were statistically highly significant and mean value of HDL was significantly low, in patients of coronary artery disease when compared with healthy subjects. In this study patients were further categorized into three groups based on serum Lp (a) and S. CRP levels: group A: S. Lp (a) levels >30 mg/dl and S. CRP >5 mg/dl, group B: S. Lp (a) levels >30 mg/dl and S. CRP levels were normal, group C: S. Lp (a) levels <30 mg/dl and S. CRP levels were >3 mg/l.

In the present study, S. Lp (a) levels >30 mg/dl were found in 55 patients and in 5 patients its levels were normal. Statistical differences between levels of S. Lp (a) in patients and healthy subjects were found to be statistically significant ($p<0.0001$). Framingham Heart Study, and atherosclerosis risk in communities (ARIC) study proved positive association of S. Lp (a) with incident coronary artery disease over long term follow up.[30,31] Kwon et al established incremental association of S. Lp (a) with poor prognosis for major adverse coronary events in patient with coronary artery disease over three years follow up, suggesting it an important risk factor.[32] Lp (a) is a powerful independent risk factor for coronary artery diseases. Its effects get multiplied by high TG, high LDL-C, and low HDL-C. This constitutes "a deadly lipid quartet".[33]

Enas has proposed a comprehensive lipid tetrad index to ascertain the total burden of dyslipidaemia in Indian population. It is calculated by the formula.

$$Lipid\ tetrad\ index = TC \times TG \times LP(a)/HDL$$

Values are taken in mg/dl. An index <10,000 is desirable, 10,000–20,000 is borderline abnormal and >20,000 is high.

In the present study, S. CRP levels ≥5 mg/dl were found in 37 patients. Among all 60 patients, 8 patients had normal S. CRP levels and in 15 patients S. CRP levels were between 3-5 mg/l. Statistical differences between levels of S. CRP in patients and healthy subjects were found to be statistically highly significant ($p<0.0001$). A study by Mach et al showed that the S.CRP concentration at the time of admission was significantly higher among patients with ACS as compared with patients with low S. CRP levels.[34] Karki et al reported that significantly high S. CRP levels of >5 mg/l were present in patients and S. CRP is a useful tool for predicting mortality during the hospital stay and at 6 weeks.[35] Out of 60 patients, 44 patients needed hospitalization and needed urgent treatment and admission rate was low in group B and group C patients. The mean mortality rate after 180 days of follow up, in group A patients was 22.7% and in group B patients was 8.7% and in group C patients was 2.3% which shows that mortality rate was higher in patients with high levels of both S. CRP and S. Lp (a) and mortality rate is low in patients with raised one parameter only either S. CRP or S. Lp (a). In individuals without known cardiovascular disease, the presence of a pro-inflammatory status i.e. high S. CRP levels, has been found to increase the risk of cardiovascular events associated with high Lp (a) levels.[36] The risk of mechanical consequences and complications of ischemic injury increases with the intensity of the inflammatory response. Increased concentrations of S. CRP carry a high risk of developing heart failure after ACS and prompt regular screening and more aggressive therapy is required. On admission, independent prognostic information was provided by the assessment of S. CRP and so the ability to identify patients with the highest risk of death and heart failure was improved. So the quantification

of both S. Lp (a) and S. CRP may help to estimate residual cardiovascular disease risk.

CONCLUSION

Patients with high levels of S. Lp (a) and S. CRP need aggressive cardiac management and close monitoring after discharge. In patients with suspected CAD, the measurement of S. CRP and S. Lp (a) levels should be done at the time of admission. This study tells that elevated S. Lp (a) and S. CRP levels independently predict complications and all-cause mortality in CAD patients. Patients with high levels of S. CRP and S. Lp (a) had high mortality rate so they are proven indicators of atherosclerosis. Lifestyle, diet, and medical interventions, has minimal effect on Lp (a) levels. Measurement of S. CRP and S. Lp (a) has potential to enhance risk stratification in ACS diagnosed patients. Further investigations should be done to establish S. Lp (a) and S. CRP's role in refining risk stratification and modifying residual risk in coronary artery disease diagnosed individuals.

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