Original Research Article

# A comparative study of serum lipoprotein 'a' [S(Lp(a)] in type - II diabetic v/s nondiabetic patients with ST-Elevation Myocardial Infarction (STEMI)

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## Abstract

**Aim:** The study was aimed to compare the serum levels of Lp(a) in between diabetic and Nondiabetic patients who presented with STEMI and to study the correlation if any between dyslipoprotien(a)emia and dyslipidemia in patients with acute STEMI(Diabetics and Nondiabetics). **Methods:** All individuals with established DM-2 along with acute STEMI and without any major overt complication were recruited from cardiology outdoor, medical outdoor and indoor for the study. The subjects were of middle and elderly age group. Complete history and comprehensive clinical examination along with all the planned investigations were alone. Each subject was investigated for glycaemic status, ECG changes, lipid profile and Lp(a) estimation. **Results:** The S.Lp(a) level were higher in nondiabetic group(36.18±19.96 mg/dl) compared to diabetic group(31.53±8.39 mg/dl) but the difference was not statistically significant(p>0.1). Also the lipid profile of these patients was deranged and there was a fair correlation between lipid variables and Lp(a). **Conclusion:** The prevalence of dyslipidemia and dyslipoprotein(a)emia is very high in those presenting with acute STEMI. All lipid variables were found to be raised except HDL along with S.Lp(a), which was found to be higher in nondiabetic patients compared to diabetics in acute STEMI but the values were significantly higher then the normal values.

**Key Words:** S.Lipoprotien-a, Dyslipidemia, Dyslipoprotienemia, DM -2, ST elevation MI.

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## INTRODUCTION

Coronary Artery Disease (CAD) is the commonest of all cardiac diseases and is the leading cause of morbidity and mortality. WHO has defined CAD as "impairment of heart function due to inadequate blood flow to the heart compared to its needs caused by obstructive changes in coronary circulation. The spectrum of CAD represent a pathological, diagnostic and risk continuum from chronic stable angina, unstable angina (USA), myocardial infarction (MI) to sudden death. The prevalence of CAD amongst diabetics in India is on the verge of rise. The diabetic state per se is the major determinant for developing CAD amongst diabetics and thus the AHA has correctly conferred that DM is a coronary equivalent and should be treated as aggressively as nondiabetics with atherogenic dyslipidemias. Atherosclerotic risk factors or cardiovascular risk factors that have emerged from studies like"community based Framingham Heart study " and seven case control studies performed by Key and colleagues are grouped into 2 categories:-Modifiable: Smoking, HTN, dyslipidemia, DM, Obesity, sedentary life style.Non Modifiable: Age, Sex, generation. Other so called "Novel risk factors" have emerged which include total plasma homocysteine, fibrinogen, lipoprotein-a [Lp

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(a)],fibrinolytic function as ascertained by tpA or PAI-1 antigens and markers of low grade inflammation such as hs-CRP. Lp(a) is a cholesterol ester rich lipoprotein composed of a particle of LDL molecule bound by sulphydryl linkage to the apoprotein a which is known to be very heterogenous glycoprotein that shows at least 75% homology with plasminogen. This apoprotein 'a' may inhibit fibrinolysis by competing with plasminogen. This strong homology of Lp (a) and plasminogen suggest that Lp(a) might represent the ideal bridge between the field of atherosclerosis and thrombosis in the pathogensis of IHD. High concentration of Lp(a) in plasma are associated with CAD and early MI. Several studies have considered Lp(a) as an independent and most prevalent risk factor for IHD. The situation is less clear in DM. Initial studies did report elevated Lp (a) in metabolically poorly controlled DM-2 (Bruckert et at;1990). But later studies reported that Lp (a) is not elevated in patients will DM-2. Studies have shown higher levels (Ramirez LL *et al* 1992); no difference (Haffner *et al* 1992); even lower levels (Rainwater DL *et al* 1994) in DM-2. The consensus appear to be that the diabetic state does not have any impact on Lp (a) concentration (Haffner *et al* 1993) though diabetic patients with CHD are found to have higher Lp (a) levels than diabetic patients without CAD (Comlecki *et al* 1997). In one study Lp (a) was found to be higher in those with CAD and Proteinuria with no association with retinopathy or PVD(Deepa R *et al* 2002). Similar study carried out by Lakhotia *et al* found that Lp (a) was significantly higher in diabetic patients with retinopathy but no association with retino or neuropathy. To the best of our knowledge there are only a few studies in India concerning Lp(a) levels in DM-2 with acute MI. Hence this study has been planned to study Lp (a) level in DM-2 with MI and to compare it with patients without DM and with MI.

## MATERIALS AND METHODS

Briefly all patients with well established DM-2 along with acute MI and without any major overt complication were included in the present study. The patients were ineligible if they-1Were suffering from diseases that directly affect the Lp(a) levels and serum lipid levels. Like End Stage Renal Disease (ESRD), nephrotic syndrome, severe liver diseases, Patients with malignancy, those with thyroid diseases, primary gout or with pregnancy. 2.Were on drugs which affect serum lipid levels like estrogen, progesterone, anabolic steroids, OCP',s glucocorticoids, cyclosporine and various lipid lowerling drugs like statins, fibric acid derivaties, nicotinic acid, neomycin etc. 3.Other causes of ST elevation like ventricular aneurysm, early repolarization synd, Prinzmetal angina etc. 4.Alcoholics 5.Patients will long standing uncontrolled diabetes. 6.Patients with overt complication of either MI or diabetes. All the patients after their informed consent were enrolled in the study and thoroughly evaluated including history, physical examination and detailed cardiovascular examination. The Patients were grouped into Group I (n=50): patient with acute STEMI without DM-2 and Group II (n=50): patient with acute STEMI with DM-2. Along with all routine investigations, some special investigations like complete lipid profile (S.cholesterol, TG, HDL,LDL,VLDL) by enzymatic kinetic assay method and S.Lipoprotein(a) level estimation by Turbilatex method were done.

**Calculation**-The absorbance difference A2-A1 for each Lp(a) calibrator will be calculated and the value found against the Lp (a) concertration will be plotted in a calibration curve. Lp(a) concertration in the sample will be calculated by interpolation its $A_2$-$A_1$ vales on the calibration curve

## RESULTS

A total of 100 patients of acute STEMI, 50 of whom are diabetic and 50 are nondiabetic without any major overt complication were studied. A total of 40(80%) males and 10(20%) females in group I and 36 (72%) males and 14 (28%) females in group II were included in the study. Majority of the subjects were in age group 51-60yrs (32%) in both the groups. The male: female ratio in nondiabetic group was 4:1 and in diabetic group was 2.57:1 respectively.

**Table 1:** Distribution of nondiabetic V/S diabetic subjects with Acute STEMI with regard to serum Lp(a) levels

| Serum Lp(a) level (mg%) | Non Diabetic | | Diabetic | |
|---|---|---|---|---|
| | No. | % | No. | % |
| <10 | 0 | 0 | 0 | 0 |
| 10-20 | 12 | 24 | 2 | 4 |
| 20-30 | 8 | 16 | 16 | 32 |
| >30 | 30 | 60 | 32 | 64 |
| **Total** | **50** | **100** | **50** | **100** |

In the nondiabetic group only 8 (16%) out of 50 had serum Lp(a) levels above 20mg% but below 30mg% and 30 (60%) out of 50 subject had Lp (a) levels >30 mg%. In comparision in the diabetic group 16 (32%) out of 50 subject had SLp(a) >20mg% and 32 (64%) had levels >30mg%. The mean Lp(a) for nondiabetic was 36.18±8.39 mg%. The p values was statistically non significant (p>0.1).

**Table 2:** Distribution of nondiabetic V/S diabetic subjects with acute STEMI with regard to their serum cholesterol and Lp(a) levels

| Total Cholesterol (mg %) | | Non diabetic Serum Lp(a) (mg%) <30 | >30 | Total | Diabetic Serum Lp(a) (mg%) <30 | >30 | Total |
|---|---|---|---|---|---|---|---|
| <200 | No | 12 | 20 | 32 | 6 | 4 | 10 |
| | % | 24 | 40 | 64 | 12 | 8 | 20 |
| >200 | No | 8 | 10 | 18 | 12 | 28 | 40 |
| | % | 16 | 20 | 36 | 24 | 56 | 80 |
| **Total** | **No** | **20** | **30** | **50** | **18** | **32** | **50** |
| | **%** | **40** | **60** | **100** | **36** | **64** | **100** |

The serum cholesterol level was elevated in 40 subjects (80%) out of 50 in the diabetic in comparision to serum Lp (a) level which was elevated in 32 patients (64%) out of 50. In the nondiabetic group serum Cholesterol was elevated in 18 (36%) in camparision to serum Lp (a)which was elevated in 30 (60%) out of 50 subjects.

**Table 3:** Distribution of nondiabetic V/S diabetic subjects with acute STEMI with regard to their serum triglyceride and Lp(a) levels

| Total triglyceride (mg %) | | Non diabetic Serum Lp(a) (mg%) <30 | >30 | Total | Diabetic Serum Lp(a) (mg%) <30 | >30 | Total |
|---|---|---|---|---|---|---|---|
| <150 | No | 10 | 18 | 28 | 4 | 4 | 8 |
| | % | 20 | 36 | 56 | 8 | 8 | 16 |
| >150 | No | 10 | 12 | 22 | 14 | 28 | 42 |
| | % | 20 | 24 | 44 | 28 | 56 | 84 |
| **Total** | **No** | **20** | **30** | **50** | **18** | **32** | **50** |
| | **%** | **40** | **60** | **100** | **36** | **64** | **100** |

The serum TG levels were elevated in 42 (84%) subjects out of 50 in the diabetic group in comparison to serum Lp (a) which was elevated in 32 (64%) out of 50. In nondiabetic groups TG was elevated in 22 (44%) subjects in comparision to Lp(a) which elevated in 30 (60%) out of 50.

**Table 4:** Distribution of nondiabetic V/S diabetic subjects with acute STEMI with regard to their serum LDL and Lp(a) levels

| Total LDL (mg %) | | Non diabetic Serum Lp(a) (mg%) <30 | >30 | Total | Diabetic Serum Lp(a) (mg%) <30 | >30 | Total |
|---|---|---|---|---|---|---|---|
| <130 | No | 10 | 16 | 26 | 16 | 14 | 30 |
| | % | 20 | 32 | 52 | 32 | 28 | 60 |
| >130 | No | 10 | 14 | 24 | 2 | 18 | 20 |
| | % | 20 | 28 | 48 | 4 | 36 | 40 |
| **Total** | **No** | **20** | **30** | **50** | **18** | **32** | **50** |
| | **%** | **40** | **60** | **100** | **36** | **64** | **100** |

The serum LDL levels were elevated in 20 (40%) subjects out of 50 in the diabetic group in comparison to serum Lp(a) which was elevated in 32 (64%) out of 50. In nondiabetic groups LDL was elevated in 24 (48%) subjects in comparision to Lp (a) which elevated in 30 (60%) out of 50.

**Table 5:** Distribution of nondiabetic V/S diabetic subjects with acute STEMI with regard to their serum HDL and Lp(a) levels

| Total HDL (mg %) | | Non diabetic Serum Lp(a) (mg%) <30 | >30 | Total | Diabetic Serum Lp(a) (mg%) <30 | >30 | Total |
|---|---|---|---|---|---|---|---|
| >40 | No | 14 | 18 | 32 | 6 | 14 | 20 |
| | % | 28 | 36 | 64 | 12 | 28 | 40 |
| <40 | No | 6 | 12 | 18 | 12 | 18 | 30 |
| | % | 12 | 24 | 36 | 24 | 36 | 60 |
| **Total** | **No** | **20** | **30** | **50** | **18** | **32** | **50** |
| | **%** | **40** | **60** | **100** | **36** | **64** | **100** |

The serum LDL levels were elevated in 30 (60%) subjects out of 50 in the diabetic group in comparison to serum Lp (a) which was elevated in 32 (64%) out of 50. In nondiabetic groups HDL was elevated in 18 (36%) subjects in comparision to Lp (a) which elevated in 30 (60%) out of 50.

## DISCUSSION

The present study was conducted to compare serum levels of Lp (a) in diabetics and nondiabetics who presented with acute STEMI. This was carried out on 50 patients of acute STEMI with diabetes and compared with 50 non diabetics who also presented with STEMI. Both the groups were age and sex matched. In the present study the age of patients varied between 31 yrs to 100yrs. The mean age of diabetic group was 58.8±11.45yrs and that of nondiabetic group was 58.48±15.16yrs. In both the groups most of the subjects belonged to 51-60 yrs(32% in both the groups). None of the patients were <30yrs and only two patients (4%) belong to age group 91-100yrs. Both the groups were adequately age matched. When diabetics and nondiabetics are compared in terms of SLp (a) in the present study it was found that in the diabetic group 16 subjects(32%) had SLp (a) above 20mg% but below 30mg%, while 32 (64%) had SLp(a) above 30mg%. In the nondiabetic group 8 subjects (16%) had serum Lp (a) above 20mg% but below 30mg% while 30subjects (60%) had serum Lp (a) above 30mg%. The mean value of Lp (a) in diabetics was 31.53±8.3 mg% and that for nondiabetics was 36.18±19.96mg. The p value was not significant (p>0.1). In a study done by D.Rajasekhar *et al* 2004, which evaluated the levels of S Lp (a) in angiographically proved coronay heart disease found a statistically significant difference in levels of SLp (a) between cardiac patients and controls (24.79±18.99 mg% VS 16.04±17.53 mg; p<0.01) but at the same time the mean Lp (a) level in CAD patients who were diabetic and those who were nondiabetic, value was not statistically significant (24.27±18.94 V/S 25.05±19.4 mg% respectively). Similar study done by Holanda *et al*, found no significant difference in levels of of SLp(a) in diabetic and nondiabetic patients suffering from acute ischemic stroke.(24.49±23.09 V/S 44.81±44.34 mg% respectively) Dr. Ghaffarzadegan *et al* in their comparative study in patients with AMI and controls found that mean Lp(a) concentration in patients was 49.18±47.44 mg/dl and that of controls was 37.94±4.9 mg/dl. The difference was statistically significant (p=0.018). They also concluded that mean Lp (a) in women was higher than men included in the study. (50mg/dl V/S 37.1 mg/dl respectively) Above mentioned studies data and present study establish the significance of elevated Lp(a) as an independent, potential and modifiable coronary risk factor without many of the other conventional risk factors, as Lp (a) has both atherogenic and thrombogenic property by accumulating in the vessel wall leading to atherosclerosis and stimulating the secretion of plasminogen activator inhibitor, thus interfering with fibrinolysis. But the levels of Lp(a) do not significantly differ among diabetics with coronary heart disease. Thus the present study findings are consistent wth the established role of Lp (a) in the causation of Ischemic heart disease.

## CONCLUSION

In the present study serum cholesterol, S.Triglycerides, S.LDL, S.VLDL levels were found to be raised along with low levels of S.HDL in diabetic patients.The levels of S.Lp (a) were found to be higher in nondiabetic patients compared to diabetic patients in acute STEMI but the values were significantly higher then the normal values. There were no significant differences between diabetic and nondiabetic patients SLp (a) levels which indicates that elevated SLp (a) levels were associated with acute STEMI irrespective of the presence of DM-2. The distribution of Lp (a) levels was higly skewed towards the higher levels in both being above 30mg/dl in 60% of both diabetic and nondiabetic patients with acute STEMI. Thus SLp (a) is clearly a more powerful, strong and independent risk factor for causing ischemic heart disese. The situation is less clear in DM. According to the statistical data collected from the present study it is now clear that diabetic state per se does not have impact on Lp (a) coneentration though diabetic patients with CAD are found to have higher Lp (a) levels than without CAD. IHD places a tremendous burden on health resources throughout the word. Improved detection and modification of risk factors could reduce the impact of this diease. In our study elevated Lp (a) levels were associated with acute STEMI irrespective of the presence of type 2 DM, so if Lp (a) level is elevated, it seems reasonable to check other major vascular risk factors.

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