Original article:

Study of association between postpranadial lipemia and coronary artery disease in Indian Population

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Abstract:

Introduction: Clinical data show a correlation between postprandial lipoproteins and the

Presence/progression of coronary artery disease. With this background the present work was planned to study

correlation of postprandial lipemia and coronary artery disease in Indian Population.

Material & methods: In present study 50 patients of coronary artery disease who have undergone coronary angiography where selected for over a period of Dec.2003 to may 2005 with reference to clinical profile and lipid profile.

Observations and results: 10(20%) patients shows hypertirglyceridemia in the fasting state while 34(68%) of patients shows hypertriglyceridemia (TG > 200mg%) in their postprandial state.

Conclusion: This study indicates that prolonged postprandial lipemia is an important risk factor for CHD.

Introduction

Postprandial lipemia, characterized by a rise in triglycerides rich lipoproteins after eating, is a dynamic, nonsteady--- state condition in which humans spend the majority of time.There are several lines of evidence suggesting that postprandial lipemia increases the risk of atherogenesis.[1] Clinical data show a correlation between postprandial lipoproteins and the presence/progression of coronary artery disease.[2]Mechanistic studies demonstrate that triglycerides rich lipoprotein remonants may have adverse effect on endothelium and can penetrate into the sub endothelial space. Exchange of core lipids between postprandial lipoproteins and low density lipoprotein (LDL)/ high density lipoproteins (HDL) increased during prolonged lipemia, resulting in small, dense, LDL particles and reduced HDL cholesterol levels. Hemostatic variables, including clotting factors, platelet reactivity and monocyte cytokine expression, may be increased during p postprandial lipemia.In the present study, we evaluated correlation between postpranadial lipemia and coronary artery disease in Indian Population.[3]

Material & methods:

In present study 50 patients of coronary artery disease who have undergone coronary angio-graphy where selected for over a period of Dec.2003 to may 2005 with reference to clinical profile and lipid profile.

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Inclusion Criteria: All patients of coronary artery disease who have undergone coronary angiography irrespective of their age and sex.

Exclusion Criteria:

      Hepatic dysfunction

      Acute & chronic pancreatitis

      Deranged renal function

      Congenital heart disease (CHD)

      Diabetics moieties on insulin

      Uncontrolled HTN

Coronary Angiography:

Coronary angiography remains the "gold standard" for identifying the presence absence of stenosis due to coronary artery disease and it provides the most reliable anatomical information for determining the appropriateness of medical therapy. Percutaneous coronary intervention of coronary artery bypass graft surgery in patient with coronary.

Sample size was determined by expert statistician.

Observations

1. Comparison of fasting serum cholesterol in significant coronary involvement and nonsignificant or normal coronaries.

No. of patients with	100-200mg%	>200mg%	Total
Significant coronary involvement	25	13	38
Non significant/normal coronary	09	03	12
Total	34	16	50

2. Comparison of postprandial serum cholesterol in significant coronary involvement and nonsignificant or normal coronaries.

No. of patients with	100-200mg%	>200mg%	Total
Significant coronary involvement	14	27	41
Non significant / normal coronary	09	00	09
Total	23	27	50

3. Comparison of fasting triglyceride in significant coronary involvement and non significant or normal coronaries.

No. of patients with	100-200mg%	>200mg%	Total
Significant coronary involvement	30	08	38
Non significant / normal coronary	10	02	12
Total	40	10	50

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4.Comparison of postparandial triglyceride in significant coronary involvement and non significant or normal coronaries.

No. of patients with	100-200mg%	>200mg%	Total
Significant coronary involvement	29	09	38
Non significant / normal coronary	07	05	12
Total	36	14	50

5.Comparison of fasting HDL cholesterol in significant coronary involvement and non significant or normal coronaries.

No. of patients with	100-200mg%	>200mg%	Total
Significant coronary involvement	20	18	38
Non significant / normal coronary	02	10	12
Total	22	28	50

6.Comparison of postprandial HDL cholesterol in significant coronary involvement and non significant or normal coronaries.

No. of patients with	100-200mg%	>200mg%	Total
Significant coronary involvement	24	14	38
Non significant / normal coronary	03	09	12
Total	27	23	50

7.Comparison of fasting LDL cholesterol in significant coronary involvement and non significant   or normal coronaries.

No. of patients with	70-100 mg%	100-130 mg%	>130mg%	Total
Significant coronary involvement	15	16	07	38
Non significant / normal coronary	07	04	01	12
Total	22	20	08	50

8.Comparison of postprandial LDL cholesterol in significant coronary involvement and non significant or normal coronaries.

No. of patients with	70-100 mg%	100-130 mg%	>130mg%	Total
Significant coronary involvement	06	20	12	38
Non significant / normal coronary	02	09	01	12
Total	08	29	13	50

9.Comparison of fasting VLDL cholesterol in significant coronary involvement and non significant or normal coronaries.

No. of patients with	15-40mg%	>40mg%	Total
Significant coronary involvement	34	04	38
Non significant / normal coronary	11	01	12
Total	45	05	50

10. Comparison of postprandial VLDL cholesterol in significant coronary involvement and non significant or normal coronaries.

No. of patients with	100-200mg%	>200mg%	Total
Significant coronary involvement	16	22	38
Non significant / normal coronary	09	03	12
Total	25	25	50

Discussion

Definition of abnormal postprandial lipemia—postprandial hypertriglyceridemia as any postprandial TG concentration (at 4.6 or 8 hours) higher than the highest TG concentration in any hour in any control individual. [4,5]This TG value was 219mg%. The present study of 50 patient was indented to evaluate risk factors associated with coronary artery disease, clinical profile, lipid profile and relation between coronary angiography profile and lipid profile.16(32%)

patients shows hypercholesterolemia in fasting state while 27(54%) patients shows hypercholesterolemia (Sr. Chol 200mg%) in their postprandial state i.e. after 8 hours of their major meal. This agrees with Jaychandran et al 1987 have found that 43% of CHD have hypercholesterolemia. Wasir et al found 44% hyperlipidemia, to be the commonest risk factor of CHD.

10(20%) patients shows hypertirglyceridemia in the fasting state while 34(68%)

of patients shows hypertriglyceridemia (TG > 200mg%) in their postprandial state. Cauilard et al reported a significant association between the magnitude of the postprandial aTG response and fasting plasma HDL cholesterol concentration. However, the subject included in their study shown a wider range of fasting TG concentration (44-390mg%) while in our study it was 85-280mg%). Axelson et al showed 50% greater TG response in CHD. The ARIC study extent that finding to a large sample men and women and support the interpretation of Axelson et al that rise in lipoprotein is of intestinal origin.26(52%) patient shows HDL<mg% in their fasting state and 24(48%) were shows HDL <30mg% in their postprandial state. This agrees with the inverse relation between risk of CHD and HDL level, in the PROCAM study 45% of subjects developed CHD had HDL cholesterol <30mg%.

23(46%) patient shows LDL more than 180 mg% in their fasting state while 26(52%) of patients shows LDL > 100mg% in their postprandial state. 5(10%) of patient in this study shows fasting VLDL >40mg% while 25(50%) shows same in their postprandial state.

In the present study criteria of 70% or more of major coronary artery except left main (50% or more) was selected which was also the criteria in one and the biggest coronary angiographic study CASS.[7]In present study 31 male (62%) and 7 female (14%) total 76% patients of CHD shows significant coronary involvement and 10(20%) patients shows normal coronaries.Percentage of insignificant coronary involvement was 2%.

This is an agreement with other studies conducted by Warren al 1979, Glover et al 1982, Kaul et al 1985, which shows that significant obstructive disease constitute largest group and there is definite male preponderance.In this study patients with significant coronary angiographic involvement shown serum cholesterol 260 ± 10, TG 230 ± 12, HDL 28 ± 2, LDL 92 ± 5, while the profile in normal angiography, serum cholesterol 140 ± 10, TG 160 ± 10, HDL 30 ± 7, LDL 65 ± 5, VLDL 20 ± 5. Suggestive exaggerated lipoprotein levels and especially in postprandial state in significant coronary angiographic involvement.[6]This agrees with ARIC study (The Atherosclerosis Risk In Communities). In the ARIC study they consider carotid thickness and derange lipid profile. There were 602 participants greater postprandial response in men exceeded that in women by 32%.

Conclusion:

This study indicates that prolonged postprandial lipemia is an important risk factor for CHD.

Bibliography

1. Abrems JJ, Grundy SM, Ginsberg H. Metabolism of Plasma TG in Hypothyroitism and hpertthyroidism in man. J Lipid Res. 1981;22;307-322
2. Assmann G., Schulte H., Relation of HDL cholesterol and TG to incidence of atherosclerotic coronary artery disease (the PROCAM Experience). Prospective cardiovascular nounstar study. Am J. Cardiol 1992-70-733-737.
3. Autin MA, Brunzell J.D., Fitch WL, Krauss RM, Inheritance of low density lipoprotein subclass patterns in familial combined hyperlipidemia. Athorosclorosis 199000;10;520-530.

4. Axelson M, Eliasson B, Joheim E, Lenner RA, Taskinon MR, Smith U. Lipid intolerance in smokers. J. Intern. Med. 1995;237;449-455

5. Dianne C. Hyson PHD, John C. Routlodge MD, and Lars Berglund MD PHD, Current Authorosclerosis Repaort 2003, 5:437-444.

6. Genovofa D. Kibvou, Katherine K. Anagnostopoulou, Noktaros Pilatis, Nikolaos, Kafaltis, Konstandine, Sordila, Elteherios Psarro and Dennis V. Cokkinos, Lo fasting low high density lipoprotein and pastprandial lipemia, Lipids in Health & Disease 2004, 3:18.

7. Glover AV, Kuber M.T., Warren S.E., Vieweg WVR, Myocardial infarction before age 36: Risk factor and artiographic analysis. Am. J. of Cardiol, 49, 1600-1982.