

An Association of High Sensitive C Reactive Protein and Lipid Profile Parameters in South Indian Population

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Abstract- C Reactive Protein (CRP) is an acute phase protein whose levels are shown to be elevated in inflammation. Atherosclerosis is known to involve inflammation and dyslipidemia is a triggering factor in its development. In this study we tried to address any association that might exist between high sensitive C reactive protein (HSCRP) and lipid profile. In this study HSCRP and the Lipid profile parameters were estimated in thirty two patients who visited the Kasturba Hospital, Manipal. The results obtained indicate a strong significant positive correlation of total cholesterol (p value =0.0001) and triglycerides (p value= 0.023) with HSCRP and a negative correlation of HDL (p value =0.16) with the same. These results indicate that there may be a role for HSCRP in screening and risk stratification of atherosclerosis.

Index Terms- HSCRP, Lipid profile, atherosclerosis

I. INTRODUCTION

Despite great progress in the field of pharmacological therapy chronic conditions like coronary syndromes (ACS), diabetes mellitus, obesity, metabolic syndrome remain the major cause of mortality and morbidity in the modern world. Inflammation plays a key role in the initiation and progression of these conditions.[1,2] C-reactive protein (CRP) is a highly conserved plasma protein that participates in the systemic response to inflammation. It is an excellent biomarker for acute-phase response and has proved to be an important and characteristic predictor of future cardiovascular disease and metabolic abnormalities in overtly seen healthy men and women. [3,4]Also, it is widely accepted that increased levels of low-density lipoprotein(LDL), triglycerides (TGs), total cholesterol (TC) and decreased levels of high density lipoprotein (HDL) are associated with atherosclerosis that may lead to predisposition to other acute coronary syndromes.[5] LDL-a component of lipid profile is known to activate a cascade of local inflammation which can lead to formation of atherosclerotic plaques.[6] Even though both CRP and Lipid Profile parameters have a role in initiation and progression of atherosclerosis, no data is available regarding the correlation between these 2 entities with respect to the risk stratification of atherosclerotic diseases. Therefore the present study is conducted to compare the HSCRP levels with the lipid profile parameters and to check any existing correlation between them.

II. MATERIALS AND METHODS:

Study design:

Ethical Clearance for this study was obtained from the Institutional Ethics Committee, Kasturba Medical College, Manipal. Thirty two individuals not suffering from any infections or having an active inflammatory condition between the age group 25 - 80 that visited the Kasturba Hospital, Manipal were included in this study. The samples were collected in plain vacutainers and were assayed for the Lipid profile and HSCRP.

Biochemical Parameters:

The estimation of the Lipid profile involving total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and triglycerides was based on the enzymatic colorimetric test using the Roche cobas e600 analyzer . The HSCRP levels were analyzed by sandwich ELISA technique using HSCRP kit.

III. RESULTS

The statistical analysis was done using the SPSS 16 software. Since the HSCRP values did not follow normal distribution, regression analysis was done keeping square root HSCRP as the dependent variable.

The results shown in Table 1 indicate a moderately strong significant correlation between HSCRP and Triglycerides with a R value of 0.35 and p value of 0.023. Also, a very strong and significant positive correlation is seen between HSCRP and Total Cholesterol with a p value of 0.0001. Alternatively a weak negative correlation is seen between HDL and HSCRP and a weak positive correlation between HSCRP and LDL.

Table 1 : The following table shows the comparison of HSCRP with the Lipid profile in terms of the Pearson coefficient and the p value obtained by regression analysis. (N=32)

PARAMETER	R VALUE*	p VALUE**
Triglycerides	0.35	0.023
Total cholesterol	0.6	0.0001
HDL	-0.18	0.16
LDL	0.20	0.14

* Pearsons coefficient

** p value obtained by regression analysis (significant if p value < 0.05)

IV. DISCUSSION AND CONCLUSION

This study showed a strong, significant positive correlation between the serum HSCRP Levels with the serum cholesterol and triglycerides. Also a weak negative correlation is seen between the serum HSCRP levels and HDL. LDL on the other hand showed a very weak positive correlation with serum HSCRP. These findings support the hypothesis that dyslipidemia can induce an inflammatory reaction which is a hall mark of atherosclerosis. CRP is an acute phase protein which is produced shortly after an inflammatory stimulus from the hepatocytes. Several cytokines like IL-1, IL-6 and TNF- α that are secreted locally in the area of the harmed tissue regulate the production of CRP.[7] According to Danish et al Obesity, cardiovascular diseases, metabolic syndrome and Type 2 diabetes mellitus are associated with high HSCRP levels as there is low grade systemic inflammation in these conditions[8,9,10].

Also, studies have shown levels of LDL, triglycerides and total cholesterol are associated with atherosclerosis.[11] The transport vehicle of cholesterol and other lipids in body is low density lipoprotein (LDL). Once oxidized, LDL is called small dense LDL and which can trigger a low grade local inflammation leading to cytokine release. Phagocytosis of oxidized LDL by monocytes transforms them into foam cells with a lipid core which is the beginning of atherosclerotic plaque formation.

Next, the storage site of triglycerides is mainly adipose tissue which was earlier considered to be a passive organ is now known to express the proinflammatory cytokine IL6. Marjolein et al suggested that excess loading of triglycerides in adipose tissue as seen in obesity can cause release of IL6 by adipose tissue which can be involved in induction of low grade systemic inflammation[12].

High serum level of high density lipoprotein (HDL) on the other hand is associated with reduced risk for development of atherosclerotic disease. HDL particles are believed to be anti atherogenic and antagonized pathways of inflammation, thrombosis and oxidation[13].

The data obtained from the study therefore supports the theory that serum LDL, total cholesterol and triglycerides were higher and HDL was lower in individuals with HSCRP on the higher side of the normal range suggesting a low grade systemic inflammation. These results indicate that there may be a role for HSCRP in screening and risk stratification of atherosclerosis. Also this suggests that there may be a role for anti-inflammatory agents along with statins in treatment of dyslipidemia.

One of the limitations of the study is that the small sample size due to which perhaps the correlation between HSCRP and LDL could not be proved very significant. We did not consider whether any of the patients were on hypolipidemic drugs which could have influenced lipid profile values. So, we could not comment on the effects of hypolipidemic drugs on HSCRP and inflammation.

REFERENCES

[1]. V. L. Roger, A. S. Go, D. M. Lloyd-Jones et al., "Executive summary: heart disease and stroke statistics-2012 update: a report from the American heart association," *Circulation*, vol.125, no. 1, pp. 188–197, 2012.

[2] S. Kaptoge, E. Di Angelantonio, G. Lowe et al., "C-reactive protein concentration and risk of coronary heart disease, stroke, and mortality: an individual participant metaanalysis," *The Lancet*, vol. 375, no. 9709, pp. 132–140, 2010.

[3] Van Leeuwen M, Van Rijswijk M. Acute phase proteins in the monitoring of inflammatory disorders. *Baillieres Clin Rheumatol*.1994;8(3):531–52.

[4] Carlson CS, Aldred SF, Lee PK, Tracy RP, Schwartz SM, Rieder M, et al. Polymorphisms within the C-reactive protein (CRP) promoter region are associated with plasma CRP levels. *Am J Hum Genet*. 2005;77(1):64–77.

[5] Graham I. European guidelines on cardiovascular disease prevention in clinical practice: executive summary. *Atherosclerosis* 2007; 194: 1-45.

[6]. Paweł Burchardt, Jakub Żurawski, et al. "Low-density lipoprotein, its susceptibility to oxidation and the role of lipoprotein-associated phospholipase A2 and carboxyl ester lipase lipases in atherosclerotic plaque formation". *Arch Med Sci* 1, February / 2013

[7] Konstantinos Kitsios¹, Maria Papadopoulou², Konstantina Kosta², et al. "High-Sensitivity C-Reactive Protein Levels and Metabolic Disorders in Obese and Overweight Children and Adolescents". *J Clin Res Pediatr Endocrinol* 2013;5(1):44-49

[8] Danesh J, Wheeler JG, Hirschfield GM, Eda S, Eiriksdottir G, Rumley A, Lowe GD, Pepys MB, Gudnason V. C-reactive protein and other circulating markers of inflammation in the prediction of coronary heart disease. *N Engl J Med* 2004;350:1387-1397.

[9]. Florez H, Castillo-Florez S, Mendez A, Casanova-Romero P, Larreal-Urdaneta C, Lee D, Goldberg R. C-reactive protein is elevated in obese patients with the metabolic syndrome. *Diabetes Res Clin Pract* 2006;71:92-100.

[10]. Kahn SE, Zinman B, Haffner SM, O'Neill MC, Kravitz BG, Yu D, Freed MI, Herman WH, Holman RR, Jones NP, Lachin JM, Viberti GC; ADOPT Study Group. Obesity is a major determinant of the association of C-reactive protein levels and the metabolic syndrome in type 2 diabetes. *Diabetes* 2006;55:2357-2364.

[11] Paweł Burchardt, Jakub Żurawski, Bartosz Zuchowski, et al. "Low-density lipoprotein, its susceptibility to oxidation and the role of lipoprotein-associated phospholipase A2 and carboxyl ester lipase lipases in atherosclerotic plaque formation" .*Arch Med Sci* 2013; 9, 1: 151-158.

[12]. Marjolein Visser, Mark H, et al. "Elevated C Reactive Protein levels in overweight and obese adults". *JAMA*, December 8, 1999. vol 282: 22

[13]. HASEEB A. KHAN, ABDULLAH S. ALHOMIDA, ET AL. " LIPID PROFILE OF PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND ITS CORRELATION WITH SYSTEMIC INFLAMMATION"
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