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High-Sensitive C–Reactive Protein with Lipid Profile Parameters in Dyslipidemic Patients: A Teaching Hospital Based Study.

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ABSTRACT

Background: Cardiovascular diseases, the leading causes of death in the world are rising rapidly in low- and middle-income countries. Our aim was to fine out the correlation between High-Sensitive C–reactive Protein with Lipid Profile Parameter. **Methods: T**he strong and statistically significant positive correlation in between High-Sensitive C–reactive Protein (hsCRP) and Total Cholesterol with (p<0.016). **Results:** Statistically significant positive correlation between High-Sensitive C–reactive Protein and Triglycerides (p<0.001). LDL-C also showed a statistically significant positive correlation with High-Sensitive C–reactive Protein (p<0.03). HDL-C also showed a statistically not significant negative correlation with High-Sensitive C–reactive Protein (p<0.32). **Conclusion:** The patients with dyslipidemia for elevated blood hsCRP levels may be done to identify those patients with an increased risk stratification of atherosclerosis. Also this suggests that there may be a role for anti -inflammatory agents along with statins in treatment of dyslipidemia.

Keywords: High-Sensitive C–reactive Protein (hsCRP), Lipid Profile and dyslipidemia.

INTRODUCTION

Dyslipidemia and subclinical inflammation are the major determinants of cardiovascular disorders. Cardiovascular diseases, the leading causes of death in the world are rising rapidly in low- and middle-income countries.[1] Disorder in lipid metabolism is one of the main determinants of cardiovascular risk. 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.[2,3] Increased levels of low-density lipoprotein cholesterol (LDL-C), triglycerides (TGs), total cholesterol (TC) and decreased levels of high density lipoprotein cholesterol (HDL-C) are associated with atherosclerosis. The primary target of lipid management is to achieve lowering of low - density lipoprotein cholesterol (LDL-C).[4] LDL-C is known to activate a cascade of local inflammation which can lead to formation of atherosclerotic plaques, ultimately leading to cardiovascular disease or acute coronary syndrome (ACS). Even though both hsCRP and Lipid Profile parameters have a role in initiation and progression of atherosclerosis, no data is currently available regarding the correlation between these two entities. Our aim was to fine out the correlation between High-Sensitive C–reactive Protein with Lipid Profile Parameter.

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MATERIALS AND METHODS

This present study was conducted in the department of Clinical Biochemistry, RKDF Medical College Hospital and Research Center, Bhopal in collaboration with the department of Medicine during the period from January 2017 to February 2018. Subjects (45) were selected purposively from the out-patient department of RKDF Medical College according to inclusion-exclusion criteria. Blood samples were obtained from the antecubital vein with the subject sitting comfortably in a chair in a quiet room and transfused into vacuum tubes containing EDTA in the morning after an overnight fasting period. After separation, blood samples were centrifuged for 10 minutes at 3000 rpm to obtain

serum. Then serum was aliquoted into two microtubes, one preserved for lipid profile measurements and another was preserved at -20oC for hsCRP estimation. Following biochemical parameters to be studied.[6]

1. Total Cholesterol (TC) by enzymatic end point CHOD-POD methods.
2. Triglyceride (TG) by enzymatic glycerol phosphate oxidase/peroxidase methods.
3. HDL-Cholesterol by direct enzymatic end point method.
4. LDL-Cholesterol by Friedewald's formula. LDL-c = Tc-HDL-c(TG/5)
5. The High-Sensitive C–reactive Protein levels were analyzed by sandwich ELISA technique using hsCRP kit.

Sample size calculation was done according to standard methods available. Statistical analysis was done using SPSS18 software.

RESULTS & DISCUSSION

This present study was conducted in the department of Clinical Biochemistry, RKDF Medical College Hospital and Research Center, Bhopal in collaboration with the department of Medicine. [Table 1] shows the strong and statistically significant positive correlation in between High-Sensitive C–reactive Protein (hsCRP) and Total Cholesterol with ($p<0.016$). Statistically significant positive correlation between High-Sensitive C–reactive Protein and Triglycerides ($p<0.001$). LDL-C also showed a statistically significant positive correlation with High-Sensitive C–reactive Protein ($p<0.03$). HDL-C also showed a statistically not significant negative correlation with High-Sensitive C–reactive Protein ($p<0.32$).

Table 1: Shows the Correlation between hsCRP with the lipid profile parameters in dyslipidemic patients

Variables	Correlation coefficient (r)	p-value
TC	0.28	0.016
TG	0.56	0.001
LDL-C	0.24	0.03
HDL-C	-0.14	0.32*

[*Statistically significant ($p<0.05$); Note:TC(Total Cholesterol), TG(Triglyceride), LDL-c (low density lipoprotein cholesterol), HDL-c(high density lipoprotein cholesterol)]

Dyslipidemia is a prominent one among the traditional biochemical risk factors of CVDs. Elevated TG, total cholesterol, and LDL cholesterol as well as decreased HDL cholesterol has been implicated with a variably increased risk of CVDs both in cross-sectional and prospective studies.[7,2] The nature and extent of dyslipidemia, however, may vary depending on the ethnic, cultural and environmental background of a particular population. CRP is an acute phase protein which is generated shortly after an inflammatory stimulus from the liver cells. Several cytokines like IL-1, IL-6 and TNF-α that are secreted locally in the area of the damaged tissue regulate the production of CRP.[10] Cardiovascular diseases, metabolic syndrome, Type-2 diabetes mellitus and obesity are associated with low grade of systemic inflammation and in these conditions, as inflammation is subclinical or low grade, hence CRP level does not increase at a greater amount as seen in severe systemic infections rather its increment is small so that highly sensitive method is needed to estimate that small amount of CRP in blood, there for hsCRP estimation have been emerged in the field of medical sciences. This in part suggests that the associations of CRP concentrations with fasting insulin, fasting glucose, and HOMA-IR could be due to the presence of a chronic systemic sub-clinical inflammation. Disease like Dilated cardiomyopathy (DCM) is associated with increased inflammatory response reflected among other markers in high sensitivity C-reactive protein (hsCRP) and soluble interleukin-2 receptor (sIL-2R) levels. There was a significant correlation between sIL-2R and hsCRP levels in dyslipidemic patients but not in normo-lipidemic patients Therefore estimation of IL-1, IL-6 and TNF-α along with HOMA-IR and sIL-2R was also essential for identification of other cardiovascular disease in immune-mediated inflammatory diseases. Above findings support the hypothesis that dyslipidemia can induce an inflammatory reaction at blood vessels which is a hall mark feature for development of atherosclerosis. Low grade inflammation is a novel risk factor in all stages of atherosclerosis and acute coronary syndrome. This present study has shown levels of LDL-C, triglycerides and total cholesterol are associated with development and progression of atherosclerosis.[11] The transport vehicle of cholesterol and other lipids in body is low density lipoprotein cholesterol (LDL-C). Once oxidized, LDL-C is called small dense LDL 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. Moreover, the storage site of triglycerides is mainly adipose tissue which was earlier considered to be a passive organ is now known to express the pro-inflammatory cytokines like IL-6. Excess loading of triglycerides in adipose tissue as seen in obesity can cause release of IL-6 by adipose tissue which can be involved in induction of low grade systemic inflammation as well as inflammation at blood vessels.[12] High serum level of high density lipoprotein cholesterol (HDL-C) on the other hand is associated with reduced risk for development of atherosclerotic disease as it is involved in reverse cholesterol transport. HDL-C particle are therefore believed to be anti-atherogenic and antagonized pathways of inflammation,

thrombosis and oxidation of LDL-C.[13] Serum amyloid A (SAA) is transported predominantly on HDL and levels of this protein increase markedly during acute and chronic inflammation in both animals and humans. Increased SAA levels predict the risk of cardiovascular disease in humans. There are evidences, showing that secretory phospholipase A2, an HDL-associated protein, and platelet-activating factor acetylhydrolase, a protein associated predominantly with LDL in humans and HDL in mice, might also play roles both as markers and mediators of human atherosclerosis. In contrast to positive acute-phase proteins, negative acute-phase proteins have received less attention. The level of Apo lipoprotein A-I (apoA-I), the major apolipoprotein of HDL, decreases during inflammation. Recent studies also indicate that HDL is oxidized by myeloperoxidase in patients with established atherosclerosis. These alterations may limit the ability of apoA-I to participate in reverse cholesterol transport. Paraoxonase-1 (PON1), another HDL-associated protein, also decreases during inflammation. PON1 is atheroprotective in animal models of hypercholesterolemia. Controversy over its utility as a marker of human atherosclerosis may reflect the fact that enzyme activity rather than blood level (or genotype) is the major determinant of cardiovascular risk. Thus, multiple lipoprotein-associated proteins that change in concentration during acute and chronic inflammation may serve as markers of cardiovascular disease. High serum level of high density lipoprotein (HDL) on the other hand is associated with reduced risk for development of atherosclerotic disease. HDL particle are believed to be anti atherogenic and antagonized pathways of inflammation, thrombosis and oxidation.[14]

CONCLUSION

These findings suggest that the patients with dyslipidemia for elevated blood hsCRP levels may be done to identify those patients with an increased risk stratification of atherosclerosis. Also this suggests that there may be a role for anti - inflammatory agents along with statins in treatment of dyslipidemia.

REFERENCES

1. Miranda JJ, Kinra S, Casas JP, Davey Smith G, Ebrahim S (2008) Noncommunicable diseases in low- and middle-income countries: context, determinants and health policy. Trop Med Int Health 13: 1225-1234.
2. Van Leeuwen M, Van Rijswijk M. Acute phase proteins in the monitoring of inflammatory disorders. Baillieres Clin Rheumatol.1994;8(3):531–52.
3. 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.
4. National Cholesterol Education Program. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA 2001;285:2486-2497.
5. Carreiro-Lewandowski E (2004) Update on selected markers used in risk assessment for vascular disease. Clin Lab Sci 17: 43-49.
6. MK Yadav, TK Mohapatra, RK Mohapatra, et al. Study on Glycated Hemoglobin & lipid profile in Type-2 Diabetes Mellitus. International Journal of Science & Research (IJSR) , June 2015; 4(6):1917-19.
7. Sacks FM, Pfeffer MA, Moye LA, Rouleau JL, Rutherford JD, et al. (1996) The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. Cholesterol and Recurrent Events Trial investigators. N Engl J Med 335: 1001-1009.
8. Verschuren WM, Jacobs DR, Bloemberg BP, Kromhout D, Menotti A, et al. (1995) Serum total cholesterol and long-term coronary heart disease mortality in different cultures. Twenty-five-year follow-up of the seven countries study. JAMA 274: 131-136.
9. Brewer HB Jr (2002) Rising to the challenge of the new NCEP ATP III guidelines: exceeding current therapeutic limitations. Am J Manag Care 8: S23-28.
10. Kitsios K, Papadopoulou M, Kosta K, Kadoglou N, Papagianni M, Tsiroukidou K. High-sensitivity C-reactive protein levels and metabolic disorders in obese and overweight children and adolescents. J Clin Res Pediatr Endocrinol 2013;5:44-49.
11. Burchardt P, Żurawski J, Zuchowski B, Kubacki T, Murawa D, Wiktorowicz K, 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;9:151-158.
12. Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Elevated C-reactive protein levels in overweight and obese adults. Jama. 1999;282:2131-2135.
13. Khan HA, Alhomida AS, Sobki SH. Lipid profile of patients with acute myocardial infarction and its correlation with systemic inflammation. Biomarker insights 2013;8:1-7.
14. Haseeb A. Khan, Abdullah S. Alhomida, Et Al. " Lipid Profile Of Patients With Acute Myocardial Infarction And Its Correlation With Systemic Inflammation" Biomarkerinsights 2013:8 1–7

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