RESEARCH ARTICLE

A STUDY OF Hs-CRP AND LIPID PROFILE IN OVERWEIGHT INDIVIDUALS

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ABSTRACT

Background: Obesity and overweight are widespread phenomena. Dyslipidaemia observed in obese people is an important risk factor for coronary heart disease. Levels of obesity have been shown to be associated with low-grade inflammation and C-reactive protein, an inflammatory marker has been associated with the presence and severity of atherosclerosis.

Aims & Objective: To evaluate the Hs-CRP levels in overweight individuals with dyslipidaemia and without dyslipidaemia, and thus assess its role in detecting cardiovascular disease (CVD) in overweight individuals.

Material and Methods: 120 overweight subjects were included in the study, they were divided into 2 groups- Group I which included subjects with dyslipidaemia and Group II included normolipemic subjects. BMI, Hs-CRP, Total Cholesterol, HDL-C, Triacylglycerol, LDL-C, VLDL-C, T-C/HDL-C, LDL-C/HDL-C, Glucose were estimated in both the groups and the data was statistically analyzed.

Results: BMI, Hs-CRP, Total Cholesterol, Triacylglycerol, LDL-C, VLDL-C, T-C/HDL-C, LDL-C/HDL-C, Glucose were significantly higher in Group I compared to group II ($p<0.05$), whereas HDL-C was significantly lower in group I than group II ($p<0.05$).

Conclusion: Hs-CRP is elevated in overweight individuals with dyslipidaemia, thus it is recommended to detect risk of CVD in them.

KEY-WORDS: BMI; Hs-CRP; Lipid Profile; Overweight

Introduction

Overweight is generally defined as having more body fat than is optimally healthy. The degree to which a person is overweight is generally described by body mass index (BMI). In India 12.6% of women and 9.3% of men are obese.[1] Being overweight increases the likelihood of developing type 2 diabetes and cardiovascular disease (CVD). Being overweight parse is not the problem rather it may be the metabolic abnormalities that often coexist with overweight and obesity.[2] It is now scientifically well established that inflammation plays a role in the initiation, growth and destabilization of atherosclerotic plaques that lead to clogged arteries and most heart attacks.[3] Lipids and lipoproteins are well known risk factors for ischemic heart disease. Elevated levels of triglyceride, cholesterol and LDL-C are documented as risk factors for atherogenesis.[4] There is growing recognition that coronary heart disease (CHD) has an inflammatory component. Prospective studies have shown that plasma C-reactive protein (CRP) concentration, a marker of the acute-phase reaction, can predict CHD events in subjects with or without established cardiovascular disease beyond what can be estimated by traditional risk factors.

Materials and Methods

120 healthy overweight (BMI>25) individuals were selected for the study. These overweight individuals were divided into two distinct groups: Group I (n=60) included people with high triglyceride and low HDL-C (dyslipidaemic group), whereas the Group II (n=60) included normolipemic people. Inclusion Criteria: Males and Females 20 – 50 yr, BMI >25. Exclusion Criteria: Acute and chronic inflammatory diseases, Diabetes, H/o smoking, H/o alcoholism, H/o

angina, myocardial infarction, H/o recent illness, H/o taking anti-inflammatory drugs or statins. Informed consent was obtained from all 120 subjects; their weight (in kg) and height (in meters) was taken. BMI was calculated by the formula BMI = Weight/Ht^2. Participants were in the 12 hr of fasting state. 3 ml venous blood was collected in 5 mg% EDTA collection bottle for plasma (for lipid profile and glucose), 3 ml venous blood was collected in plain bottle and allowed to clot to separate serum (for CRP). Plasma and Serum were separated within one hour after sample collection. Care was taken to avoid haemolysis. The following methodology was applied to the samples to obtain the required biomarker levels; HS-CRP- Automation-Immunoturbidimetry method; Total Cholesterol-Spectrophotometer- Cholesterol Oxidase enzymatic method; HDL-C- Spectrophotometer-Cholesterol Oxidase enzymatic method; Triacylglycerol- Spectrophotometer- GPO-PAP enzymatic method; LDL-C-by Friedwalds equation; VLDL-C-TG/5; T-C/HDL-C ratio; LDL-C/HDL-C ratio; GLUCOSE-GOD-POD method. Data obtained was analyzed by SPSS statistical software (v 15.0)

Results

Table-1: Comparison of Group I and Group II

Parameter	Group I	Group II	P value
BMI	27.80 ± 1.05	27.08 ± 1.01	0.000
Hs-CRP	2.86 ± 1.31	2.08 ± 0.92	0.000
T-C	200.70 ± 32.80	173.91 ± 28.09	0.000
HDL-C	36.88 ± 5.09	56.60 ± 10.57	0.000
TAG	216.68 ± 50.47	88.93 ± 25.81	0.000
VLDL	43.33 ± 10.09	17.79 ± 5.19	0.000
LDL	122.28 ± 29.13	99.50 ± 29.66	0.000
T-C/HDL-C	5.54 ± 1.18	3.18 ± 0.83	< 0.0001
LDL/HDL-C	3.38 ± 0.93	1.85 ± 0.75	< 0.0001
Glucose	98.93 ± 12.89	90.26 ± 10.47	0.000

P < 0.05, significant

Unpaired T test was used for comparison between the two groups. The results were expressed as Mean ± SD and p value. The mean BMI was higher in group I than group II. Plasma Hs-CRP levels were significantly higher in group I than the group II ($p<0.05$). Group I individuals had significantly higher levels of T-C, TAG, VLDL, LDL,T-C/HDL-C ratio, LDL-C/HDL-C ratio and Glucose when compared to group II($p<0.05$). The HDL-C levels were significantly lower in group I when compared to group II.

Discussion

In the present study the dyslipidaemic group had significantly higher fasting levels of serum Hs-CRP than the normolipemic group. The normolipemic group had the mean Hs-CRP of 2.08, which is higher than the normal reference range (<1mg/l). This is because as they are all overweight and adipose tissue is a source for the production and release of cytokines, which will stimulate the synthesis of CRP by the liver.[5] Dyslipidaemic individuals have significantly higher glucose levels than the normolipemic and are more at risk of getting diabetes. The cause might be due to insulin resistance. Similar results (high levels of Hs-CRP and Glucose) were observed by Philip barter et al.[6] In a recent statement issued jointly by the American Heart Association and the Centers for Disease Control, the Hs-CRP test was recommended for use in the identification of risk for CVD.[7] As the presence of inflammation can precede a heart attack or stroke by eight or more years[8], Hs-CRP testing can allow for effective preventative therapies[7].

In the present study out of 120 overweight individuals 60 were dyslipidaemic and 60 were normolipemic. Although overweight (BMI>25) individuals tend to be insulin resistant, hyperinsulinemic, glucose intolerant, and dyslipidaemic, not all overweight or obese individuals are insulin resistant, nor do they all have the characteristic disturbances in glucose or lipid metabolism.[9-12] Unknown genetic factors could be the cause for this difference. The dyslipidaemic group had significantly higher levels of Total Cholesterol, TAG, VLDL, LDL, T-C/HDL-C, LDL-C/HDL than the normolipemic individuals. However the HDL-C levels were significantly low in dyslipidaemic individuals than the normolipemic individuals. Plasma triglyceride and high-density lipoprotein (HDL) cholesterol levels are independently associated with insulin resistance[13] and are independent predictors of CVD[14]. In addition, the plasma concentration ratio of total cholesterol to HDL cholesterol is well recognized as a predictor of CVD [15] and is also highly correlated with insulin resistance[16]. The cause for dyslipidaemia in overweight individuals could be primary insulin resistance which might be due to genetic factors.

Mechanisms underlying the observed association of dyslipidaemia with the other metabolic derangements are uncertain. Dyslipidaemia has been shown to be an important risk factor for CAD[17], since Triacylglycerol brings changes in LDL particle size, density, distribution and composition producing small dense LDL which is more atherogenic. Considerable evidence has established the presence of oxidized components of LDL in atherosclerotic lesions. Other cytokines regulated by components of oxidized LDL can activate the leukocytes within the intimal layer, provoking their production of further inflammatory mediators. Moreover, these activated leukocytes can generate reactive oxygen species that augment oxidant stress, the constant companion of inflammation in atherosclerosis.[18] These inflammatory mediators(TNF-α,IL-6) will stimulate the synthesis of CRP by the liver.[19] Thus Hs-CRP levels are elevated in this study. Estimation of serum triglyceride levels is thus an indirect measurement of LDL particle size.

A growing number of studies suggest that CRP is an independent risk factor for atherosclerotic vascular disease. It has been suggested that Hs-CRP may not only be a marker of generalized inflammation but directly and actively participate in atherogenesis.[20] CRP binds to the LDL particle in atherosclerotic plaques leading to activation of complement and tissue factor production by macrophages[21], thus being pro-inflammatory and contributing to atherogenesis.

Limitations: The present study is a small scale study so large scale studies are indicated to further establish the findings.

Conclusion

Hs-CRP is a simple cost effective test, which can predict the cardiovascular risk. Hence it is recommended in overweight individuals.

References

1. Bhalwar R. Text Book of Public Health and Community Medicine. 1st ed. Pune: Dept of Community Medicine, AFMC. 2009. P. 1196.
2. Bray GA. Medical consequences of obesity. J Clin Endocrinol Metab 2004;89:2583–2589
3. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. Nature 1993;362:801–809.
4. Lipid Research Clinic Program. The lipid research clinic coronary primary prevention trial results II. J Am Med Assoc 1984;251:364-74.
5. Mohamed-Ali V, Pinkney JH, Coppack SW. Adipose tissue as an endocrine and paracrine organ. Int J Obes Relat Metab Disord. 1998;22:1145–1158.
6. Barter P, McPherson YR, Song K, Kesäniemi A, Mahley R, Waeber G, et al. Serum insulin and inflammatory markers in overweight individuals with or without dyslipidemia. J Clin Endocrinol Metab 2007;92:2041–2045.
7. Pearson TA, Mensag GA, Alexander RW, Anderson JL, Cannon RO 3rd, Criqui M et al. Markers of Inflammation and Cardiovascular Disease: Application to Clinical and Public Health Practice. A Statement for Healthcare Professionals from the Centers for Disease Control and the American Heart Association. Circulation. 2003;107:499-511.
8. American Heart Association. Inflammation, Heart Disease and Stroke: The Role of C-Reactive Protein. Dallas, Texas: American Heart Association; 2001.
9. Jones CN, Abbasi F, Carantoni M, Polonsky KS, Reaven GM. Roles of insulin resistance and obesity in regulation of plasma insulin concentrations. Am J Physiol Endocrinol Metab. 2000;278:E501-8.
10. McLaughlin T, Abbasi F, Carantoni M, Schaaf P, Reaven G. Differences in insulin resistance do not predict weight loss in response to hypocaloric diets in healthy obese women. J Clin Endocrinol Metab. 1999;84:578-81.
11. Zavaroni I, Bonini L, Fantuzzi M, Dall'Aglio E, Passeri M, Reaven GM. Hyperinsulinaemia, obesity, and syndrome X. J Intern Med.1994;235:51-6.
12. Reaven G, Segal K, Hauptman J, Boldrin M, Lucas C. Effect of orlistat-assisted weight loss in decreasing coronary heart disease risk in patients with syndrome X. Am J Cardiol. 2001;87:827-31.
13. Laws A, Reaven GM. Evidence for an independent relationship between insulin resistance and fasting plasma HDL-cholesterol, triglyceride and insulin concentrations. J Intern Med. 1992;231:25-30.
14. Hokanson JE, Austin MA. Plasma triglyceride level is a risk factor for cardiovascular disease independent of high-density lipoprotein cholesterol level: a meta-analysis of population-based prospective studies. J Cardiovasc Risk. 1996;3:213-9.
15. Kinosian B, Glick H, Garland G. Cholesterol and coronary heart disease: predicting risks by levels and ratios. Ann Intern Med. 1994;121:641-7.
16. Jeppesen J, Facchini FS, Reaven GM. Individuals with high total cholesterol/HDL cholesterol ratios are insulin resistant. J Intern Med. 1998;243:293-8.
17. Han TS, Williams K, Sattar N, Hunt KJ, Lean ME, Haffner SM. Analysis of obesity and hyperinsulinemia in the development of metabolic syndrome: San Antonio Heart Study. Obes Res 2002;10:923–931.
18. Libby P, Ridker PM, Hansson GK. Inflammation in Atherosclerosis: From Pathophysiology to Practice

J Am Coll Cardiol. 2009 December 1; 54(23): 2129–2138.
19. Maachi M, Piéroni L, Bruckert E, Jardel C, Fellahi S, Hainque B, et al. Systemic low grade inflammation is related to both circulating and adipose tissue TNFa, leptin and IL-6 levels in obese women. Int J Obes 2004; 28: 993
20. Espliguero RA, Avanzas P, Sales JC, Aldama G, Pizzi C, Kaski JC. C-reactive protein elevation and disease activity in patients with coronary artery disease. Eur Heart J 2004;25:401-8.
21. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART STUDY). Lancet 2004;364:937-52.

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