ORIGINAL ARTICLE

A Study Showing Association between Hscrp and Fasting Plasma Glucose in Overweight and Obese Patients in a Tertiary Care Centre in Gujarat, India

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ABSTRACT

BACKGROUND: Obesity is rapidly growing health problem in both developed and developing countries. In adults obesity is associated with hyperinsulinemia, insulin resistance, dyslipidemia, and vascular dysfunction. C-Reactive Protein (CRP), formerly considered solely as a biomarker for inflammation, is now viewed as a prominent partaker in endothelial dysfunction and atherosclerosis **AIMS & OBJECTIVES:** The present study aims to establish correlation between elevated hscrp levels and elevated fasting plasma glucose levels in overweight and obese patients. **MATERIAL & METHODS:** The present study was undertaken at a tertiary care centre in Gujarat, India for a period of 2 years. Inclusion criteria includes healthy males and females more than 18 years with body mass index ≥ 25kg/m^2 whereas all the comorbidities like diabetes mellitus, hypertension, stroke, coronary artery disease are excluded. **RESULTS:** There was statistically significant positive correlation between elevated Fasting Blood Sugar(FBS) level and HSCRP level (elevated FBS : normal FBS ratio of HSCRP was 3.26 : 1.85, Z value 3.35, p value <0.0012). **CONCLUSION:** Thus there is a direct correlation with increased hscrp and fasting plasma glucose which in itself a risk factor for atherosclerotic cardiovascular diseases and metabolic syndrome.

Key words: HSCRP, Obesity, Body Mass Index (BMI)

INTRODUCTION

Obesity is rapidly growing health problem in both developed and developing countries. In adults obesity is associated with hyperinsulinemia, insulin resistance, dyslipidemia, and vascular dysfunction. Recent evidence indicates that obesity may represent a low-grade chronic inflammatory state as reflected by the elevation in a number of inflammatory markers in serum, such as interleukin 6 (IL-6), tumor necrosis factor-α (TNF-α), soluble tumor necrosis factor receptor II (sTNF-RII), and C-reactive protein (CRP)[1]

C-Reactive Protein (CRP), formerly considered solely as a biomarker for inflammation, is now viewed as a prominent partaker in endothelial dysfunction and atherosclerosis[2,3]. Serving clinically for several years as a nonspecific Marker for inflammatory processes, CRP, with the advent of high-sensitivity assays, has emerged as one of the most powerful independent predictors of cardiovascular diseases[2,3]

In this study we studied correlation between elevated hscrp levels and elevated fasting plasma glucose levels in overweight and obese patients.

MATERIALS AND METHODS

A trial of 50 asymptomatic subjects having BMI ≥ 25 in the age group more than 18 years visiting to medicine department were included in the study. All these subjects were clinically evaluated and appropriate investigations were carried out in these subjects.

At the time of enrollment, each volunteer was given health history questionnaire mentioned in the Performa. Anthropometric measurements were done. Height and weight were measured by standard procedures. Body-Mass Index was calculated by following formula

BMI= Weight in Kilograms / Height in Meter^2.

Based on health history questionnaire, patients were classified into life style

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groups sedentary and non-sedentary. Fasting blood samples were collected in all the subjects and serum glucose measured. Plasma concentrations of HSCRP were measured by a highly sensitive nephelometric assay using a monoclonal antibody to HSCRP coated on polystyrene beads. Blood samples for the assay were obtained using the standard venipuncture technique into standard collection tubes. This was followed by statistical analysis done by Epi Info version7.2 software.

Inclusion Criteria: Healthy males and females more than 18 years with body mass index $\geq 25 kg/m^2$ were included in the study.

Exclusion Criteria:

- Subjects with ischemic heart disease.
- Subjects with Hypertension.
- Subjects with diabetes mellitus.
- Subjects with history of strokes.
- Subjects with history of arthritis.
- Subjects with ongoing fever.
- Pregnant female patients.
- Subjects with liver function test abnormalities.
- Subjects receiving any form of therapies, including aspirin, cyclooxygenase-2 inhibitors and statins.
- Subjects with HSCRP more than 10 mg/l.
- Subjects with renal failure.
- Subjects having history of carcinoma colon, carcinoma breasts, renal cell carcinoma, and adenocarcinoma of esophagus.

RESULTS

HSCRP levels (mg/L) were calculated in these subjects. According to HSCRP value patients were stratified in different risk groups.

- 10 subjects had HSCRP < 1 mg/L – Low risk.
- 12 subjects had HSCRP between 1 to 2 mg/L – Moderate risk.
- 16 patients had HSCRP between 2 to 3 mg/L – High risk.
- 12 patients had HSCRP > 3 mg/L – Very high risk.

In study group 10 subjects (20% of total 50 subjects) had HSCRP < 1, 12 subjects (24% of total 50 subjects) had HSCRP 1-2, 16 subjects (32% of total 50 subjects) had HSCRP 2-3 and 12 subjects (24% of total 50 subjects) had HSCRP > 3. In subjects with HSCRP < 1, 50% were overweight (BMI 25-29.9) and 50% were moderately obese (BMI 30-39.9). In subjects with HSCRP 1-2, only 1 subject was overweight (BMI 25-29.9) rest all were moderately obese (BMI 30.-39.9). In subjects with HSCRP 2-3 all (100%) subjects had BMI ≥ 30 which was also true for subjects with HSCRP > 3. After statistical analysis body mass index was linearly related with HSCRP value 9 p value 0.000053).[table-1]

Graph 1 and table 2 shows that 12 subjects (24% of total 50 subjects) had elevated fasting blood sugar levels and in these patients, mean HSCRP was 3.26 while 38 patients (76% of total 50 subjects) had normal fasting blood sugar level and in these patients mean HSCRP was 1.85, which shows that there is statistically significant correlation between HSCRP and FBS level (elevated FBS : normal FBS ratio of HSCRP was 3.26 : 1.85, z value 3.35, p value < 0.0012)

DISCUSSION & CONCLUSION

In present study we found that subjects with impaired fasting glucose had significantly higher values of HSCRP. So there is positive correlation between impaired FBS and HSCRP (elevated FBS : normal FBS ratio of HSCRP was 3.26 : 1.85, z value 3.35, p value < 0.0012).Obesity is a component of metabolic syndrome so subjects with impaired fasting glucose may represent metabolic syndrome. These subjects have high risk of complications as demonstrated by higher HSCRP values in these subjects. The finding of a significant relationship between HSCRP and obesity in adults who are just beginning to show evidence of the insulin resistance syndrome raises the possibilities that with additional maturation, the HSCRP/Insulin resistance association will become evident in these at-risk individuals. Insulin resistance and HSCRP levels are strongly correlated in adults[4], indicating underlying low grade inflammation.

Relationship of BMI and HSCRP in

present study was consistent with other studies focusing same issue.

John S et al[5] in 1999 showed that concentrations of CRP were related to insulin resistance as calculated from the homeostasis model assessment model, blood pressure, HDL, and triglyceride, and to markers of endothelial dysfunction (plasma levels of von Will brand factor, tissue plasminogen activator, and cellular fibronectin). There was a close correlation of mean standard deviation score for the levels of acute phase markers and score of insulin resistance syndrome variables (r=0.59, P<0.00005), this relationship being weakened only marginally by removing measures of obesity from the insulin resistance score (r=0.53, P<0.00005).

Antoinette Moran et al[6] in 2005 showed that there was no difference in mean CRP levels among boys (n=189, CRP 1.10@0.46 mg/l) and girls (n=153, CRP 1.16@0.63 mg/l ; P=0.320. There was also no difference between CRP and Tanners stage. CRP adjusted for BMI was significantly greater in black subjects compared with white subjects (P= 0.03). There was a strong correlation of CRP and adiposity in both sexes. CRP levels were related to fasting insulin levels (r=0.16, P=0.003).

Thus there is a direct correlation with increased hscrp and fasting plasma glucose which in itself a risk factor for atherosclerotic cardiovascular diseases and metabolic syndrome. Whether hscrp is directly involved in pathogenesis of metabolic syndrome and cardiovascular disease or merely a metabolic marker is not known.

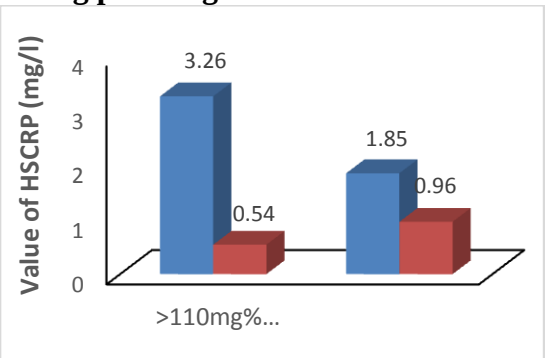
Table 1: HSCRP level of the case studied.

HSCRP	No.	Percent
< 1mg/l	10	20
1-2 mg/l	12	24
2-3 mg/l	16	32
> 3 mg/l	12	24
Total	50	100

Table 2: comparison of HSCRP and FBS (MG%) level.

FBS (mg%)	Mean HSCRP(mg/L)	SD(mg/L)
> 110 mg%	3.26	0.54
≤ 110 mg%	1.85	0.96
Total	2.19	1.08
Z test applied	Z value is 3.35	
	P value is <0.05	
	Difference is significant	

Graph1: Comparison of HSCRP and fasting plasma glucose



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