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*Full Length Research Paper*

# High-sensitivity C-reactive protein a surrogate marker of insulin resistance

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**High-sensitivity C-reactive protein (hsCRP) is predictor of cardiovascular disease (heart attack, stroke) in apparently healthy individuals (Pfützner et al., 2004). Very few studies have specifically evaluated the usefulness of hsCRP as a marker of insulin resistance in normoglycemic young adults. The aim of this study was to assess whether hsCRP can be used to detect insulin resistance in normoglycemic healthy young adults. 80 normal volunteers were evaluated for insulin resistance. A standard oral glucose tolerance test (OGTT) was used, plasma glucose and insulin levels assessed at 0(fasting), 30, 120 min. Insulin resistance was calculated using mathematical models like HOMA IR, QUICKI, insulinio genic index (ISI), ISI 0-120. hsCRP was assessed from fasting sample by turbidimetric method. Subjects hsCRP (mg/L) values <1 were grouped as Group A (low risk) and >1 Group B (intermediate and high risk) subjects. Subjects with hsCRP >1 in Group B had a significantly higher body weight, waist circumference, body mass index (BMI) and waist hip ratio. OGTT parameters and the insulin resistance and sensitivity parameters in Group B subjects were significantly higher in subjects with hsCRP>1. ISI 0-120 which takes into account the second hour OGTT values show significantly lower insulin sensitivity with hsCRP >1. hsCRP significantly increased with increasing BMI, waist circumference, waist-hip ratio (WHR) & insulin resistance (IR) indices. It was observed that as insulin resistance increases, hsCRP also increases. The finding of this study point to the significant role a simple blood test like hsCRP can play in the early detection of insulin resistance in normal healthy subjects.**

**Key words:** hsCRP, IR, C-reactive protein, insulin resistance, insulin sensitivity, HOMA IR, ISI 0-120, normoglycemic, oral glucose tolerance test (OGTT), type-2 diabetes (T2DM), diabetes.

## INTRODUCTION

C-reactive protein (CRP) is an acute-phase protein which rises in response to inflammation, it activates the complement system via the C1Q complex (Thompson et al., 1999; Pepys and Hirschfield, 2003;). CRP is an exquisitely sensitive systemic marker of inflammation and tissue damage. In the mid 1990s, immunoassays for C-reactive protein (hs-CRP), with greater sensitivity than those previously in routine use, revealed that increased CRP values, even within the range previously considered normal, strongly predict future coronary events. These findings triggered widespread interest, where the clinical use of CRP measurement had been largely ignored (Tillett and Francis Jr., 1930; Clyne and Olshaker, 1999; Lloyd-Jones et al., 2006). Earlier work suggested a prognostic association between increased hsCRP production and outcome after acute myocardial infarction (de Beer, 1982) and in acute coronary syndromes (Berk et al., 1990).

Data from the IRIS-II study also showed that hsCRP may also be a good marker of macrovascular risk in type 2 diabetic patients. In the state of pathologically increased demand on the beta cells, intact proinsulin appears in the plasma along with insulin and C-peptide due to the inability of enzymes to cleave excess proinsulin

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(Pfützner et al., 2004). Elevated levels of plasma high-sensitivity C-reactive protein (hsCRP) are associated with insulin resistance/hyperinsulinemia and cardiovascular autonomic dysfunction in type 2 diabetic patients without insulin treatment (Anana et al., 2005; Pepys and Hirschfield, 2003). Diabetes mellitus (DM) counts as a coronary heart disease (CHD) risk equivalent.

Insulin resistance exists when a normal concentration of insulin produces a less than normal biological response. As our understanding of the mechanisms underlying the genesis of glucose intolerance has grown, the role of insulin resistance (IR) has emerged as a critical one (Wallace and Matthews, 2002). IR is a central feature in the natural history of impaired glucose tolerance (IGT), type-2 diabetes (T2DM) and may be a factor in the etiology of hypertension, dyslipidiemia and risk of atherosclerotic heart diseases (Gutt et al., 2000). Hence, recognition of IR in the pre-disease state would be beneficial, as it affords potential implementation of interventions designed to reduce such disease development (McAuley et al., 2001). The clinical profile of average Indian T2DM patient is different from that of a white Caucasian. At diagnosis, Indian patients are a decade younger; despite their thinness, Indians are more insulin resistant and hyperinsulinemic. Family history of diabetes is more common in Indians. The high susceptibility of Indians to T2DM is largely unexplained. No specific genes have yet been identified but 'thrifty genotype' has been a useful teleological concept (Yajnik, 2004). The lifestyle of an urban Indian is fast becoming 'Western' and widespread mechanization has reduced physical activity (Yajnik et al., 1995).

## Rationale for the study

High-sensitivity C-reactive protein (hsCRP) was shown in large epidemiological studies to predict the risk of cardiovascular disease in apparently healthy individuals (Pfützner et al., 2004). Very few studies have specifically evaluated the usefulness of highly sensitive C- reactive protein as a marker of insulin resistance in normo-glycemic young adults (McAuley et al., 2001; Matsuda and De Fronzo, 1999; Hanley et al., 2003).

## Objective of the study

The objective was to assess whether hsCRP can be used as a biomarker to detect insulin resistance in normo-glycemic healthy young adults.

## MATERIALS AND METHODS

80 normal young adult volunteers in the age range of 18 to 25 years were evaluated for insulin resistance. Volunteers, who satisfied the inclusion and exclusion criteria, were educated regarding the study; an informed consent was obtained as per ICH GCP good clinical practice guidelines. Subjects with infections, inflammatory diseases, tissue injury and corticosteroids medications were excluded from the study. There was no financial liability on the study subjects.

All subjects underwent a detailed general physical examination including blood Pressure, body weight, height, hip and waist circumference. All measures were done while subjects wore light clothes without shoes and BMI, waist hip ratio calculated.

A standard oral glucose tolerance test (OGTT) was performed on all subjects and blood sample was analyzed for hsCRP. The normal subjects were grouped as Group A: If hs-CRP level is lower than 1.0 mg/L (a person has a low risk of developing cardiovascular disease) and Group B: If hs-CRP is above 1.0 (up to 3.0 mg/L, a person has an average risk and if hs-CRP is higher than 3.0 mg/L, a person is at high risk).

A standard (75 g) OGTT was performed on all the study subjects. After an overnight fast, the study subjects ingested the OGTT solution within 2 min. Blood samples for determination of plasma glucose and insulin levels were drawn using disposable scalp vein set at 0 (fasting), 30, and 120 min after solution ingestion.

### Assays

Fasting (basal), 30, and 120 min venous plasma glucose during OGTT was determined by glucose oxidase method on site using glucose auto analyzer. Concentrations of total cholesterol, triglyceride and HDL cholesterol were determined by enzymatic kinetic method using an auto analyzer. VLDL cholesterol level was calculated using the formula Triglyceride/5 and LDL using the formula [Total Cholesterol – (HDL + VLDL)].

The serum plasma was stored at -20°C until assayed. Corresponding specific insulin concentration was determined by radioimmuno assay (RIA) using a human specific antibody RIA kit, which does not cross-react with human proinsulin. This immuno-assay uses the principle where there is competition between a radioactive and non-radioactive antigen for a fixed number of antibody site. Insulin in protein based buffer with sodiumazide as preservative, I-125 labeled insulin, were used as reagents in this procedure. Reagents were pippetted into the test tube and incubated at 2 to 8°C for 16 h. Then the precipitating reagent was added and the tubes were incubated at room temperature for 10 to 15 min. The tubes were centrifuged for 20 min at 1500-x g. The tubes were then decanted and washed to remove any traces of unbound reagents. All the tubes were counted in the Gamma counter for 1 min. A standard curve was plotted for the bound / total percent (B/T %) against insulin concentration on semi log paper. The insulin concentration was estimated from the standard curve. Highly sensitive C- reactive protein (hs CRP) was assessed from fasting sample by turbidimetric method.

### Measurement of insulin resistance

In this study, we use mathematical models to measure IR. Commonly used physiological models like homeostatic model assessment (HOMA) (Matthews et al., 1985; Wallace et al., 2004), QUICKI (quantative insulin sensitivity check index) (Katz et al., 2000), $ISI_{0-120}$ (insulin sensitivity index) (Gutt et al., 2000), insulinogenic index (Kim et al., 2001), etc., were used to obtain indices of hepatic and whole body insulin resistance / sensitivity from measurement of plasma glucose and insulin concentrations obtained during oral glucose tolerance test (Stumvoll et al., 2000) in non-diabetic young adults to detect early occurrence of insulin resistance and evaluate the predictive value of hsCRP in detection of insulin resistance.

Homeostatic model assessment HOMA-IR = (FPI x FPG)/22.5 and HOMA-%B = (20 x FPI)/(FPG 3.5), where FPI is fasting plasma

insulin concentration (mU/l) and FPG is fasting plasma glucose (mmol/l). Quantative insulin sensitivity check index QUICKIE = 1/[log (FPI) +log (FPG)] = 1/[log (FPI x FPG)], where FPI is the fasting plasma insulin (micro units per ml) and FPG is fasting plasma glucose level (milligram per dL). Insulin sensitivity index ISI $_{0\text{-}120}$ : m = (7500mg + (o min glucose 120 min glucose) x 0.19 x BW) / 120 min, and MCR = m / MPG and ISI 0-120 = MCR / log MSI = m / MPG / log MSI {Mean plasma glucose (MPG); the mean of the 0 and 120 min glucose values from OGTT is used the obtain the metabolic clearance rate (MCR) which corrects for the potential influence of different blood glucose concentration on glucose uptake rate. To correct the skewness of distribution, the mean serum insulin (MSI, mIU/l) calculated as the mean of the 0 and 120 min insulin values, is logarithmically transformed}. Insulinogenic index (Berk et al., 1990) IGI = [30 min insulin level fasting insulin level (IU/ml)] / [30 min plasma glucose fasting plasma glucose (mg/dl)].The data was systematically collected in the case record form designed for the study. Various parameter of insulin resistance and sensitivity were calculated using mathematical models and their formulas.

**Statistical analysis**

The student t test and Mann Whitney U test have been carried out to find the significant difference between various OGTT parameters and indices between subjects in Group A and B. Chi-square and Fisher Exact test have been used to find the significant difference of frequencies between with and without family history of diabetes. Statistical diagnostic values, such as sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) have been computed at various cut –off values of indices obtained from literatures. The odds ratio has been computed to find relationship of indices for the various cut-off. The Pearson correlation coefficient between IR indices and clinical and lab parameters have been computed. The statistical software SPSS 10.0 and Systat 8.0 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables, etc.

## RESULTS

In this prospective study of 80 normoglycemic young adult subjects in south Indian urban agglomerate, the mean age of the study population was 19 years (18 to 25) years (SD+1.69). The sex distribution was male: 41.3% (33) and female: 58.8% (47). 50% of the subjects were siblings of diabetics. All the study subjects, Clinical and lab parameters were within normal limits. The mean hSCRP of the study population was 1.69 (0.1 -13.9) mg/L. Subjects with hsCRP (mg/L) values <1 were grouped a Group A (low risk) and >1 Group B (intermediate and high risk) subjects. Comparison of clinical parameters (Table 1) revealed that subjects in group B had a significant higher body weight, waist circumference, body mass index (BMI) and waist-hip ratio (WHP). The lipid profiles were not significantly different from the two groups.Comparison of OGTT parameters and the insulin resistance and sensitivity parameters (Table 2) reveal significantly and 120 min that group B (hsCRP >1) have higher 30 glucose and insulin values (Figure 1) and insulin sensitivity index ISI $_{0\text{-}120}$ which take into account the second hour OGTT values (Figure 2) show significantly lower insulin sensitivity index. hsCRP was observed to strongly correlate with the clinical markers of Insulin resistance like BMI (Figure 3), waist circumference, WH ratio (Figure 4).

Subjects were stratified by hsCRP at various cutoff values ranging from < 0.8, 0.9 to 3.5 and >3.5. hs CRP significantly increased with increasing BMI, waist circumference and WHR (,Table 3). Since the prevalent values of IR values derived from indices like HOMA, QUICKI, ISI, etc., are not known for our population, the cutoff values used are mean values from literature of normoglycemic, impaired glucose tolerant and diabetic subjects of other populations. HOMA_IR value = 1 is normal, HOMA_IR value = 2 is seen in the 5$^{th}$ and 6$^{th}$ decade subjects with normal OGTT, HOMA_IR value = 3 is seen in elderly subjects with impaired glucose tolerance (de Beer, 1982). As the HSCRP increases HOMA IR increases insignificantly (trend observed) and QUICKI and ISI $_{0\text{-}120}$ decreases. Clear trend is observed for ISI $_{0\text{-}120}$ as Insulin Sensitivity decreases (Figure 5) with the increasing hsCRP with p=0.218 (Table 4). hsCRP was observed to strongly correlate with the clinical markers of insulin resistance like BMI, waist circumference, WH ratio. hsCRP as a marker of IR in normoglycemics has a sensitivity of 48 to 51% and specificity of 66 to 69%, and positive predictive value of 74 to 86% (Table 5).Siblings of diabetics had a hsCRP of 1.417 (SD+ 2.094) whereas, siblings of non diabetics had a hsCRP of -0.9175 (SD+ 2.136) (t -3.841 p-0.000).

## DISCUSSION

The maintenance of normal glucose tolerance in healthy subjects is achieved mainly by three important factors, that is, insulin secretion, tissue glucose uptake: peripheral (primarily muscle) and splanchnic (liver and gut) and suppression of hepatic glucose production (Wallace and Matthews, 2002). It is hypothesized that the development of T2DM is a two-step process. The first step is transition from normal to impaired glucose tolerance, for which insulin resistance is the main determinant, and the second and later step is worsening from impaired glucose tolerance to T2DM, in which beta cell dysfunction plays a critical role (Saad et al., 1991). The increment for the prevalence of T2DM and IGT warrants lowering the cutoffs of normoglycemia to help predict the future development of diabetes (Rheea, 2006). Subclinical inflammation and insulin resistance, forerunners to CHD and T2DM, may be pathophysiologically interlinked (Edward et al., 2003). High CRP concentrations signifycantly correlate with insulin resistance and the metabolic syndrome in adults (Ridker, 2003; Vikram, 2006). C-reactive protein (CRP) is one of the best studied markers for systemic subclinical inflammation, and may have prognostic value in predicting the future risk of cardiovascular events (Haverkate et al., 1997). In cross- sectional studies, highly sensitive CRP has been found to correlate with increased triglyceride, decreased HDL, increased

**Table 1.** Comparison of clinical parameters.

| Variable | hsCRP (mg/L) | | Significance |
|---|---|---|---|
| | Group A hSCRP < 1.0 (n = 51) | Group B hsCRP > 1.0 (n = 28) | |
| Age (Years) | 18.96±1.74 | 19.07±1.65 | t=0.273; P=0.784 |
| Male (%) | 21 (41.2) | 11 (39.3) | 0.870 |
| Female (%) | 30 (58.8) | 17 (60.7) | |
| Siblings of diabetics (%) | 24 (47.1) | 16 (57.1) | 0.391 |
| Height (cm) | 163.91±9.72 | 162.14±8.70 | t=0.802; p=0.425 |
| Weight (kg) | 57.51±10.74 | 63.13±14.04 | t=1.989; p=0.050* |
| BMI (kg/m$^2$) | 21.38±3.08 | 24.09±5.11 | t=2.936; p=0.004** |
| Waist (cm) | 73.10±8.53 | 78.66±13.2 | t=2.272; p=0.026* |
| HIP (cm) | 91.50±7.35 | 94.66±10.97 | t=1.528; p=0.131 |
| WHR waist hip ratio | 0.80±0.06 | 0.83±0.08 | t=1.944; p=0.056+ |
| SBP (mm Hg) | 119.73±6.03 | 119.57±6.72 | t=0.104; p=0.917 |
| DBP (mm Hg) | 74.24±5.29 | 74.21±4.91 | t=0.017; p=0.986 |
| **Lipid profile (mg/dl)** | | | |
| Total cholesterol | 157.31±29.44 | 156.25±20.63 | t=0.170; p=0.866 |
| Triglycerides | 93.61±24.68 | 100.75±33.69 | t=1.078; p=0.284 |
| HDL | 40.59±4.44 | 40.61±5.34 | t=0.017; p=0.987 |
| VLDL | 18.71±4.93 | 19.50±5.46 | t=0.659; p=0.512 |
| LDL | 98.03±29.85 | 96.32±20.73 | t=0.269; p=0.789 |
| Total cholesterol | 157.31±29.44 | 156.25±20.63 | t=0.170; p=0.866 |

Categorical variables are presented in No. (%) and p values are obtained by Chi-square test/Fisher exact test; results on continuous variables are presented in mean ± SD, student t-test is used to find the significance. BMI = body mass index

**Table 2.** Comparison of OGCT, glucose, insulin and insulin resistance index.

| Variable | HsCRP | | Significance |
|---|---|---|---|
| | Group A <1.0 (n=51) | Group B >1.0 (n=28) | |
| **OGTT value** | | | |
| Glucose | | | |
| 0 min | 80.92±10.26 | 83.61±9.14 | t=1.155; p=0.252 |
| 30 min | 107.22±19.84 | 118.39±22.77 | t=2.272; p=0.026* |
| 120 min | 87.49±14.97 | 96.68±19.55 | t=2.337; p=0.022* |
| **Serum insulin** | | | |
| 0 min | 8.23±6.98 | 8.08±5.06 | t=0.098; p=0.922 |
| 30 min | 69.77±65.57 | 66.75±45.29 | t=0.217; p=0.829 |
| 120 min | 31.17±27.08 | 49.58±39.25 | t=2.455; p=0.016* |
| **Insulin resistance value** | | | |
| HOMA_IR | 1.65±1.37 | 1.71±1.19 | t=0.183; p=0.855 |
| HOMA%B | 243.70±319.31 | 173.1±152.95 | t=1.100; p=0.275 |
| Insu_gen_Index | -0.44±20.18 | 2.88±5.05 | t=0.855; p=0.395 |
| QUICKI | 0.32±0.08 | 0.3±0.06 | t=0.970; p=0.335 |
| ISI(0-120) | 68.81±24.44 | 54.73±16.13 | t=2.735; p=0.008** |
| RATIO_IO_VS_GO | 0.1±0.09 | 0.1±0.05 | t=0.385; p=0.701 |

**Table 3.** Association of hsCRP with anthropometry parameters.

| hsCRP level | BMI (kg/m$^2$) (Mean ± SD) | Waist circumference (cm) (Mean ± SD) | Waist-Hip ratio (Mean ± SD) |
|---|---|---|---|
| <0.8 | 21.50±2.85 | 72.88±7.70 | 0.79±0.05 |
| 0.8-3.5 | 22.13±4.63 | 73.24±10.98 | 0.79±0.06 |
| >3.5 | 25.93±5.18 | 85.95±12.35 | 0.86±0.08 |
| Significance by ANOVA | P=0.004** | P<0.001** | P<0.001** |

As the HSCRP increases the Anthropometry parameters increases significantly (P<0.001).

**Table 4.** Association of HSCRP with IR indices parameters.

| HSCRP levels | HOMA (Mean ± SD) | QUICKI Mean ± SD) | ISI $_{0-120}$ (Mean ± SD) |
|---|---|---|---|
| <0.8 | 1.66±1.43 | 0.32±0.08 | 67.06±23.77 |
| 0.8-3.5 | 1.73±1.07 | 0.30±0.06 | 61.66±22.19 |
| >3.5 | 1.72±1.36 | 0.29±0.05 | 54.27±17.44 |
| Significance by ANOVA | P>0.05 | P=0.481 | P=0.218 |

As the HSCRP increases HOMA IR increases insignificantly (trend observed) and QUICKI and ISI $_{0-120}$ decreases. Clear trend is observed for ISI $_{0-120}$ as it decreases with the increasing HSCRP with p=0.218.

**Table 5.** Diagnostic relationship of IR Indices with hsCRP

| Diagnostic parameters (HSCRP >0.8 ) | HOMA (>1.0) | QUICKI (<0.33) | ISI $_{0-120}$ (<80.0) |
|---|---|---|---|
| Sensitivity (%) | 50.94 | 50.94 | 48.49 |
| Specificity (%) | 66.67 | 66.67 | 68.75 |
| Positive predictive value (%) | 75.00 | 75.00 | 86.11 |
| Negative predictive value (%) | 40.91 | 40.91 | 25.00 |
| Accuracy (%) | 56.25 | 56.25 | 52.50 |
| Odds ratio | 1:2.08 | 1:2.08 | 1:2.06 |

blood pressure and increased fasting plasma glucose concentrations, suggesting its association with increased prevalence metabolic syndrome associated with IR (Mendall et al., 1997; Pasceri et al., 2000). Few studies have established the association of CRP with IR independent of obesity (Taniguchi et al., 2002). In a recent study, CRP was found to significantly associate with several surrogate measures of IR like fasting insulin, the Raynaud index, the quantitative insulin sensitivity check index, and the McAuley index, HOMA, QUICKI, the insulin: glucose ratio and the Avignon index in non- diabetics (Meng et al., of 2007). Because the simplicity of measurement, stability, and improved high-sensitivity method, CRP may be useful as a clinical measure for identifying individuals at risk for IR (Ridker et al., 2004). The aim of this study was to find out whether high- sensitivity C-reactive protein (hsCRP) has any relevance in early detection of insulin resistance even in normoglycemic young adults. "High sensitivity" CRP assays is a simple and inexpensive test, it has been endorsed by both the Centers for Disease Control and Prevention and by the American Heart Association as a part of the routine global risk assessment to better determine risk of heart disease and prevent clinical events. Levels of CRP less than 1, 1 to 3, and greater than 3 mg/L discriminate between individuals with low, moderate, and high risk of future heart attack and stroke (Ridker, 2003). Despite its lack of specificity, CRP has now emerged as one of the most powerful predictors of cardiovascular risk. Even more remarkable, CRP's predictive power resides in the range between 1 to 5 µg/mL, which was previously regarded as normal in the era preceding the high-sensitivity CRP test. Hence a high sensitive assay is required (Edward et al., 2003). CRP levels were shown to be associated with fasting insulin levels in healthy 2 to 3 years old children (Shea et al., 2003), and young (18 to 22 years) Japanese (Kazumi et al., 2003).

The pathophysiological association of hsCRP and insulin resistance assumes great importance in Indian adolescents and young adults in whom substantial

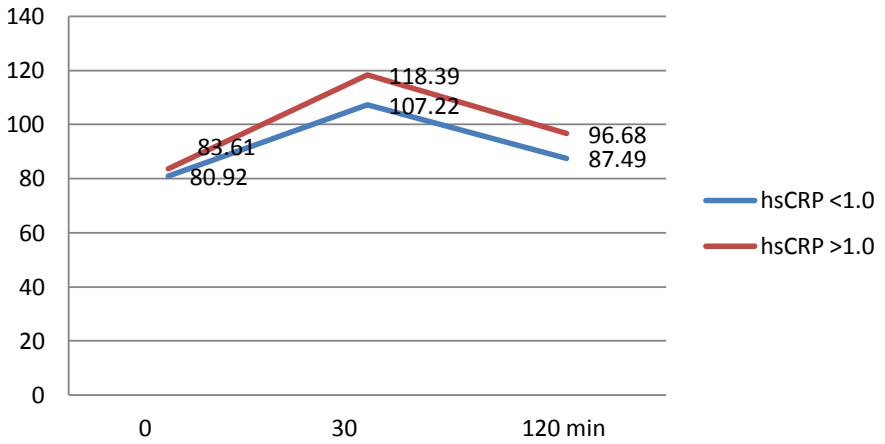


**Figure 1.** Serum glucose time trend during OGTT comparatively significantly higher glucose values in subjects with hsCRP >1 at 30 min t=2.272; p=0.026*, at 120 min t=2.337; p=0.022*.

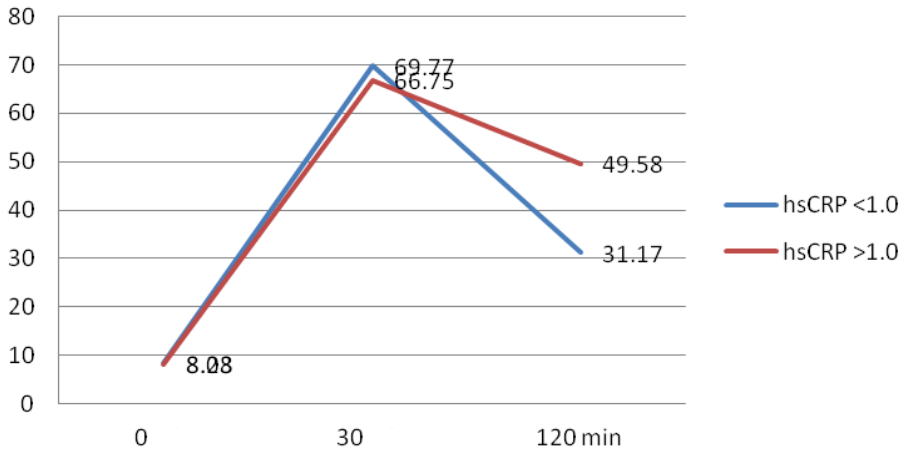


**Figure 2.** Insulin response to glucose load during 2 h OGTT shows a significantly higher sustained serum insulin in the late phase at 120 min (t=2.455; p=0.016*).

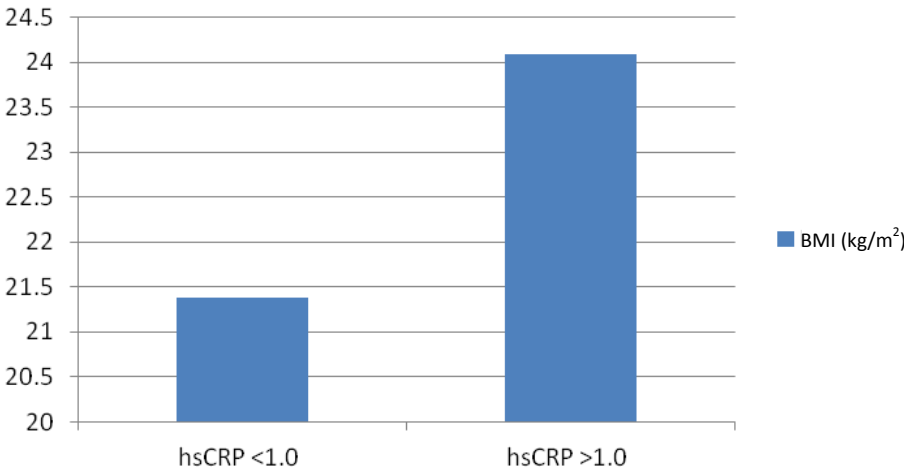


**Figure 3.** Subjects with hsCRP >1 had significantly higher BMI (t=2.936; p=0.004**).

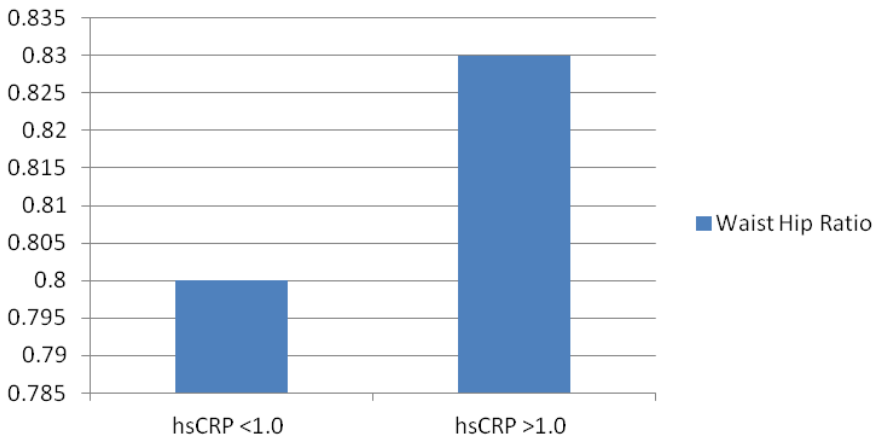


**Figure 4.** Subjects with hsCRP >1 had significantly higher Waist Hip ratio (t=1.944; p=0.056+).

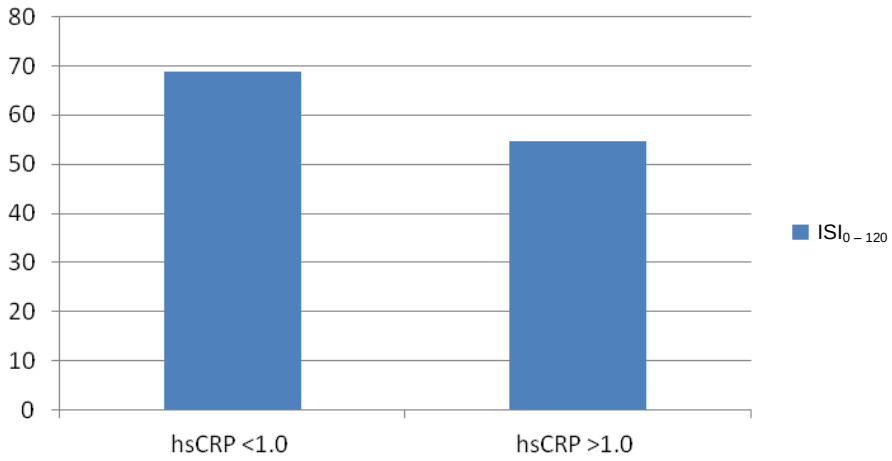


**Figure 5.** Even in normoglycemics, subjects with hsCRP>1 had significantly lower insulin sensitivity index ($ISI_{0-120}$) (t=2.735; p=0.008**).

prevalence of hyperinsulinaemia (Misra et al., 2004) and elevated CRP levels 1 have been demonstrated by other groups. Elevated levels of C-reactive protein (CRP) are also known to be associated with insulin resistance and metabolic syndrome in adults (Forouhi et al., 2001). In our study, higher hs-CRP (>1 mg/L) levels correlated significantly with increasing BMI, waist circumference, WH ratio. 30 min glucose and 120 min insulin also correlated with hs- CRP. An important observation of this study is the presence of good correlation between hs-CRP and surrogate markers of insulin resistance especially $ISI_{0-120}$ which take into account the 0 to 120 min insulin and glucose values in efficiently interpreting the IR indices, unlike the other indices based on fasting values. C-reactive protein has been shown to be associated with type 2 diabetes, but whether CRP has a causal role is not yet clear. A meta-analysis of Rotterdam study to evaluate the association of baseline serum CRP and incident diabetes during follow-up was investigated. The risk of diabetes was significantly higher in the haplotype with the highest serum CRP level compared with the most common haplotype (OR 1.45, 95% CI, 1.08 to 1.96). These findings support the hypothesis that serum CRP may also play a role in the development of diabetes (Dehghan, 2007).

## Conclusion

In this study, significant increase in hsCRP was seen with increasing BMI and waist circumference which are clinical markers of insulin resistance. It was also observed that even in normoglycemic healthy subjects, as insulin resistance increases, hsCRP also increases. This finding of

this study points to a significant role hsCRP plays in the early detection of insulin resistance. Further population based studies are indicated to establish the role of hs-CRP as a predictor of insulin resistance.

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