Original Research Article

Role of type of diet and life style on chronic inflammatory marker

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Abstract

Background: Subclinical chronic inflammation plays a key role in the development of atherosclerosis, a key factor in the initiation of CVDs. Biomarkers of inflammation are therefore considered predictors of future CVD events. CRP is an acute-phase protein and is one of the most sensitive and most recurrent markers of inflammation. Many studies have shown that an increase in the concentration of HSCRP is associated with an increased risk of CVDs in different populations around the world. **Material and Methods:** Total 50 subjects of both sexes who were healthy but having BMI > 25, were recruited for this study. At the time of enrollment, each volunteer was given health history Questionnaire. Based on that we classified the subjects in two groups according to their type of diet (Vegetarian vs. Non vegetarian) and life style (sedentary vs. Non sedentary). Blood samples collected for fasting HSCRP values and then appropriate statistical test applied for interpretation of results. **Observations and Results:** Statistical analysis did not show any statistically significant correlation of type of diet with HSCRP value (non-vegetarian: vegetarian diet ratio of HSCRP was 2.21: 2.14, z value 0.77, P value > 0.1251) while, there was statistically significant correlation found for lifestyle with HSCRP value (sedentary: non sedentary lifestyle ratio of HSCRP was 2. 43: 1.80, z value 21, P < 0.05). **Key Word:** HSCRP, Life Style, Vegetarian Diet, Chronic Inflammation.

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INTRODUCTION

In recent years, an increasing incidence of chronic non communicable diseases (NCDs) related to diet, obesity, cardiovascular diseases (CVDs), including hypertension and diabetes mellitus type 2 (DM2), are becoming a 21st-centuryepidemic. A total of 57 million deaths occurred in the world during 2008; 36 million (63%) were a result of NCDs. The leading causes of NCD deaths in 2008 were CVDs (48% of NCD deaths). WHO projections show NCDs will be responsible for a substantially increased total number of deaths in the next decade¹. India is among

the countries in which the high prevalence of chronic NCDs is observed. Data in the literature show a relation between inflammation and chronic diseases, such as CVDs, DM2 (2), and metabolic syndrome³. Inflammation is a necessary response of the immune system to acute infection or trauma; however, a prolonged inflammatory state has detrimental health effects (4).Subclinical chronic inflammation plays a key role in the development of atherosclerosis, a key factor in the initiation of CVDs (5, 6).Increased expression and activation of C-reactive protein (CRP) and proinflammatory cytokines such as IL-6, is associated with atherogenesis and CVDs. Biomarkers of inflammation are therefore considered predictors of future CVD events^{7,8}. CRP is an acute-phase protein and is one of the most sensitive and most recurrent markers of inflammation. CRP is synthesized and excreted primarily by hepatocytes in response to proinflammatory cytokines. CRP is present in the serum of healthy individuals in small quantities, and its concentration increases rapidly over the course of inflammation or necrotic processes9. However, it was noted that slightly elevated concentrations, measured with the use of HSCRP, indicate that inflammation is under

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way in the vascular wall⁹. Many studies have shown that an increase in the concentration of HSCRP is associated with an increased risk of CVDs in different populations around the world. Elevated concentrations of CRP reflect endothelial dysfunction through the deterioration of its anti-inflammatory activity ¹⁰.CRP is correlated with the components of metabolic syndrome: atherogenic dyslipidemia, obesity, and insulin resistance¹¹ Adipose tissue plays an important role in the body's inflammatory processes⁵.Obesity, therefore, contributes to the formation of a proinflammatory environment¹². There is also a close relation between CRP and body fat⁵. However, the main determinant of serum HSCRP concentration is not only the amount of body fat but also its distribution. It was shown that adipocyte abdominal fat may have a higher concentration of IL-6¹³ and higher CRP secretion (14) than subcutaneous depots. CRP concentrations are higher in obese patients, especially patients with excess visceral fat, than in patients of normalweight¹⁵. The aim of our study is to correlate the type of diet (vegetarian vs. Non vegetarian) and life style (sedentary vs. Non sedentary) with the concentration of inflammatory marker -HSCRP.

MATERIALS AND METHODS:

We recruited healthy but over weight or obese (BMI \geq 25) subjects of both sex of the age group more than 18 years based on Asia-Pacific criterio16 visiting to medicine department OPD. At the time of enrollment, each volunteer was given health history Questionnaire. 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 Meter2 Based on Questionnaire, Persons who had at least two non vegetarian meals in a week for last 6 months were labeled as Nonvegetarian and who don't were labeled as vegetarian. Persons who were doing any form of aerobic or anaerobic exercises for at least 6 hours in a week since last 6 months were classified as physically active/Non sedentary life style while others who don't were classified as physically inactive/sedentary life style. Subjects were excluded if they had CVD, stroke, type 2 diabetes mellitus, hypertension (or taking any antihypertensive medications), if they were taking aspirin or any other anti-inflammatory medications, or were women on hormone replacement therapy. Fasting blood samples were collected in all the subjects. Blood samples for the assay were obtained using the standard venipuncture technique into standard collection tubes. Plasma concentrations of HSCRP were measured by a highly sensitive nephelometric assay using a monoclonal antibody to HSCRP coated on polystyrene beads. The assay consists of a suspension of polystyrene particles

coated with marine monoclonal antibodies to HSCRP. The concentration of suspended particles is optimal for agglutination measurement by nephelometry. When reagents are mixed with samples containing HSCRP, the intensity of the scattered light in the nephelometer depends on the HSCRP content of the sample. Therefore, the HSCRP concentration can be determined by comparison with dilutions of a standard of known concentration. The lower sensitivity of the HSCRP assay was 0.2 mg/liter. Statistical analysis done by various methods to determine correlation between HSCRP level and various demographic and biochemical parameters. While applying z test, p value is calculated from z value by following data (p=0.48 at z value 0, p=0.1251 at z value 1, p=0.05 at z value 2, p=0.0012 at z value 3).Due to lack of data about equivalent z and p values above z value of 3, it is presumed that p value is <0.0012 if z value is above 3.

OBSERVATION AND RESULTS

In all the Fifty (50) patients HSCRP was measured. Depending on HSCRP levels patients were divided into four risk groups.

- 10subjectshadHSCRP<1mg/L–Low risk.
- 12subjectshadHSCRPbetween1to2mg/L-Moderate risk.
- 16patientshadHSCRPbetween2to3mg/L-High risk.
- 12patientshadHSCRP>3mg/L–Veryhigh risk. Table1: HSCRP level of the case studied

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

Correlation was tested between HSCRP levelsand life styleandtype of diet and other data using statistical methods. Subjects were selected after using above mentioned inclusion and exclusion criteria. In our study 40 subjects (80% of total 50 subjects) were non vegetarian and 10 subjects (20% of total 50 subjects) were vegetarian.

| Table 2: Comparison of HSCRP with type of diet | | | |
|--|-------------------------------|------|--|
| Type of Diet | Highly sensitive CRP (Mg/L) | | |
| | Mean | SD | |
| Non-veg. | 2.21 | 1.05 | |
| Veg. | 2.14 | 1.14 | |
| Total | 2.19 | 1.04 | |
| | Z value is 0.77 P > 0.05 | | |
| Z test applied | | | |
| | Difference is not significant | | |

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Graph 1: Comparison of HSCRP with type of Diet

Table 2 and Graph 1 shows that mean HSCRP in vegetarian subjects is 2.14 and in non-vegetarian subjects is 2.21.Sstatistical analysis did not show any statistically significant difference in HSCRP value in both groups (non-vegetarian: vegetarian diet ratio of HSCRP was 2.21: 2.14, z value 0.77, P value > 0.1251) which shows that there is no effect of diet on HSCRP value. In our study group 31 subjects were having sedentary life style. Out of which 14 are females (70% of total 20 females) and 17 are males (56.66% of total 30 males). In another group of 19 subjects of non-sedentary life style 5 are females (25% of total 20 females) and 14 are males (46.66% of total 30 males).

| Table 3: Lifestyle among the cases | | | | |
|--|---------------------------|------------|--|--|
| Life style | No. | Percentage | | |
| Sedentary | 31 | 62 | | |
| Non Sedentary | 19 | 38 | | |
| Total | 50 | 100 | | |
| | | | | |
| Table 4: Comparision of HSCRP by Lifestyle | | | | |
| Lifestyle | HSCRP mg/I | | | |
| _ | Mean | SD | | |
| Sedentary | 2.43 | 1.03 | | |
| Non Sedentary | 1.80 | 1.00 | | |
| Total | 2.19 | 1.06 | | |
| Z test applied | Z value is 21 | | | |
| | P < 0.05 | | | |
| | Difference is Significant | | | |



Table 4 and Graph 2shows that mean HSCRP in sedentary life style group is 2.43 and in non-sedentary life style group is 1.80. After statistical analysis, there is statistically significant differencefound between both groups(sedentary :non sedentary lifestyle ratio of HSCRP was 2.43 : 1.80, z value 21, P < 0.05). This finding shows that level of HSCRP is more in person, having sedentary life style. Thus patients with sedentary life style may have more risk of future complications.

DISCUSSION

In the present study we tried to find out relationship between type of food and lifestyle with one of the marker of chronic inflammation that is HSCRP. In this study, there was no statistically significant difference of type of diet on HSCRP was observed (Non Vegetarian: Vegetarian diet ratio was 2.21: 2.14, z Value was 0.77, and P Value was > 0.1251). However, some studies suggest effect of diet on biomarkers of inflammation. Nettleton et al.¹⁷ showed that a model based on the consumption of wholegrains, fruits, nuts, and green leafy vegetables was significantly inversely correlated with concentrations of CRP and IL-6 in a multi-ethnic population. The Mediterranean diet is the best studied model of nutrition in the context of many diseases, including all components of the metabolic syndrome. Traditionally, the Mediterranean diet is characterized by high consumption of olive oil, vegetables, fruit, vegetable protein, whole grains, legumes, nuts, fish, Low Fat dairy products, low to moderate consumption of poultry, low intake of red meat, and moderate alcohol consumption. Olive oil is the main source of fat in this diet model¹⁸. Many observational, prospective, and randomized studies confirm the inverse relation between the Mediterranean diet and inflammatory markers¹⁹. The Nurses' Health Study (20) which analyzed several diet quality indexes, showing that use of the Mediterranean diet, as measured by the alternative Mediterranean Diet index, was associated with significantly lower concentrations of CRP and IL-6 (24% and 16%, respectively). Sharman et al^{21} compared weight loss with very Low Carbohydrates diets (<10% of energy) with low fat (<30% of energy) on inflammatory markers in men with excess body weight. The investigators found that both diets resulted in a significant reduction in body weight, HSCRP, and other determined inflammatory markers. These data suggest that in the short term, weight loss is a major force underlying the reduction of inflammatory markers. Though it is difficult to determine which diet model is optimal for reducing inflammation in humans, evidence is strong and consistent for beneficial effect of the Mediterranean diet on IL-6 and CRP concentrations. Long term vegetarians have better antioxidants status and

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coronary artery disease risk profile than do apparently healthy omnivores. However available evidence are indirect which requires further testing in both epidemiological and clinical investigative studies Many studies have also demonstrated the anti-inflammatory effect of the plant diet model but here in our study we could not find Statistically significant difference between vegetarian and Non vegetarian diet. In this study, lifestyle had positive correlation with BMI and HSCRP (sedentary : Non sedentary life style life style ratio was 2.43: 1.80, z value was 21, p was < 0.05). Subjects with sedentary life style had higher BMI and HSCRP values indicating that subjects that are physically inactive are more prone to develop future atherosclerosis and other coronary events. Role of Physical activity on reduction in atherosclerosis process is well established. Higher value of HSCRP in peoples having sedentary lifestyle is also found in many cross sectional and horizontal studies of which one that conducted by Okita $et al^{22}$ reported that supervised aerobic exercise significantly decreased HSCRP levels by 35%. Mattusch *et al*²³ also reported that CRP median fell from 1.19 to 0.82 mg/L after 9 months of endurance training (P < .05). Ryan and Nicklas²⁴ also found that 6 months of diet and exercise in overweight and obese postmenopausal women decreases HSCRP levels. You etal²⁵ found that diet plus exercise, but not diet alone, decreased plasma HSCRP by 34% in obese postmenopausal women. Being physically active was inversely associated with HSCRP levels, which get confirmed in the results of studies with younger²⁶ and obese women populations²⁷. The mechanisms involved with the beneficial effect of exercise seem to be related to the cytokines produced by muscle cells, which could exert a protecting role against chronic diseases associated with low-grade inflammation, including improvement on endothelial function and nitric oxide synthesis²⁸.

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