Original Article

C Reactive Protein

C Reactive Protein Concentrations can Predict Metabolic Syndrome in Adults

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ABSTRACT

Objective: This study analysedthe correlations between C reactive protein (CRP) serum concentrations and demographics and anthropometric aspects of developing metabolic syndrome components in adults.

Study Design: A cross-sectional study.

Place and Duration of study: This study was carried out in Azad Kashmir Sheik Khalifa Bin Zyad Hospital Muzaffarabad from March 2012 to April 2013.

Materials and Methods: Demographic, anthropometric parameters of MS such as body mass index (BMI) and waist hips ratio (WHR), biochemical and clinical data were collected from 115 adults of age ranged between 22-55yearsold.

Results: Adults BMI had direct correlation with CRP concentrations. In our study CRP concentrations were statistical significant correlated with age (r = 0.282, p = <0.002),BMI (r = 0.787, p = <0.001), waist hips ratio (r = 0.850, p =< 0.001) and weight (r = 0.662, p =<0.001). The height had poor correlation with CRP (r = 0.101, p = 0.825).Thecorrected CRP (r = 0.101, p =0.825) was also poorly correlated to CRP concentrations.

Conclusion: Statistical analysis has shown there is direct correlation between BMI, WHR and CRP concentrations which suggests that inflammation might be an important event in the development of metabolic disorders in adults.

Key words: Adults, Risk factors, C reactive protein, Metabolic syndrome, Obesity.

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INTRODUCTION

Overweight and obesity in adults are major public health hazardsworldwide. Obesity is defined as an excess of body fat. The body fat is clinically quantified by calculations of body mass index (BMI).It is calculated by dividing measured body weight in kilograms by height in meters squared.[1] Increase in nutritional status has shown significant increase of overweight. Overweight is defined as BMI 25-29.9 whereas obesity as 35-39.9.Distribution of body fat deposit aroundwaist and flank is greater hazardous than fat in buttocks and thigh. Obese patients with waist >102cm in men and 88cm in female or with waist –hip ratios >1.0 in men and >0.085in women have greater risk of diabetes mellitus, coronary artery disease.This abdominal obesity is associated with insulin resistance henceglucose intoleranceand poor fatty acid metabolism resulting in type 2 diabetes mellitus. Insulin resistance in tissue with associated Hyperinsulinemia and hyperglycemia, and adipokines lead to vascular endothelial dysfunction in addition to disturbances of lipid profile and hypertension.These changes promote the development of atherosclerotic coronary heart disease.Patientswith abdominal obesity with higher waist hipratios without excess of total body weight do develop coronary heart disease. The existence of theserisk factors likeabdominal obesity, hyperglycemia, Hyperinsulinemia, dyslipidemia, and hypertension have been named as metabolic syndrome.In obesity, excess of body fat deposits as visceral and abdominal fat and hence contribute to increase WHR. Fatty acids impair glucose mediated glucose uptake. Disturbances of lipid and glucose metabolism can cause hypertrigly ceridemia with accumulation in cardiac and skeletal muscles. The increased blood level of FFA and glucose enhance secretion of pancreatic insulin resultingin Hyperinsulinemia. Hyperinsulinemia inturnincreases sodium reabsorption, sympathetic nerve stimulation and promotes atherosclerosishence contributes to hypertension. FFA flux to liver is alsoassociated with increase triglyceride abundance of very low density lipoprotein hence resulting in hypertrigly ceridemia. High correlation between obesity and MS has been demonstrated in overweight/obese children, adolescents and adults[2-8]. The chronic mild inflammation in hepatocyte and adipose tissue is associated with higherconcentrations of proinflammatory cytokines such C reactive protein, interleukin (IL)-1IL-6 and tissue necrosis factor. These cytokines elevation have been observed in response to infection, trauma and malignancies .In obese patients CRP elevation has been observed in coronary vascular disease, diabetes mellitus and metabolic Syndrome.[9-14]

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The study was designed to investigate correlation between metabolic syndromecomponents and CRP concentrations in adults.

MATERIALS AND METHODS

Clinical, demographics, anthropometrics and biochemical data were obtained and entered in data processing. This study was approved by the Ethics Committee. Patients with pregnancy, breast feeding mothers, paraplegics and malignancies were excluded. Weight, height and waist hip ratios (WHR) were measured. Body Mass Index (BMI) was estimated by dividing weight in kilograms (kg) by height in square meters(m^2). Patients were categorized by BMI as under weight, overweight and obese according to standardized criteria.Metabolic syndrome components were assessed by body configurations i.e. BMI and abdominal adiposity by WHR .[15-17]

Data was analysed by using theStatistical Package for Social Science (SPSS) version 20.The level of significance was set at p<0.05.Chi square,Spearman correlation, Univariate analysis and PLUM-Ordinal Regression analysis were used to show association/ correlation of demographic anthropometrics component of MS and CRP concentrations.

RESULTS

A total of 115, 85 (73.9%) males and30 (26.1%) females were enrolled for study. The mean age of the adults was 29.29±7.71). Table no 1 and 2 shows the mean values for the MS components of the patients in association with CRP. BMI was directly correlated with CRP, WHR and weight in adults. In our study CRP concentration was directly correlated with age (r = 0.282, p = <0.002),BMI (r = 0.787, p = <0.001), waist hips ratio (r = 0.850, p =< 0.001) and weight (r = 0.662, p =<0.001).The height had poor correlation with CRP (r = 0.101, p = 0.825 1) and corrected CRP (r = 0.101,p =0.825) was also poorly correlated to CRP concentrations.

Table No.1; Correlation(r) between C-reactive protein concentration and demographics, anthropometrics Components of MS

Clinical variable	Total	r	P-Value
N (%)	**115(%)**		
Age (mean ± SD)	29.3 ± 7.7	.282	.002
Male	85(73.9%)		.001
Female	30(26.1%)		.323
BMI(kg/m^2)	18(12.9.6%)	.778	<0.001
Waist hip ratio (WHR)	8(5.7)	.850	<0.001
Weight		.662	<0.001
Height	21(15)	.088	.347
Corrected CRP	39(27.9)	.101	.825

*P value for Spearman correlation between CRP concentration and component of MS

Table No.2 showed Chi square and Univariate analysis of CRP concentrations and demographic anthropometric parameters among the adults. Table no.3 showed PLUM-Ordinal regression analysis between BMI categories and CRP. Adults with BMI of each categories for normal, underweight and overweight had strong statistical significant association with CRP (p = <0.001)). A non significant correlation was observed between CRP concentrations and female gender (p = 0.323), height (p = 0.347) and corrected CRP (p = 0.825).

Table No. 2:C-reactive protein concentration and Demographics, anthropometrics Components of MS

Clinical variable	mean ± SD	P-Value
N (%)	**115(%)**	.
Age (mean ± SD)	29.3 ± 7.7	.001
Male N (%)	85(73.9%)	.975
Female N (%)	30(26.1%)	
BMI(kg/m^2)	28.3 ±3.6	.348
Weights (kg)	62.1 ±12.2	.002
Weight hips ratios	.92 ± .15	<.001*
Heights		.034
Corrected CRP		.020

*Univariate analysis of variances; Chi-Square

Table No.3: BMI Categories and CRP relationship

Clinical variable	Frequency	Valid Percent	P-Value
Normal	56	48.7	<0.001
Underweight	25	21.7	<0.001
Overweight	25	21.7	<0.001
Obese	9	7.8	
Total	115	100.0	

PLUM-Ordinal Regression analysis

DISCUSSION

In this cross-sectional study CRP concentrations were higher in overweight and obese adults(considering BMI).Most of the previous studies with children, adolescents and adult showed a positive association between CRP concentrations and BMI.[18-21] It is an established fact that BMI is not an accurate way to measure adiposity. Our findings are consistent with strong correlation between CRP and higher BMI.This may be suggestive of significantassociation ofadiposity and low grade inflammation. An apparent reason for these findings is that the excess of adipose tissue is able to secrete inflammatory adipokines.The studies have shown that interleukin-6 and a tumor necrosis factor stimulate liver for CRP production.[18-21]Gender body fat deposition has been associated with variedCRP concentration. In female higher mean values of BMI and CRP have been observedas compared to male. We observed direct correlation between CRP concentrations and MS componentse.i. BMI and WHR.

In our study Spearman correlation analysis revealed that MS components were strongly correlated to the concentrations of CRP. The mechanisms underlying the correlation of BMI due to obesity hadnot beenevaluated yet. Obesity is supposed to be to adipose tissue remodeling.This could be explained on the basis of hyperplasia, adipocyte hypertrophy and increased infiltration of immune cells such as lymphocytes and macrophages in adipose tissue.The imbalance between production of pro-inflammatory and anti-inflammatory adipokinesis also a proposed hypothesis. The macrophages infiltration inflammatory markers can act on adipose tissue to control metabolic activities. The disturbances of adipokines, immune system, angiogenesis, lipid metabolism and insulin sensitivity mechanisms come in to play to contribute to the ultimate outcome in obesity.Obesity measured by BMI and WHR are majordeterminants of the MS.[22-23]

The limitations in our study was, the inflammatory biomarker used to show relationship between the exposure and outcome. This CRP concentration was non-specific for subclinical chronic inflammation of adipose tissue metabolism. Parameters not considered in our study were lipid profiles, blood glucose, and insulin level because of logistics and cost implications. Our study demonstrates statistical significant correlation between demographics, anthropometric and CRP concentrations in adults. The elevatedCRP in turn might have implications as a high risk for developing CAD and T2DM later in life.[24]

CONCLUSION

Statistical analysis has shown there is direct correlation between BMI, WHR and CRP concentrations which suggests that inflammation might be an important event in the development of metabolic disorders in adults.

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