

# Correlation of CRP, Fasting Serum Triglycerides and Obesity as Cardiovascular Risk Factors

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## ABSTRACT

**Objective:** To determine the correlation of C-reactive protein (CRP) with fasting triglycerides (TG) among pre-obese and obese patients without established diagnosis of coronary artery disease (CAD).

**Study Design:** A comparative cross-sectional study.

**Place and Duration of Study:** Mayo Hospital, Lahore, from January to June 2010.

**Methodology:** Patients with BMI > 23 kg/m<sup>2</sup> aged between 18 - 65 years were inducted and above variables were studied. Patients with signs of fluid retention, collagen vascular disease, CAD, patients on corticosteroids, immunomodulators or lipid lowering medications and febrile patients were not recruited. Body mass index was also determined. Independent sample t-test was applied to see the mean difference of age, CRP level and triglycerides level in relation to gender. Chi-square test was used to see the association between qualitative variables. ANOVA was applied to see CRP and fasting serum TG level in relation to BMI categories. Pearson correlation and simple linear regression was applied to see the dependency of CRP and triglycerides with BMI. P-value ≤ 0.05 was taken as significant.

**Results:** Raised CRP was major finding among all groups of BMI. Most of obese and pre-obese patients were young and middle aged and belonged to pre-obese group followed by class-1 and class-2 obesity. CRP level increased with body mass index. No such trend was observed for triglycerides. There was an intermediate positive correlation between CRP and BMI and triglycerides and BMI showed a weak negative correlation. If BMI increases by 1 unit on the average, CRP rises by 0.239 times and this unit rise was significant. Whereas 1 unit rise increase in triglycerides on the average cause CRP to decrease -0.006 times but this value was insignificant.

**Conclusion:** Raised CRP and high fasting TG were major findings in all age groups especially among young and middle aged people. Obesity, hypertriglyceridemia and raised CRP are interrelated suggesting that obesity is not only linked to hypertriglyceridemia but vascular inflammation among pre-obese and obese without overt diabetes mellitus causes high CRP as well and this can be used as a marker to predict the future risk of CAD. However, in the absence of dyslipidaemia, raised CRP can still be considered as a strong predictor of CAD and stroke.

**Key Words:** CRP (C-reactive protein). Fasting serum triglycerides (TG). Obesity. BMI (body mass index). Coronary artery disease (CAD).

## INTRODUCTION

Coronary artery disease has multiple risk factors, some of which are modifiable and others are non-modifiable. CRP has always been considered as a non-specific inflammatory marker that is raised not only in inflammatory conditions and infections but also plays an important role in the pathogenesis of coronary artery disease (CAD).<sup>1</sup> CRP is one of the modifiable risk factors for CAD.<sup>2</sup> It is released from liver in response to infections, inflammations or any process that results in cell injury or cell death. It is one of the strong predictors of coronary artery disease and can be used for cardiovascular risk assessment in patients with predilection to develop ischemic heart disease.<sup>3,4</sup>

Obesity is another strong risk factor for cardiac problems. At increasing BMI cardiovascular risk

increases and all this is attributable to the deposition of visceral fat that is found in mesenteric and omental regions. Visceral fat is dangerous than subcutaneous fat as it accelerates the process of atherogenesis and hence the future risk of developing CAD.<sup>5</sup>

Triglycerides are highly atherogenic important component of plasma lipids. They are commonly elevated among the type-2 diabetics, familial combined hyperlipidemia or over-weight people especially those with metabolic syndrome, contributing to cardiovascular risk due to atherogenesis. Multiple research studies suggest that fasting triglycerides are considered to be independent risk factor for CAD but recently some researchers have laid more stress on non-fasting TG as strong predictor of CAD and death.<sup>6</sup>

The main rationale of this study was to determine the correlation of fasting serum triglycerides and CRP levels in pre-obese and obese subjects without established diagnosis of coronary artery disease.<sup>7</sup> Since CRP, raised triglycerides and raised BMI are well known cardiovascular risk factors, so it was important to know whether there existed some relationship between them

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or one factor exerted its effect independent of the other risk factors.<sup>8</sup>

The objective of this study was to determine the correlation of C-reactive protein (CRP) with fasting triglycerides (TG) among pre-obese and obese patients without established diagnosis of coronary artery disease (CAD).

### METHODOLOGY

It was a comparative cross-sectional study. After approval from institutional review board of Mayo Hospital, Lahore, it was conducted in medical wards on one hundred and eight patients who were admitted in emergency or OPD clinic fulfilling study criteria from January to June 2010. The patients who were willing to participate having age between 18 - 65 years and BMI > 23 kg/m<sup>2</sup> were recruited. Patients with signs of fluid retention like cardiac, renal or hepatic failure, ascites due to any cause, collagen vascular disease, febrile illness, patients with established diagnosis of ischemic heart disease (on the basis of clinical history, ECG changes and elevated cardiac enzymes) or patients on corticosteroids, immune modulators or lipid lowering medications were excluded from the study. After assessing recruitment criteria, 108 patients were selected. Informed consent was taken from patients and BMI was calculated using formula:

$$\text{BMI} = \text{weight (kg)}/\text{Height (m)}^2.$$

Instead of WHO criteria, WPRO criteria (BMI criteria for Asians by Regional Office for Western Pacific Region of WHO) for obesity were applied as WPRO criteria have BMI cut off point much lower than WHO criteria.<sup>9</sup> It was important as obesity among Asians is increasing at an alarming rate and is associated with more cardiovascular risk than non-Asians due to higher tendency to accumulate visceral fat at lower BMI.<sup>10</sup> Patients of BMI  $\geq$  23 kg/m<sup>2</sup> were recruited.

According to these criteria of BMI, patients were divided into three groups. First group of pre-obese patients (over weight patients at risk) with BMI of 23 - 24.9 kg/m<sup>2</sup>, second group (class-1 obesity) with BMI between 25 - 29.9 kg/m<sup>2</sup> and third group (class 2 obesity) with BMI > 30 kg/m<sup>2</sup>. After an overnight fast, blood samples were collected for fasting triglycerides and CRP levels. CRP was measured by Latex enhanced nephelometry. Fasting TG was measured by enzymatic assays on a Hitachi analyser. CRP level of < 1 mg/l was considered normal, values between 1 - 2.9 mg/l were considered to be border- line high and those > 3 mg/l were taken as high.<sup>11</sup> Plasma triglyceride level of less than 150 mg/dl were taken as normal, levels between 150 - 199 mg/dl were considered borderline whereas levels more than 200 mg/dl were considered high.<sup>12</sup> Data entry and analysis was done by using Statistical Package for Social Sciences (SPSS) version 15. Quantitative

variables were presented by using mean and standard deviation. Qualitative variables were presented by using frequency table and percentages. Independent sample t-test was applied to see the mean difference of age, CRP level and triglycerides level in relation to gender. Chi-square test was used to see the association between qualitative variables. CRP level, fasting serum triglycerides and body mass index values were categorized and association of these categories was seen with age groups of patients. ANOVA was also applied to see CRP and fasting serum triglycerides level in relation to body mass index categories. Pearson correlation and simple linear regression was applied to see the dependency of CRP and triglycerides with body mass index. P-value  $\leq$  0.05 was taken as significant.

### RESULTS

This study was conducted on 108 subjects with BMI > 23 kg/m<sup>2</sup>. Among these patients, there were 34 (31.5%) males and 74 (68.5%) female patients. As this is a general belief that obese subjects are at risk of developing diabetes mellitus, dyslipidemias, atherosclerosis and cardiovascular disease. Most of the study population belonged to young or middle age group. There were 41 (37.9%) patients between age of 18 - 39, 36 (33.3%) patients in the age group of 40 - 49, 23 (21.3%) patients in the age group of 50 - 59 and only 8 (7.4%) patients were above 60 years (Table I).

We had 66 (61.1%) patients with high CRP level and among these there were 40 (97.5%) patients in young and middle age group. Borderline high CRP was found in 38 (35.2%) patients and among this group 31 (86.1%) patients were between 40 - 49 years. Normal CRP was found in only 4 (3.7%) patients and they were between 40 - 49 years of age. In terms of statistical significance, a significant association was present between CRP and age of the patients ( $p < 0.001$ ) showing that young patients had higher level of CRP in this study (Table I).

Hypertriglyceridemia was found in 68 (62.9%) patients and most of these patients were young and middle aged. Borderline high TG was seen in 23 (21.2%) patients and most of them were more than 50 years of age. Normal TG was found in 17 (15.7%) patients. There was significant association of fasting serum triglyceride levels and age of the patients. Thus, mostly young and middle aged patients exhibited high triglyceride levels ( $p = 0.001$ , Table I).

There were 57 (52.7%) pre-obese patients at risk. Among them, most were young and middle aged. Class-1 obesity was found in 35 (32.4%) patients and class 2 obesity in 16 (14.8%) patients. Body mass index of the patients showed significant association with age of the patients as mostly young and middle aged patients had obesity as compared to other age groups ( $p < 0.011$ ). Mean age of male and female patients was  $47.47 \pm$

**Table I:** CRP, fasting serum triglycerides and body mass index with respect to age group of patients.

	Age groups				p-value
	18 - 39 years	40 - 49 years	50 - 59 years	60 - 65 years	
CRP (ng/l)					
Normal (< 1 ng/l)	0 (0%)	4 (11.1%)	0 (0%)	0 (0%)	< 0.001*
Borderline high (1 - 3 ng/l)	1 (2.4%)	31 (86.1%)	4 (17.3%)	2 (25%)	
High (> 3 ng/l)	40 (97.5%)	1 (2.7%)	19 (82.6%)	6 (75%)	
Triglycerides (mg/dl)					
Normal (< 150 mg/dl)	9 (21.9%)	0 (0%)	8 (34.7%)	0 (0%)	< 0.001*
Borderline high (150 - 199 mg/dl)	4 (9.7%)	0 (0%)	14 (60.8%)	5 (62.5%)	
High (> 199 mg/dl)	28 (68.2%)	36 (100%)	1 (4.3%)	3 (37.5%)	
BMI (kg/m <sup>2</sup> )					
Pre obese (23 - 24.9 kg/m <sup>2</sup> )	18 (43.9%)	27 (75%)	9 (39.1%)	3 (37.5%)	0.011*
Class-1 (25 - 29.9 kg/m <sup>2</sup> )	15 (36.5%)	9 (25%)	7 (30.43%)	4 (50%)	
Class-2 (> 30 kg/m <sup>2</sup> )	8 (19.5%)	0 (0%)	7 (30.43%)	1 (12.5%)	

\*Chi-square's test / Fisher exact's test was applied.

**Table II:** Descriptive statistics for age (years), CRP and triglycerides with respect to gender of patients.

	N	Mean	SD	p-value
Age (years)				
Male	34	47.47	10.41	0.012
Female	74	42.72	8.22	
CRP (ng/ml)				
Male	34	4.85	2.32	0.442
Female	74	4.45	2.60	
Triglycerides (mg/dl)				
Male	34	192.11	46.91	0.170
Female	74	205.06	44.48	
CRP (ng/ml)				
Pre-obese (23 - 24.9)	57	3.75	2.42	< 0.01*
Class-1 (25 - 29.9)	35	4.88	2.24	
Class-2 (> 30)	16	6.81	1.93	
Triglycerides (mg/dl)				
Pre-obese (23 - 24.9)	57	206.59	43.18	0.398
Class-1 (25 - 29.9)	35	195.40	49.39	
Class-2 (> 30)	16	193.25	44.53	

Analysis of Variance (ANOVA) was applied.

10.41 and 42.72 ± 8.22 years respectively. According to p-value, significant difference was present in age of male and female patients. Male patients' age was higher as that of female patients. Mean CRP level in male and female patients was 4.85 ± 2.32 and 4.45 ± 2.60 ng/ml respectively. Mean triglycerides level in male and female was 192.11 ± 46.91 and 205.06 ± 44.48 mg/dl respectively. Similarly, mean CRP and triglycerides levels were statistically same in male and female patients (Table II).

Mean CRP level in pre-obese patients was 3.75 ± 2.42 ng/ml, in class-1 and 2 obese patients it was 4.88 ± 2.24 and 6.81 ± 1.93 ng/ml respectively. It was observed that as body mass index increased, CRP level also increased and significant difference was present in CRP level with respect to body mass index (p < 0.01). This difference was assessed by using ANOVA. As ANOVA was significant, multiple comparison test was also applied to see the mean difference within body mass index categories (Table II).

**Table III:** CRP with respect to body mass index and fasting serum triglycerides.

	Body Mass Index (BMI)			p-value
	(Pre-obese at risk) 23 - 24.9 (kg/m <sup>2</sup> )	(Class-1 obesity) 25 - 29.9 (kg/m <sup>2</sup> )	(Class-2 obesity) > 30 (kg/m <sup>2</sup> )	
C-reactive protein				
Normal (< 1 ng/ml)	4 (7.0%)	0 (0%)	0 (0%)	0.003
Borderline high (1 - 3 ng/ml)	27 (47.3%)	10 (28.5.3%)	1 (6.2%)	
High (> 3 ng/ml)	26 (45.6%)	25 (71.4%)	15 (93.7%)	
Fasting serum triglycerides (mg/dl)				
Normal (< 150 mg/dl)	7 (12.2%)	8 (22.8%)	2 (12.5%)	0.054
Borderline high (150 - 199 mg/dl)	8 (14.1%)	8 (22.8%)	7 (43.7%)	
High (> 199 mg/dl)	42 (73.6%)	19 (54.2%)	7 (43.7%)	
	Fasting Serum Triglycerides			p-value
	(Normal) < 150 mg/dl	(Borderline high) 150 - 199 mg/dl	(High) > 199 mg/dl	
C-reactive protein				
Normal (< 1 ng/ml)	0 (0%)	0 (0%)	4 (5.8%)	< 0.001
Borderline high (1 - 3 ng/ml)	1 (5.8%)	4 (17.5%)	33(48.5%)	
High (> 3 ng/ml)	16 (94.1%)	19 (82.6%)	31 (45.5%)	

Note: p-value ≤ 0.05: Significant

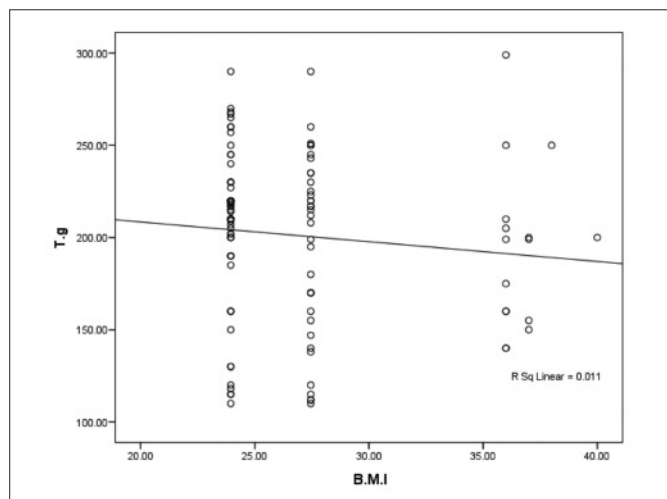


Figure 1: Correlation between body mass index and fasting serum triglycerides.

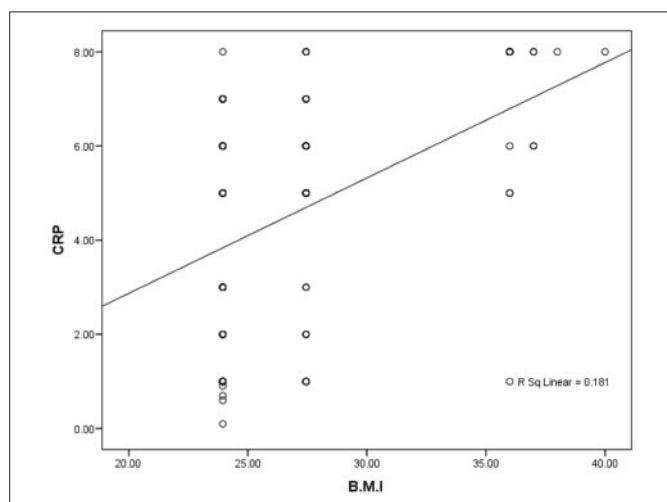


Figure 2: Correlation between body mass index and CRP level.

Mean triglycerides level in pre-obese patients was  $206.59 \pm 43.18$  mg/dl, in class-1 and 2 obese patients it was  $195.40 \pm 49.39$  and  $193.25 \pm 44.53$  mg/dl. No significant difference was present in triglycerides level with respect to body mass index ( $p = 0.398$ , Table II).

Correlation coefficient for CRP and body mass index was 0.425. Correlation between CRP and body mass index was significant and value of  $r$ -shows an intermediate positive correlation between these two variables. On the other hand, correlation coefficient for triglycerides and body mass was ( $r = -0.103$ ) showing a negative and weak correlation between triglycerides and body mass index (Figure 1 and 2).

According to the results, C-reactive protein levels were significantly associated with BMI of patients as most of the obese and pre-obese subjects exhibited raised or borderline high CRP.

High CRP was found in 66 (61.1%) patients. There were 38 (35.2%) patients with borderline CRP levels. Normal

CRP was seen in only 4 (3.7%) patients and all of them were pre-obese with high TG level (Table III).

CRP was significantly raised in pre-obese group and class-1 obese patients ( $p 0.003$ ). High triglycerides were seen in 68 (62.9%) patients. There were 23 (21.3%) patients of borderline high TG level. Normal TG was seen in 17 (15.7%) patients with most patients belonging to pre-obese group and class-1 obesity. Hypertriglyceridemia was a major finding in pre-obese group of patients and class-1 obese group of patients ( $p < 0.054$ , Table III).

Figure 1 describes correlation between body mass index and fasting serum Triglycerides. No linear trend was found between BMI and Triglycerides and correlation coefficient for Triglycerides and body mass index showed weak negative insignificant correlation ( $r = -0.103$ ).

Figure 2 describes correlation between Body mass index and CRP level. Correlation between these two variables was ( $r = 0.425$ ) which showed an intermediate positive correlation. If Body Mass index increases by 1 unit on the average, CRP rises by 0.239 times. This unit rise increase in CRP level in relation to body mass index was significant. Whereas 1 unit rise increase in triglycerides on the average cause CRP to decrease  $-0.006$  times but this unit decrease in CRP level was insignificant in relation to triglycerides.

## DISCUSSION

CRP is a well-known risk factor for cardiovascular disease.<sup>13</sup> But the correlation between CRP, BMI and plasma lipids is not clear that whether they exert their effect independently or they are dependent on each other. Raised CRP and borderline high level was a major finding in all age groups especially young and middle aged patients irrespective of BMI.<sup>14</sup> Many studies suggested an increase in CRP level with advanced age in general population. This is contrary to result of this study as most of the patients with raised CRP were young and middle aged. The possible reason of difference could be different ethnic origin of study populations and the presence of low grade vascular inflammation in overweight and obese patients. According to Larkin *et al.*, CRP was significantly raised in adolescents with obesity and sleep disordered breathing and might conferred increased cardiovascular risk.<sup>15</sup> There was no significant difference regarding BMI and CRP in either gender, anyhow triglyceride levels were significantly raised among females as compared to male patients.<sup>16</sup> According to a meta-analysis in 1996 by Hokanson, fasting triglyceride is independent cardiovascular risk factor due to highly atherogenic triglyceride rich remnant lipoprotein and therefore, it can be a strong predictor of CAD in women and those men with low HDL cholesterol.<sup>17</sup> The prospective Copenhagen male study

showed a positive correlation of TG level with risk of CAD, higher the TG level more will be the cardiovascular risk.<sup>18</sup> Similarly, women health study conducted on about 26509 people, showed association of non-fasting TG with increased cardiovascular risk.<sup>19</sup>

In this study, out of 108 patients, 68 (62.9%) patients had high TG level and 23 (21.3%) patients had borderline high TG. This was indicative of a strong association between increasing BMI and fasting TG level. There were 66 (61.1%) patients with high CRP levels and 38 (35.2%) patients with borderline high CRP and only 4 (3.7%) patients had normal CRP level. This supports the fact that obesity, dyslipidemia and raised CRP can co-exist and are interrelated.<sup>20</sup>

According to a European study by Forouhi *et al.*, adiposity especially the presence of visceral fat is mainly responsible for low grade vascular inflammation and raised CRP levels which are surrogate markers of systemic inflammation and these cardiovascular risks factors are interrelated in the etiology of metabolic syndrome. As obese people are at risk of developing type-2 diabetes mellitus due to insulin resistance, the resulting hyperglycemia can affect collagen cross linking and protein matrix in vessel wall by causing oxidative damage. This releases free radicals that can further contribute to dyslipidemia and atherosclerosis. CRP is also released due to vascular inflammation and thus all these risk factors can play together and result in a cardiovascular event.<sup>21</sup> There were 66 (61.1%) patients with raised CRP and 16 (24.2%) of them had normal TG levels. This finding was supported by results of Umed *et al.*, according to which prevalence of high CRP among patients with normal lipid concentration ranged from 28.8 to 35.3%.<sup>22</sup>

According to Sheikh *et al.*, CRP is raised among patients presenting with acute ischemic chest pain. Since this is a marker of vascular inflammation, therefore, subjects with raised CRP should be treated by stratification of risk factors to improve outcomes in terms of cardiovascular morbidity and mortality.<sup>2</sup>

The present results showed that there were 17 (15.7%) patients with normal triglyceride levels and most of them showed raised CRP level. Similarly, normal CRP was found in only 4 patients and all of them had high TG levels. This signifies that raised CRP causes a low grade inflammation in the arterial walls of obese patients and it can initiate a cascade of atherogenesis and hence poses a threat of coronary artery disease even in the absence of dyslipidaemia.<sup>23</sup> There were 23 (21.3%) obese patients with borderline high triglyceride levels. In this group, 19 patients had CRP level > 3 mg/l and 4 patients had CRP level in the range of 1 - 3 mg/l. This result indicates that CRP is an independent CV risk factor but still synergistic effect of raised CRP along with the presence of dyslipidemia could not only lead to

accelerated process of atherosclerosis but increased risk of future cardiovascular events as well.<sup>24</sup>

There were only 4 patients with normal CRP levels and all four had hypertriglyceridemia. Although it was not a major finding as most of the obese patients with dyslipidemia had manifested either raised or mildly elevated CRP. This supports the fact that higher BMI is associated with raised CRP level irrespective of plasma lipids.<sup>7</sup> Thus, raised CRP was a major finding among most of the pre-obese as well as obese subjects including those with normal triglyceride levels and others with hypertriglyceridemia in this study. This indicates importance of routine assessment of CRP as a cardiovascular risk factor among high risk subjects with one or more risk factors for CAD like diabetes, hypertension, obesity, dyslipidaemia or smoking etc. Stratification of these risk factors by life style modification and pharmacological treatment should be the ultimate objective in all such patients for primary prevention of CAD.<sup>25</sup>

## CONCLUSION

Raised CRP and high fasting TG were major findings in all age groups especially among young and middle aged people. Most of the study population belonged to pre-obese group (52.7%) followed by class-1 and class-2 obesity. Obesity, hypertriglyceridemia and raised CRP are interrelated suggesting that increasing BMI results in vascular inflammation among pre-obese and obese without overt diabetes mellitus and this leads to high CRP level which can be used as a marker to predict the risk of future CAD. Stratification of risk factors at this stage by implementation of life style modification is important for primary prevention of CAD. However, in the absence of dyslipidaemia, raised CRP can still be considered as a strong predictor of CAD and stroke.

## REFERENCES

1. Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. *N Engl J Med* 1999; **340**:448-54.
2. Sheikh AS, Sheikh A, Sheikh S, Yahya S. the prognostic significance of C-reactive protein as an independent predictor for coronary events in patients presenting with chest pain. *The Intern J Cardiol* 2010; **8**:1.
3. Danesh J, Whincup P, Walker M, Lennon L, Thomson A. Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. *Br Med J* 2000; **321**: 199-204.
4. Pepys MB, Berger A. The renaissance of C-reactive protein. It may be a marker not only of acute illness but also of future cardiovascular disease. *BMJ* 2001; **322**:4-5.
5. McLaughlin T, Abbasi F, Lamendola C, Liang L, Reaven G. Differentiation between obesity and insulin resistance in the association with C-reactive protein. *Circulation* 2002; **106**: 2908-12.
6. Libby P, Ridker PM, Maseri A. Clinical cardiology: new frontiers inflammation and atherosclerosis. *Circulation* 2002; **105**:1135-43.

7. Khan S, Hafizullah M, Faheem M, Nadeem M, Shah I. Level of high-sensitivity C-reactive protein in patients with chronic stable angina. *J Med Sci* 2012; **20**:124-7.
8. Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Elevated C-reactive protein levels in overweight and obese adults. *JAMA* 1999; **282**:1231-5.
9. Anuurad E, Shiwaku K, Nogi A, Kitajima K, Enkhmaa B. The new BMI criteria for Asians by the Regional office for Western Pacific Region of WHO are suitable for screening of overweight to prevent metabolic syndrome in elder Japanese workers. *J Occup Health* 2003; **45**:335-43.
10. Karthikeyan G, Koon K, Islam S, McQueen JM, Prem P. Lipid profile, plasma apolipoproteins, and risk of a first myocardial infarction among Asians an analysis from the interheart study. *J Am Coll Cardiol* 2009; **53**:244-53.
11. Fogoros RN. New guidelines on CRP testing: new recommendations from the AHA and CDC [Internet]. [updated 2003 Nov 28]. Available from: <http://heartdisease.about.com/cs/generallinks/a/syncope seizure.htm>
12. McPhee SJ, Papadakis MA, Baron RB. Lipid disorders. *Curr Med Diagn Treatment* 2010; **49**:1123-33.
13. Ridker PM. Clinical application of C-reactive protein for cardiovascular disease detection and prevention. mini-review: expert opinions. *Circulation* 2003; **107**:363-9.
14. Williams MJ, Milne BJ, Hancox RJ, Poulton R. C-reactive protein and cardiorespiratory fitness in young adults. *Eur J Cardiovasc Prev Rehabil* 2005; **12**:216-20.
15. Larkin EK, Rosen CL, Kirchner HL, Isser AS, Emancipator JL. Variation of C-reactive protein levels in adolescents association with sleep-disordered breathing and sleep duration. *Circulation* 2005; **111**:1978-84.
16. Thorand B, Baumert J, Doring A, Herder C, Kolb H. Sex differences in the relation of body composition to markers of inflammation. *Atherosclerosis* 2006; **184**:216-24.
17. 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.
18. Langsted A, Freiberg JJ, Nordestgaard BG. Fasting and non-fasting lipid levels. influence of normal food intake on lipids, lipoproteins, apolipoproteins, and cardiovascular risk prediction. *Circulation* 2008; **118**:2047-56.
19. Mora S, Rifai N, Buring JE, Ridker PM. Fasting compared with non-fasting lipids and apolipoproteins for predicting incident cardiovascular events. *Circulation* 2008; **118**:993-1001.
20. Parvez A, Ihsanullah, Rafiq A, Ahmad N, Khan EH. Relationship of glycemia and triglycerides with BMI in diabetic patients. *J Ayub Med Coll Abbottabad* 2010; **22**:147-9.
21. Forouhi NG, Sattar N, McKeigue PM. Relation of C-reactive protein to body fat distribution and features of the metabolic syndrome in Europeans and South Asians. *Inter J Obesity* 2001; **25**:1327-31.
22. Ajani UA, Ford ES, Mokdad AH. Prevalence of High C - reactive protein in persons with serum lipid concentrations within recommended values. *Clin Cam* 2004; **50**:1618-22.
23. Ridker PM, Hennekens CH, Buring JE, Rifai N. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. *N Engl J Med* 2000; **342**: 836-43.
24. Ishaq M, Beg MS, Ansari SA, Hakeem A, Ali S. Coronary artery disease risk profiles at a specialized tertiary care center in Pakistan. *Pak J Cardiol* 2003; **14**:61-8.
25. Clearfield MB. C - reactive protein: a new risk assessment tool for cardiovascular disease. *JAOA* 2005; **105**:409-16.

