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ELEVATED SERUM CRP LEVELS IN SMOKERS: A COMPARERIVE CROSS SECTIONAL STUDY AT HYDERABAD, PAKISTAN

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Abstract:

C-reactive protein is a marker of inflammation that possesses a diagnostic value in much ongoing pathology. The habit of Smoking is so common that almost all age groups of both genders are involved in this around the globe. We aimed this study to evaluate the serum levels of this inflammatory marker in smokers and compare it with the non-smoker volunteers. It is already a known fact that smoking predisposes a person to many diseases (heart and lung diseases to malignancies).We aimed to evaluate and compare serum CRP levels in smokers and non- smokers. This cross sectional study was carried out at department of biochemistry, Isra hospital lab at Isra University Hyderabad that lasted for 6 months June 2016- Dec 2016. The project was approved by institutional ethical review committee inclusion and exclusion criteria were constructed for Smokers and non-smoker subjects which were selected by probability sampling. A total 60 individuals was selected for study and divided into group A (30 non-smokers) and group B (30 smokers). Demographic data was sought on proforma was designed for demographic data collection. Serum CRP levels were assessed in Isra hospital lab after drawing the blood from the willing and consent providing individuals. Collection of blood was under aseptic measures. Serum mean C-reactive protein levels in smokers was 1.09±0.82mg/dl while it was0.27± 0.23mg/dl in non- smokers that was significant statistically (p-value 0.001). BMI was calculated from height and weight and comparison was done among two groups using student's t-test on SPSS Version 22. Frequency and percentage was counted for duration of smoking and number of cigarette smoked per day. Mean age of the study population was 35.5 years, minimum age was 26 and maximum was 44 years. There was no significant difference in BMI of the smokers 29.47+ 3.81 and non-smokers 27.71+3.56Kg/m^2 (p-value calculated was 0.67).Serum CRP level was found significantly higher in smokers in comparison to non-smokers.

Key Words: *CRP, Atherosclerosis, Smoking*

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INTRODUCTION:

Systemic inflammation results into elevation of about 30 polypeptides in acute phase response multiple etiologies e.g infection, diabetes, hypertension and injuries etc but erythrocyte sedimentation rate(ESR) and C-reactive protein (CRP) are more commonly used in clinical practice[39].CRP has two main physiological functions first is activating the Classical complement pathway through its binding to C1q [1]. Attaching the human immunoglobulin Fc receptors and thus opsonizing the particles for macrophages is another job of CRP [2-4]. CRP is also involved atherosclerotic lesions through reduction of the nitric oxide (NO) synthase expression along with prostacyclin synthase and adhesion molecules and enhancing the LDL-C uptake by macrophages [5]. CRP is measured differently by various laboratories according to their thresholds but normal CRP level is considered as < 10 mg/L or < 1 mg/ dl [6]. Normal CRP levels are higher in pregnancy with and without preeclampsia owing to mild systemic inflammation [7]. Tobacco Smoking causes tissue injury and increases certain pro-inflammatory substances including CRP, interterleukins-6, TNF-α and Fibrinogen etc. [8]. Literature supports elevation of CRP in smokers [9]. Smoking is a major risk for the development of atherosclerosis along with lack of physical activity, dyslipidemia, obesity and depression [10].Trend of Tobacco smoking is increasing day by day so among the young generation despite of much public awareness about its harmful effects. At what stage smoking brings about the pathology is not exactly known but early detection of inflammatory markers especially the CRP will help to prevent these diseases in the early phase. Serum CRP levels may help as screening tool for smokers especially in the early decades of life. So we designed this cross section study to see the difference in serum CRP levels among the smokers and non-smokers.

METHODOLOGY:

ERC (ethical review committee) of the institute approved this project research design on 60 volunteers selected by random sampling. Two groups were made as group A (30 smokers0 and group B (30 non-smokers). Consent was obtained (Informed written) from the study subjects and data was obtained on a proforma prepared for this study. Samples collection of blood for serum CRP levels was carried out according to human laboratory protocols jell tubes were used to carry the blood samples to research laboratory of the Isra University hospital.

Inclusion criteria:

Chronic smokers with ≥ 4 cig/day for ≥5 years

Age range 20-50 years,

Smokers and non- smokers without any known illness

Exclusion criteria:

On off smokers or seasonal smokers

Age above 50years and below 20 years

Chronic diseases patients

Statistical Analysis:

SPSS Version 22 and student's t-test were used tools to compare mean serum CRP levels and BMI between smokers and non-smokers. Frequency and Percentage were calculated for duration and number of cigarette smoked. Significant figure was a p-value <0.05.

RESULTS:

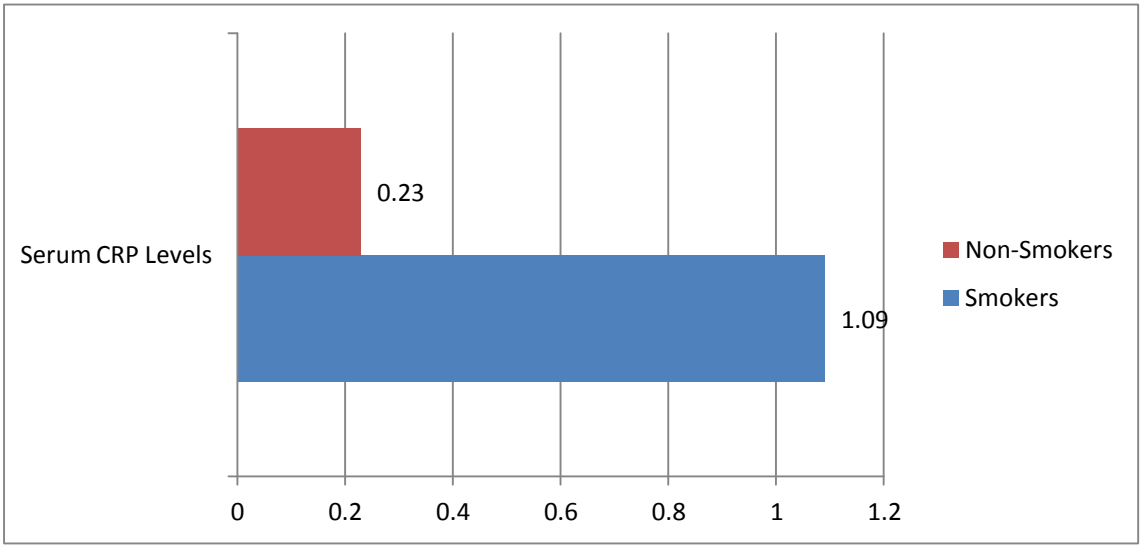
Study population mean age was 35.5 years with 26 years minimum and 44years maximum was 44. Mean C-reactive protein levels in group A (smokers) was 1.09±0.82mg/dl and in group B (non-smokers)it was0.27± 0.23mg/dl p-value 0.001 (significant statistically) . BMI was calculated from height and weight and found non- significant statistically smokers (29.47+ 3.81Kg/m^2) and non-smokers (27.71+3.56Kg/m^2) with p-value calculated 0.67(Table01). Frequency and percentage50% of the study population was smoking 12-15 cig/day while 26.66% were smoking ≥20cig/day and smokers of 8-10 cig/day were 20%. The percentage of 8-10 years smokers was 50% while it was 26.66% for 10-12 years smokers and20% were smoking for a duration above 12 years however only 3.4% of the participants were found smoking for 5-7 years(Table 02).

Table 01. Comparison of mean serum CRP levels and BMI among study participants

Study Parameters and Units	Group A(Smoker n=30)	Group B(Non-Smoker n=30)	P-Value
Serum CRP (mg/dl)	1.09±0.82	0.27± 0.23	0.001
Body Mass Index (Kg/m^2)	29.47+ 3.81	27.71+3.56	0.67

Table 02. Frequency and percentage of number of cigarette smoked and duration of smoking

Duration of Smoking	Frequency and percentage	No. of Cig/day	Frequency and percentage
Above 12 Years	06 (20%)	Above 20cig/day	08 (26.66%)
10-12 Years	08 (26.66%)	12-15 cig/day	15 (50%)
8-10 Years	15 (50%)	8-10 cig/day	07 (20%)
5-7 Years	01 (3.4%)	-------	-------



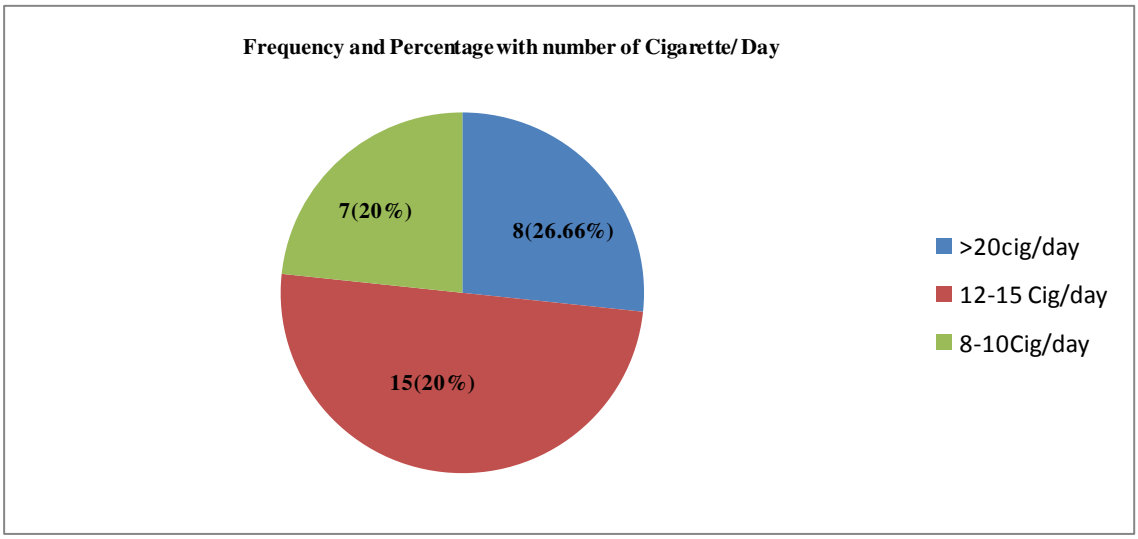


Fig 02: Pie chart of the frequency and percentage in terms of number of cigarette/day

DISCUSSION:

Consistent tour finding were results reported by Joshi AR et al(2013) showing raised CRP levels cases(smokers) in comparison to controls(non-smokers)[11]. Results by Ohsawa et al(2005) also favor our results with elevated CRP levels among cigarette smoking subjects in respect to non-smokers[12]. Mahrukh S et al(2011) concluded her results increased CRP along with C3, C4 (complement system markers) among smokers that is consistent to our finding although she attributed these changes with monocyte activation but we were limited to CRP levels due to financial issues [13]. Observations of Aral et al (2006) are also similar to our results [14]. NHANES-III (National Health and Nutrition Examination Survey) reported tobacco inhalation substantially contributes to certain pathologies with elevated CRP along with leukocytes count and fibrinogen levels [15]. Studies by Sin et al and Pinto-Plata et al (2006) also agree with what we observed [16,17]. Chronic elevation of CRP is also mediated by IL-6and both are may be the indicators of progression of any growth or malignancy smoking being specifically associated with lung cancer [18-21]. CRP has been suggested by some researchers as an independent predictor of cardiovascular diseases [22,23]. Synthesis of CRP by hepatocytes is stimulated by cytofactors released from adipose tissues along with nicotine [24]. Raised CRP is also observed in obesity, hypertension and diabetes [25]. According to WHO there exist an approximate 1 billion tobacco consumers world around with representing one third of 15 years of age. IL- 1, IL-6, amyloid A and in particular CRP have strong impact on vascular diseases [26]. Smokers are at higher risk for multiple diseases as well as it drains a big proportion from the pocket money so it should be avoided specially our youth should be educated through seminars and workshops as the smoking trend is also increasing in females. Screening of young smokers is suggested through serum CRP levels and those having higher levels should be further evaluated for many tobacco-associated diseases so that timely early stage management and effective plans may be adopted.

CONCLUSION:

Serum CRP are found elevated in cigarette smokers as compared tonon-smokers

REFERENCES:

1. M. H. Kaplan and J. E. Volanakis (1974). Interaction of C reactive protein complexes with the complement system. I. Consumption of human complement associated with the reaction of C reactive protein with pneumococcal C polysaccharide and with the choline phosphatides, lecithin and sphingomyelin, Journal of Immunology 112(6):2135–2147.
2. M. Di Napoli, M. S. Elkind, D. A. Godoy, P. Singh, F. Papa, and A. Popa-Wagner(2011).Role of C-reactive protein in cerebrovascular disease: a critical review. Expert Review of Cardiovascular Therapy 9(12):1565–1584.
3. D. Bharadwaj, M.-P. Stein, M. Volzer, C. Mold, and T. W. Du Clos (2007). The major receptor for C-reactive protein on leukocytes is Fcγ receptor II. Journal of Experimental Medicine 190(4): 585–590.
4. M.-P. Stein, C. Mold, and T. W. Du Clos (2000). C-reactive protein binding to murine leukocytes requires Fcγ receptors. Journal of Immunology 164(3):1514–1520.
5. Oliver zimmermann, kefei li, myron zaczkiewicz, matthias graf, zhongmin liu, jan torzewski (2014) c-reactive protein in human atherogenesis: facts and fiction,mediators of inflammation. Article ID 561428, 6 pages http://dx.doi.org/10.1155/2014/561428
6. Samar Firdous, M. Omar Khan Lodhi, Kashif Siddique(2016). CRP among normotensive overweight and obese patients, JCPSP 26(3): 191-194.
7. Zaima Ali, Faraz Ahmed Bokhari, Saima Zaki, Uzma Zargham, Ambreen Tauseef, Shaheena Khakan(2015).Correlation of CRP Levels in Third Trimester with Fetal Birth Weight in Pre-eclamptic and Normotensive Pregnant Women JCPSP 25(2): 111-114.
8. Richard A. Brown,Eduard Shantsila, ChetanVarma, Gregory Y.H. Lip. amjmed.(2016).10.022.
9. Dilyara G. Yanbaeva, Mieke A. Dentener, Eva C. Creutzberg, Geertjan Wesseling, Emiel F. M. Wouters. (2007). 131(5):1557-1566.
10. Ashique A A, Abdul R M, Humayion K, Barkat A M(2017) Reduction of Serum Lipid Profile by Escitalopram in Depressive Patients: A Cardio Protective Aspect of SSRI Use. J Cardiol & Cardiovasc Ther 4(4): 555642. DOI: 10.19080/JOCCT.2017.04.555642
11. Joshi AR, Salvi S, Phadke AV(2013). Platelet Aggregability And C-Reactive Protein In Male

Smokers. International Journal of Basic and Applied Physiology 2(1):109-13.

12. Ohsawa M, Okayama A, Nakamura M, Onoda T, Kato K, Itai K(2005). CRP levels are elevated in smokers but unrelated to the number of smokers and are decreased by long term smoking cessation in male smokers. Preventive Medicine Journal 41(2):651-656.
13. Mahrukh S, Nageen H (2011). Levels of inflammatory markers complement C3, complement C4 and C-reactive protein in smokers. African Journal of Biotechnology. 10(82): 19211-19217.
14. Aral M, Ekerbicer HC, Celik M, Ciragil P, Mustafa G (2006). Research communication: Comparison of effects of smoking and smokeless tobacco "Maras powder" use on humoral immune system parameters. Mediator Inflammation Journal. 20:1-4.
15. Gan WQ, Man SF, Sin DD(2005) The interactions between cigarette smoking and reduced lung function on systemic inflammation. Chest. 127:558-564.
16. D D Sin, N R Anthonisen, J B Soriano, A.G Agusti(2006). Mortality in COPD: Role of comorbidities. European Respiratory Journal 28: 1245-1257.
17. Pinto Plata VM, Mullerova H, Toso JF, Feudjo-Tepie M, Soriano JB, Vessey RS(2006). C-reactive protein in patients with COPD in control smokers and non-smokers. Thorax 61(1):23-28.
18. M. Y. Abeywardena, W. R. Leifert, K. E. Warnes, J. N. Varghese, and R. J. Head(2009). Cardiovascular biology of interleukin-6 ,Current Pharmaceutical Design 15;15; 1809–1821
19. W. B. Ershler and E. T. Keller (2000). Age-associated increased interleukin-6 gene expression, late-life diseases, and frailty," Annual Review of Medicine, 51;245–270.
20. J. A. Nettleton, L. M. Steffen, E. J. Mayer-Davis et al(2006).Dietary patterns are associated with biochemical markers of inflammation and endothelial activation in the Multi-Ethnic Study of Atherosclerosis (MESA)," American Journal of Clinical Nutrition, 83;6;1369–1379.
21. M. Hara, Y. Matsuzaki, T. Shimuzu et al (2008).Preoperative serum C-reactive protein level in non-small cell lung cancer," Anticancer Research 27(4):3001–3004
22. D.-K. Kim, S. Y. Oh, H.-C. Kwon et al (2009).Clinical significances of preoperative serum interleukin-6 and C-reactive protein level in operable gastric cancer," BMC Cancer 9;155
23. J. Danesh, P. Whincup, M. Walker et al (2000). Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses, British Medical Journal 321;199–204.
24. Lao XQ, Jiang CQ, Zhang WS, Adab P, Lam TH, Cheng KK, et al(2009). Smoking, smoking cessation and inflammatory markers in older Chinese men-The Guangzhou Biobank Cohort Study. Atherosclerosis 203: 304-10.
25. Dehghan A, Kardys I, Demaat MP, Uitterlinden AG, Sijbrands EJ, Bootsma AH, et al(2007). Genetic variation, C-reactive protein levels, and incidence of diabetes. Diabetes 56: 872-8.
26. AliyaHisam, Mahmood Ur Rahman, EhsanKadir, NailaAzam and Sumaira Masood(2014). Proportion of exposure of passive smoking in teenage group and symptoms precipitated after exposure to second hand smoke, Journal of the College Physicians and Surgeons Pakistan 24(6): 446-448.