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Correlation Between C-Reactive Protein and Blood Sugar Profile and Lipid Profile in Obesity After Lifestyle Modification for 3 Months

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ABSTRACT

Background. C-reactive protein (CRP) is a phylogenetically highly conserved plasma protein that participates in the systemic response to inflammation and its plasma concentration increases during inflammatory states. The purpose of the study was to determine the correlation between CRP and blood sugar profile and lipid profile in obesity undergoing lifestyle modification for 3 months.

Method. The study was conducted on hospital nurses with BMI >25 (obesity) who were selected voluntarily. Before making lifestyle modifications (diet and physical exercise) for 3 months, patients are given lighting, understanding, and training on lifestyle modifications. Patients were examined for BMI, FPG, PPG, HbA1c, LDL, HDL, and ApoB before and after 3 months of lifestyle modification.

Result. Lifestyle modification for 3 months had been performed on 20 patients with a BMI of 30.9±4.15 kg /m^2 (obesity). There is a significant correlation between CRP and PPG, HbA1C, LDLC, and ApoB ($p < 0.005$).

Conclusion. There is a significant correlation between CRP and glucose profile and lipid profile on obesity undergoing lifestyle modification for 3 months.

Keywords: CRP, Lifestyle Modification, Profil Glucose, Profil Lipid

ABSTRAK

Latar Belakang. *C-reactive Protein (CRP) adalah protein plasma yang sangat terkonservasi secara filogenetik dan mempunyai respons sistemik terhadap peradangan dan konsentrasinya meningkat selama keadaan inflamasi. Tujuan dari penelitian ini adalah untuk mengetahui korelasi antara CRP dan profil gula darah dan profil lipid pada obesitas yang menjalani modifikasi gaya hidup selama 3 bulan.*

Metode*. Penelitian dilakukan pada perawat rumah sakit dengan BMI >25 (obesitas) yang dipilih secara sukarela. Sebelum melakukan modifikasi gaya hidup (diet dan latihan fisik) selama 3*

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bulan, pasien diberikan pencerahan, pemahaman, dan pelatihan modifikasi gaya hidup. Pasien diperiksa terhadap BMI, FPG, PPG, HbA1c, LDL, HDL, dan ApoB sebelum dan sesudah 3 bulan modifikasi gaya hidup.

Hasil. *Modifikasi gaya hidup selama 3 bulan telah dilakukan pada 20 pasien dengan BMI 30,9±4,15 kg/m^2 (obesitas). Ada korelasi yang signifikan antara CRP dan PPG, HbA1C, LDLC, dan ApoB ($p < 0,005$).*

Kesimpulan. *Terdapat korelasi yang signifikan antara CRP dengan profil glukosa dan profil lipid pada obesitas yang mengalami modifikasi gaya hidup selama 3 bulan.*

Kata Kunci*: CRP, Modifikasi Gaya Hidup, Profil Glukosa, Profil Lipid*

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1 Introduction

C-reactive protein (CRP) is a phylogenetically highly conserved plasma protein that participates in the systemic response to inflammation and its plasma concentration increases during inflammatory states, a characteristic that has long been employed for clinical purposes.[1] Tissue necrosis is a potent acute phase stimulus, and following AMI, there is a major CRP response, the magnitude of which reflects the extent of myocardial necrosis.[2]

CRP is produced by the liver and adipocytes and its levels may increase in response to active infection or acute inflammation. It is regulated by cytokines IL-1, IL-6, and TNF-α, and moderate increases are linked to chronic inflammatory conditions such as atherosclerosis independently of the lipid parameters. It is also related to insulin resistance and hyperglycemia.[3] C-reactive protein measured by highly sensitive assays (hs-CRP), is a very sensitive marker of the inflammatory activity in the arterial wall.[4] It is an important predictor of cardiovascular risk apart from the traditional risk factors.[5] It is interesting to note that chronic hyperglycemia stimulates the release of various inflammatory cytokines (IL 6; TNF α) and induces the secretion of acute phase reactants by the liver, which in turn results in the elevation of CRP in association with elevated fasting plasma glucose.[6] Studies had shown that elevated CRP levels are associated with an increased risk of future development of diabetes mellitus. Also, people with diabetes mellitus had elevated levels of CRP than non–diabetics.[7] Obesity in association with MS leads to the production, besides CRP, of high levels of proinflammatory markers.[8] A moderate increase in the levels of hs-CRP is associated with chronic inflammatory conditions such as atherosclerosis.[9]

In atherosclerotic lesions, hs CRP interacts with lipoprotein LDL resulting in a succession of atherosclerotic plaque.[10] In comparison to other inflammation markers, the levels of C-reactive protein are stable with no diurnal variation and can be estimated easily. An increased level of CRP was related to an increased level of serum insulin higher and hyperglycemia.[11] Increased values of

serum C-reactive protein or CRP in diabetics is evidence that Inflammation also plays a key role in developing the complications of diabetes.[12]

2 Method

The study was conducted on hospital nurses with BMI >25 (obesity) who were selected voluntarily. Before making lifestyle modifications (diet and physical exercise) for 3 months, patients are given lighting, understanding, and training on lifestyle modifications.

Patients were examined for BMI, FPG, PPG, HbA1c, LDL, HDL, and ApoB before and after 3 months of lifestyle modification Plasma lipids and glucose were measured centrally by fully automated techniques. We measured concentrations of LDLC by Friedewald formula, HDLC by enzymatic techniques, and ApoB by immunoturbidimetry

Statistical analysis

Statistical analysis was done using the SPSS package and MS excel. Pearson correlation or Spearman correlation test and p values were calculated. P values <0.05 was considered to be significant.

3 Result

Twenty patients who carried out lifestyle modifications for 3 months with a BMI of 30.9±4.15 kg / m2 (obesity)

Table 1 Basic Research Data

Parameters	Mean±SD (n=20)
BMI (kg/m^2	30.98 ±4.15
FPG (mg/dl)	91.70±20.64
PPG (mg/dl)	112.35±37.71
HbA1c	5.52±0.68
LDLC (mg/dl)	129.40±29.88
HDLC (mg/dl)	45.30±10.01
ApoB (mg/dl)	100.25±19.05
CRP (mg/dl)	3.00±2.22

Abbreviations: BMI: body mass index; FPG: fasting plasma glucose; PPG: postprandial plasma glucose; HbA1c, glycosylated hemoglobin; LDLC: low-density lipoprotein cholesterol; HDLC, high-density lipoprotein cholesterol; ApoB: apolipoprotein B

Based on table 2, there is a significant correlation between CRP and PPG, HbA1C, LDLC, and ApoB ($p < 0.005$)

Table 2 Correlation between CRP and Profile of Blood Sugar and Lipid Profile

Parameters	r	p
BMI (kg/m^2	0.198	0.201
FPG (mg/dl)	0.344	0.096
PPG (mg/dl)	0.433	0.028*
HbA1c	0.424	0.031*
LDLC (mg/dl)	0.592	0.003*
HDLC (mg/dl)	0.097	0.342
ApoB (mg/dl)	0.431	0.029*

Abbreviations: BMI: body mass index; FPG: fasting plasma glucose; PPG: postprandial plasma glucose; HbA1c, glycosylated hemoglobin; LDLC: low-density lipoprotein cholesterol; HDLC, high-density lipoprotein cholesterol; ApoB: apolipoprotein B

4 Discussion

The level of LDL-chol is modified as oxidized LDLs can begin inflammation by stimulating endothelial cells to release CRP.[13] This release of CRP stimulates the oxidized form of LDL receptor 1 from macrophages, which in turn enhances the uptake of modified form LDLs. CRP also induces the activation of complement via activation of oxidized LDL receptor 1, this activation of this receptor help in vasomotor dysfunction, endothelial exudation, and proatherogenic actions of CRP.[14] A direct correlation between the level of hs-CRP with the level of oxidized LDL and forecast myocardial infarction and acute coronary syndrome.[15] We found significantly higher levels of total cholesterol, TG, LDL, VLDL, CRP, and lower levels of HDL in AMI compared to that of control subjects. We also found a strong positive correlation of CRP with total cholesterol, triglyceride, LDL-C, and VLDL-C and a significant negative correlation with HDL-C in AMI patients.[16] A significant positive correlation of inflammatory marker hs CRP was observed with fasting blood sugar, serum cholesterol, and LDL- cholesterol. On the other hand, a non-significant negative correlation between inflammatory markers was observed with serum HDL cholesterol and triglycerides.[17] There was a significant positive correlation between CRP and total cholesterol (p<0.05). There was no significant correlation between CRP and LDL cholesterol (p>0.05). There was a negative correlation between HDL cholesterol and CRP. There was a significant positive correlation between CRP and triglyceride levels (p<0.05). There was a significant correlation between CRP and HbA1C (p<0.05).[18] In this obesity study, there was a significant correlation between CRP and FPG, HbA1c, LDL, and ApoB, the results of this study were different from other studies because they had cardiovascular disease.

5 Conclusion

There is a significant correlation between CRP and glucose profile and lipid profile on obesity undergoing lifestyle modification for 3 months.

REFERENCES

[1]. Black S, Kushner I, Samols D. C-reactive Protein. J Biol Chem. 2004;279(47):48487-90.

[2]. Pepys MB, Hirschfield GM. C-reactive protein: a critical update. J Clin Invest. 2003;111:pp.1805-12.

[3]. Dongway, A.C., Faggad, A.S., Zaki, H.Y. and Abdalla, B.E. C-Reactive Protein has Associated with Low-Density Lipoprotein Cholesterol and Obesity in Type 2 Diabetic Sudanese. *Diabetes, Obesity & Metabolism*: *Targets and Therapy*, 2015,**8**:pp.427-435.

[4]. Kamath DY, Xavier D, Sigamani A, Pais P. High sensitivity C-reactive protein (hs-CRP) & cardiovascular disease: An Indian perspective. Indian J Med Res 2015;142:pp.261-8

[5]. Mohieldein AH, Hasan M, El-Habiby MI. High Sensitivity C-Reactive Protein as Atherogenic Marker Among Type 2 Diabetes. Eur Sci J 2017;33(13):pp.403

[6]. Lin Y, Rajala MW, Berger JP. Hyperglycaemia induced production of acute phase reactants in adipose tissue. J Biol Chem. 2001;276(45):pp.42077- 83

[7]. Tutuncu Y, Satman I, Celik S, Dinccag N, Karsidag K, Telci A. A comparison of hs-CRP levels in new diabetes groups diagnosed based on FPG, 2- hPG, or HbA1c criteria. J Diabe Res 2016.

[8]. Contreras-Rodríguez, A., Gómez-Díaz, R.A., Tanus-Hajj, J., Talavera, J.O., Mondragón-González, R. and Wacher, N.H. (2015) Carotid Intima-Media Thickness, Ankle-Arm Index, and Inflammation Profile in Mexican Patients with Early and Late Onset Type 2 Diabetes. *Revista de Investigación Clínica*, 2015;**67**:pp.240-9

[9]. Coffman, E. and Richmond-Bryant, J. Multiple Biomarker Models for Improved Risk Estimation of Specific Cardiovascular Diseases Related to Metabolic Syndrome: A Cross-Sectional Study. *Population Health Metrics*, 2015;**13**:pp.7. http://dx.doi.org/10.1186/s12963-015-0041-5

[10]. Tsai S, Clemente-Casares X, Revelo XS, Winer S, and Winer DA, "Are obesity-related insulin resistance and type 2 diabetes autoimmune diseases?" Diabetes 2015;64(6):pp.1886-97

[11]. Uemura H, Katsuura-Kamano S, Yamaguchi M. Relationships of serum high-sensitivity C- reactive protein and body size with insulin resistance in a Japanese cohort. Herder C, ed. PLoS ONE. 2017;12(6):e 0178672.

[12]. Devaraji S, Dasu MR, Jialal I. Diabetes is a proinflammatory state: a translational perspective Expert Rev Endocrinol Metab. 2010 Jan 1;5(1):pp.1928.

[13]. Chu CS, Wang YC, Lu LS et al. Electronegative low-density lipoprotein increases C-reactive protein expression in vascular endothelial cells through the LOX-1 receptor. PLoS One 2013; 8 (8):e70533

[14]. Hein TW, Qamirani E, RenY. Selective activation of lectin-like oxidized low-density lipoprotein receptor-1 mediates C-reactive protein-evoked endothelial vasodilator dysfunction in coronary arterioles. Circ Res 2014;114(1):pp.92-100

[15]. Lin GM, Liu K, Colangelo LA, Lakoski SG, Tracy RP, Greenland P. Low-Density Lipoprotein Cholesterol Concentrations and Association of High-Sensitivity C-Reactive Protein Concentrations With Incident Coronary Heart Disease in the Multi-Ethnic Study of Atherosclerosis. Am J of Epidemiol 2016;183(1):pp.4652

[16]. Rathore V, Singh N, Rastogi P, Mahat RK, Mishra MK, Shrivastava R. International Journal of Research in Medical Sciences. Lipid profile and its correlation with C-reactive protein in patients of acute myocardial infarction. Int J Res Med Sci. 2017 May;5(5):pp.2182-6

[17]. Naim Ahmad Nizami, Rukhsana Malik, Asad Mahmood Khan and Badar Jamal. Correlation of Inflammatory C-reactive Protein with Lipid Profile and Fasting Blood Sugar in Type-2 Diabetic Using Insulin Esculapio 2018:14(04):pp.246-9

[18]. Gulati SK. Study To Evaluate The Correlation Between Various Biochemical Parameters (Hba1c, Lipid Profile, And Crp) Among Type 2 Diabetes Mellitus Patients. International Journal of Advanced Research in Medicine 2020; 2(1):pp.75-8. https://doi.org/10.22271/27069567.2020.v2.i1a.131