



Research Paper

Comparison of C - reactive protein in normotensive & hypertensive type 2 diabetic patients

Authors

Randhir K.Pandey^{1*}, Suresh Babu Kondaveeti², Manju Pandey³, Madhuri Gupta⁴

^{1,2,4}Department of Biochemistry, ³Department of Gen. Medicine

National Institute of Medical Sciences and Research, Jaipur

*Corresponding Author

Dr Suresh Babu Kondaveeti

Associate Professor, Department of Biochemistry, National Institute of Medical Science & Research, NIMS University, Jaipur, Rajasthan

Abstract

Background: *The present study was aimed to comparison the C-reactive protein with microalbuminuria among type 2 diabetic subjects in hypertension.*

Introduction: *CRP production is part of the nonspecific acute-phase response to most forms of inflammation, infection and tissue damage. The association between CRP and hypertension could be related in part as follows: correlation between elevated CRP and arterial stiffness; association between CRP and metabolic syndrome, one of whose criteria is hypertension and the possibility that CRP may directly contribute to reduced nitric oxide synthesis in endothelial cells, leading to increased vascular resistance.*

Material & Methods: *The present study includes of 500 type 2 diabetic subject & divided into two groups based on hypertension. Biochemical parameter such as C-reactive protein (CRP) was analysed in the department of biochemistry of NIMS medical college. Hypertension was taken according to definition of WHO; as systolic blood pressure (SBP) ≥ 140 mmHg and diastolic (DBP) ≥ 90 mmHg.*

Result: *There was significant difference between the patient and control groups with regard to age, blood pressure, lipid profile, and CRP. The study also showed significant positive correlations between CRP, age and blood pressure.*

Conclusion: *The levels of CRP were elevated in hypertensive individuals, which suggests the possibility of an inflammatory pathogenesis in hypertension.*

Keywords: *C-reactive protein, normotensive, hypertension.*

Introduction

Hypertension is one of the health concern because it is a major risk factor for a number of cardiovascular diseases including stroke, atherosclerosis, type II diabetes, coronary heart disease, and renal disease. It affects 26% of adults worldwide, and its prevalence is predicted to

increase to 29% by 2025^[1]. Data from more than 30 epidemiologic studies have shown a significant association between elevated serum concentrations of C-reactive protein (CRP) and the prevalence of underlying atherosclerosis, the incidence of first cardiovascular event in individuals at risk for atherosclerosis, and the risk

of recurrent cardiovascular events among patients with established diseases^[2]. In recent years, the role of the inflammatory process in the pathogenesis of hypertension has been suggested. Consequently, the relationship between CRP and Hypertension can be evaluated^[3]. In recent years, the term CRP has been used widely. One common misunderstanding has been the incorrect belief that CRP is different in some way from CRP. The fact is that CRP only denotes the utilization of an assay designed to measure very low levels of CRP, that is, the so-called low grade inflammation^[4]. Low grade and acute inflammation states differ from each other in several ways. For instance, the latter occurs in response to infection and tissue injury and the former is induced in response to metabolic stress^[5]. In one study, it was suggested that low-grade inflammation causes endothelial dysfunction and impaired nitric oxide availability, leading to an increased production of oxidative stress^[6]. Moreover, the relationship between this form of inflammation and obesity, a major risk factor of hypertension has been evaluated before^[7]. The aim of our work was to compare

the level of CRP in hypertensive and normotensive individuals.

Materials and Methods

The present study includes 500 type 2 diabetes mellitus patients with microalbuminuria coming to NIMS Medical College & Hospital, Jaipur were considered for the study. Based on blood pressure, they were divided into two groups. 250 normotensive patients were considered in Group A and 250 hypertensive patients were considered in group B.

Inclusion Criteria Cases

Diabetes mellitus, Age 26 to 65 years, Pressure $\geq 140/90$, over weight to Obese patient and for Control Healthy subjects, No diabetes, Age 26 to 65 years, Pressure $\leq 140/90$

Exclusion Criteria

- 1) Patients with infection.
- 2) Overt nephropathy.
- 3) Pre-existing kidney/ prostatic disease.
- 4) Congestive heart failure.
- 5) Pregnancy.
- 6) Receiving any hypolipidaemic drugs.

Results

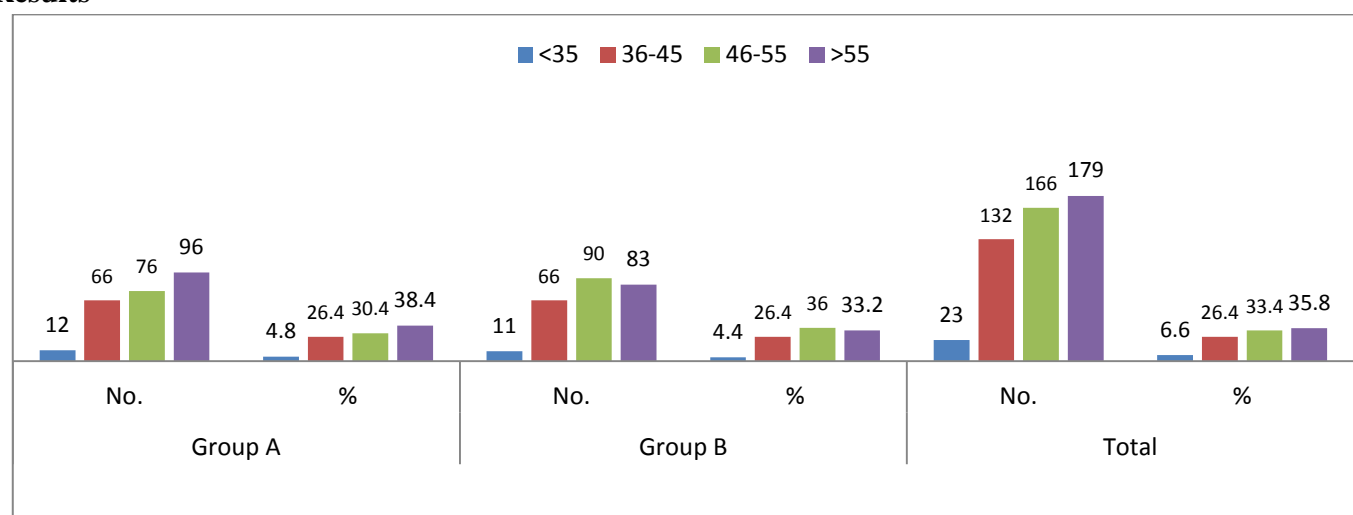


Fig:-1 Age wise distribution of patients

In this study the minimum age was 26 years old and maximum age was 65 years old. Out of total 500 patients, 58% were males while 42% were females. The Fig-1 shows that in normotensive diabetic group (Group A) patients were maximum

in age group >55 (38%) whereas in hypertensive diabetic group (Group B) maximum number of patients (36%) were in the age group of 46-55 years.

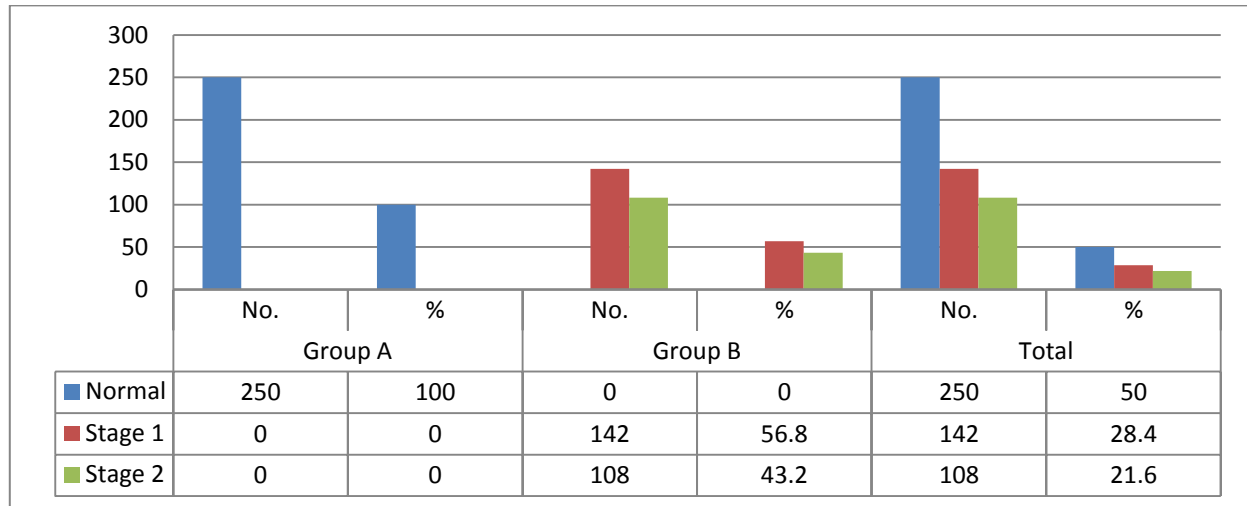


Fig:-2 Distribution of patients according to Hypertension

Fig-2 shows that in group A all patients had normal blood pressure while in group B 142 (56.8%) patients had stage 1 hypertension and 108 (43.2%) had stage 2 hypertension.

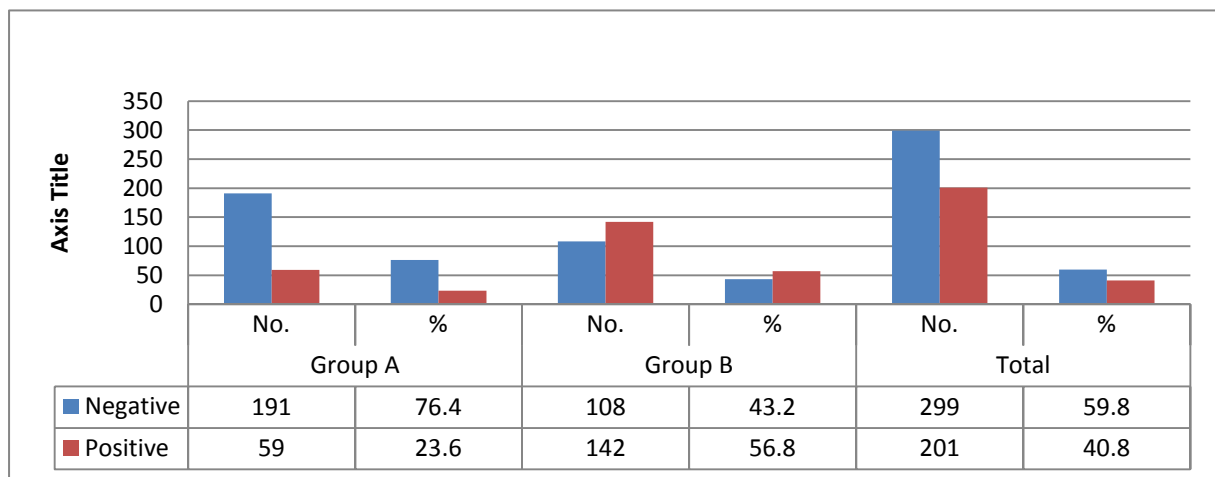


Fig:-3 Distribution of patients according to CRP

Fig-3 shows that in group A 59 (23.6%) patients had positive CRP value while in group B 142 (56.8%) patients had positive CRP values (>3mg/L). The difference was statistically highly significant with P<0.05

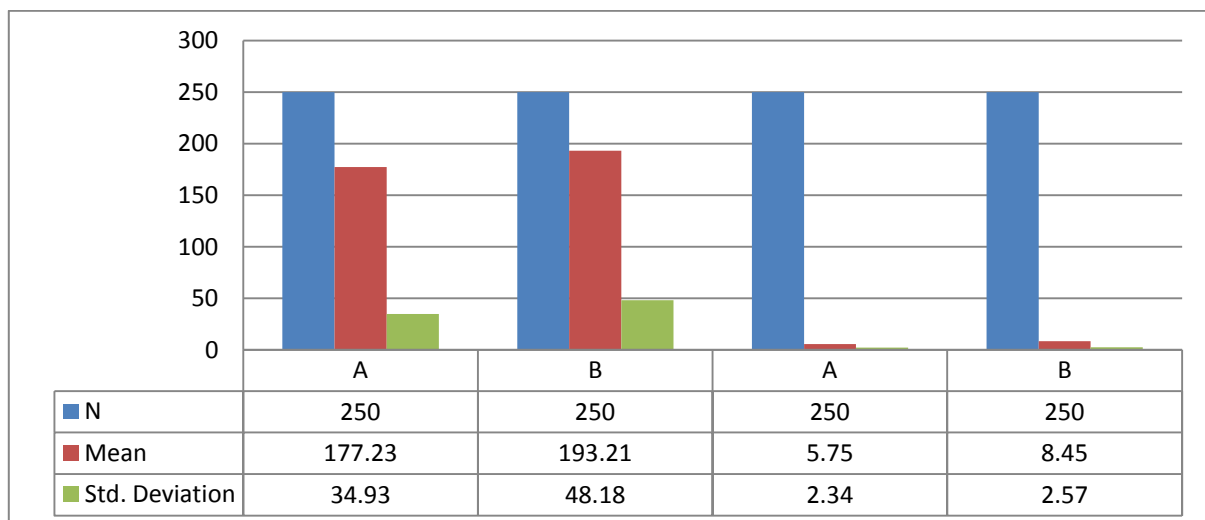


Fig-4 Comparison of CRP & UACR in two groups

Fig-4 shows that normotensive diabetic patients (group A) with a mean microalbuminuria and CRP value of 177.23 ± 34.93 & 5.75 ± 2.34 respectively as compared to hypertensive diabetic patients (group B) had higher levels of

Table-1 Distribution of UACR& CRP with blood pressure

		Group A		Group B	
		UACR	CRP	UACR	CRP
SBP	R	0.0028	0.003	0.158	0.06
	P Value	0.964	0.962	0.012	0.34
DBP	R	0.093	-0.08	0.11	0.05
	P Value	0.19	0.20	0.08	0.43

Table-1 shows that there is statistically significant correlation of systolic blood pressure with UACR and CRP in normotensive diabetics as compared to hypertensive diabetics. Whereas there is a positive correlation of UACR and CRP with diastolic blood pressure in hypertensive diabetics but it is not statistically significant.

Discussion

In group A all patients had normal blood pressure while in group B 142 (56.8%) patients had stage 1 hypertension and 108 (43.2%) had stage 2 hypertension. Group A with SBP & DBP of 125 ± 5.49 & 82.9 ± 4.59 respectively as compared to both SBP & DBP (mm Hg) were higher in group B i.e. 158 ± 7.05 & 90.1 ± 6.055 respectively. When group A & B compared statistically there was highly significant difference in the mean values of SBP & DBP ($p < 0.001$). There was no correlation of CRP with SBP in group A while when correlation coefficient (r) was applied there was statistically significant correlation of CRP with systolic blood pressure (SBP) ($P < 0.05$) ($r = 0.6$) in group B. Also there was no correlation of CRP with SBP in group A, while there was statistically significant correlation of UACR with systolic blood pressure ($P < 0.05$) ($r = 0.158$) in group B. On the other hand, there was no correlation found between CRP or UACR with diastolic blood pressure in both the groups.

The above mentioned data shows that systolic blood pressure is strongly correlated with CRP and UACR while diastolic blood pressure is not our finding is supported by Lakoski et al^[8] who

microalbuminuria and CRP with a mean value of 193.21 ± 48.18 & 8.45 ± 2.57 respectively. The difference was statistically highly significant with $p < 0.05$

conducted a study in 6814 men and women ages 45 to 84 years old recruited in six U.S. communities and they concluded that systolic BP and pulse pressure, but not diastolic pressure, were associated with CRP ($p = 0.0001, 0.0001$ & 0.5 respectively). Similar study done by schillaci et al^[9] in 135 newly diagnosed, never treated patients with hypertension and 40 healthy matched non-hypertensive controls concluded that among hypertensive patients, plasma CRP was related to 24-h systolic blood pressure ($r = 0.28$, $p < 0.01$) and pulse pressure ($r = 0.32$, $p < 0.01$), but not to diastolic blood pressure ($r = 0.12$, $p < 0.2$). Similar results were found in other studies done by stuveling et al^[10], Tsioufis et al^[11] and Nakamura et al^[12].

This study shows that the hypertensive diabetic patients (group B) had higher levels of microalbuminuria and CRP with a mean value of 193.21 ± 48.18 & 8.45 ± 2.57 respectively as compared to normotensive diabetic patients (group A) with a mean microalbuminuria and CRP value of 177.23 ± 34.93 & 5.75 ± 2.34 respectively. The difference was highly statistically significant with $p < 0.05$. We also found that there was a statistically significant positive correlation between CRP and UACR in hypertensive diabetic group (group B) and normotensive diabetic group (group A) but the correlation was stronger ($r = -0.713$; $p < 0.001$) in group B as compared to group A ($r = 0.664$; $p < 0.001$). This shows that hypertension positively effects urinary albumin excretion and CRP.

Stuveling et al^[10] had also shown that the association between microalbuminuria and CRP was more significant ($p < 0.001$) in subject with high mean arterial pressure. The concluded that BP positively modified the relationship between micrialbuminuria and CRP. Tsioufis et al^[10] had shown that microalbuminuria is accompanied by increase in CRP level in the setting of hypertension, reflecting a diffuse atherosclerotic process. So, the finding in the present study are consistent with the above mentioned study results.

Conclusion

In conclusion our results suggest that increased serum CRP levels associated with hypertension more significantly with type 2 diabetic case. Thus estimation of CRP can be a potential tool for individuals at the risk in development of hypertension and eventually cardiovascular disease in type 2 diabetes.

Acknowledgment

The authors are thankful to the management NIMS university, Jaipur, Rajasthan, India for allowing this doctoral study to carry out and for providing the facilities of needed. Also the authors are very much thankful to Dr. R.C. Gupta (Prof & Head), Dr. M. Gupta (Assoc. Prof.), Department of Biochemistry for their valuable suggestion during the study.

References

1. Monaster SH, Ahmed MK, Braik AG. Comparison between strain and strain rate in hypertensive patients with and without left ventricular hypertrophy: a speckle-tracking study. *Menoufia Med J* 2014;27:322-328.
2. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Canon RO, Criqui M, et al. Marker of inflammation and cardiovascular disease. *Circulation* 2003;107:499-511.
3. Ridker PM, Silvertown JD. Inflammation, C-reactive protein, and atherothrombosis. *J Periodontol* 2008;79(Suppl):1544-1551.
4. Kushner I, Salmpns D, Magray MA. Unifying biologic explanation for highsensitive C-reactive protein and low grade inflammation. *Arthritis Care Res (Hoboken)* 2010;62:442-446.
5. Bianchi ME. DAMPs, PAMPs and alarmins: all we need to know about danger. *J LeukocBiol* 2007;81:1-5.
6. Taddei S, Caraccio N, Viridis A, Dardano A, Versari D, Ghiadoni L. Low-grade systemic inflammation causes endothelial dysfunction in patients with Hashimotos thyroiditis. *J ClinEndocrinolMetab* 2006;91:5076-5082.
7. Maachi M, Pieroni L, Bruckert E, Jardel C, Fellahi S, Hainque B, et al. Systemic low-grade inflammation is related to both circulating and adipose tissue TNF, leptin and IL-6 levels in obese women. *Int J Obes Relat Metab Disorder* 2004;28:993-997.
8. Lakoski SG, Cushman M, Palmas W, Blumenthal R, D'Agostino J, Herrington DM. The Relationship Between Blood Pressure and C- Reactive Protein in the Multi-Ethnic Study of Atherosclerosis (MESA). *J Am CollCardiol.* 2005 Nov 15;46(10):1869-74.
9. Schillaci G, Pirro M, Gemelli F, Pasqualini L, Vaudo G, Marchesi S, et al. Increased C-reactive protein concentration in never-treated hypertension: the role of systolic and pulse pressures. *J Hypertens.* 2003 Oct;21(10):1841-6.
10. Stuveling EM, Bakker SJL, Hillege HL, Burgerhof JGM, de Jong PE, Gans ROB, et al. C-reactive protein modifies the relationship between blood pressure and microalbuminuria. *Hypertension.* 2004 Apr;43(4):791-6.
11. Tsioufis C, Dimitriadis K, Chatzis D, Vasiliadou C, Tousoulis D, Papademetriou V, et al. Relation of microalbuminuria to

adiponectin and augmented C-reactive protein levels in men with essential hypertension. *Am. J. Cardiol.* 2005 Oct 1;96(7):946-51.

12. Nakamura M, Onoda T, Itai K, Ohsawa M, Satou K, Sakai T, et al. Association between serum C-reactive protein levels and microalbuminuria: a population-based cross-sectional study in northern Iwate, Japan. *Intern. Med.* 2004 Oct;43(10):919-25.