

The Study of CRP as an Inflammatory Marker in Diabetes Mellitus with relation to the Level of Glycosylated Haemoglobin: A Case- Control Study

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ABSTRACT

Diabetes mellitus is a group of metabolic disorders characterized by hyperglycemia. According to several studies, it is noticed that diabetic patients have progressive inflammation with severity. Aim of our study to determine the correlation of CRP and HbA1c in patients with Type 2 Diabetes mellitus.

Materials and Method: This study was conducted through clinical and laboratory assessments at the Department of Biochemistry. Total 300 subjects are studied, 150 Type 2 Diabetic patients and 150 Non-diabetic subjects are studied in this study.

Results: In present study we found that patient having T2DM with high HbA1c and high levels of CRP when compared to the control group. CRP levels in our study were significantly higher in type 2 diabetes patients compared to the Non-diabetic group.

Introduction

Diabetes Mellitus (DM) are a group of metabolic disorders which has the phenotype of hyperglycemia. DM is a global endemic with rapidly increasing prevalence in both developing and developed countries (1). In addition to hyperglycemia, patients with diabetes may develop

macroangiopathy, microangiopathy and neuropathy. Diabetic nephropathy (DN) is one of the most common microvascular complications of diabetes mellitus; occurring in approximately 40–50% of patients with diabetes (2). WHO has declared India as “Diabetic Capital of the world”. The prevalence of both type 1 and type 2 DM are increased, type 2 DM is expected to rise more rapidly in future because of increased obesity and reduced activity levels. The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. Glycated hemoglobin (HbA1c) is routinely used as a diagnostic tool for measuring long term glycemic control. In accordance with its function as an indicator for the mean blood glucose level, HbA1c predicts the risk for the development of diabetic complication in diabetes patients. The chronic hyperglycemia of diabetes is associated with damage and failure of various organs, especially the eyes, kidneys, nerves, heart, and vascular system. Studies have shown that inflammatory states are positively correlated with the level of HbA1c in patients with or without diabetes (3,4). Inflammation attaches importance to the formation and progression of coronary artery atherosclerosis. HsCRP is an acute-phase reactive and nonspecific inflammatory marker that is mainly produced in hepatocytes (5). Studies have shown that hsCRP can detect coronary heart disease and predict future cardiovascular events (6,7). Among patients undergoing PCI, higher CRP levels during the procedure can predict 10-year mortality and myocardial infarction (MI) (8). However, no one has studied the effect of inflammation on the correlation between HbA1c and cardiovascular events. Among inflammatory biomarkers; C-reactive protein (CRP) has gained attention. CRP is an acute-phase protein synthesized by the liver in response to interleukin-6 and other pro-inflammatory cytokines.(9) Elevated CRP levels have been associated with systemic inflammation, cardiovascular disease, and, more recently, micro vascular complications of diabetes, including diabetic neuropathy, nephropathy, and retinopathy. While several studies suggest a positive association between CRP levels and the incidence or severity of DM, others have reported inconsistent or even inverse relationships, highlighting the need for further investigation.(10-12) This study aims to evaluate the association between CRP plasma levels in patients with T2DM to gain a clearer understanding of the role of inflammation and endothelial damage in the pathogenesis of DM. These contrasting results may arise because of ethnic and genetic differences in baseline CRP expression, differences in analytic methods, and variations in the assessment of DM. The aim of this study was to assess whether plasma CRP levels were independently associated with the presence and severity in T2DM patients. This would help

to clarify the role of systemic inflammation in the DM physiology and also guide to understand the evidence reported in the literature.

Materials and Methods

Study Design

This study was conducted through clinical and laboratory assessments at the Department of Biochemistry at Shri Rawatpua Sarkar Institute of Medical Sciences and Research, between August 2025 and December 2025. This study was approved by the institutional ethics committee and written informed consent was taken from all the participants.

Participants

The inclusion criteria were as follows: Diagnosed with T2DM, age \geq 18 years, and complete clinical data and laboratory test results (13). The exclusion criteria included the following: Secondary diabetes mellitus, primary nephrotic syndrome, glomerulonephritis and secondary nephrotic disease like systemic lupus erythematosus, as well as acute complications of diabetes mellitus, infection and severe heart and lung diseases. In this study, we recruited 150 patients having T2DM and Control (150) inclusive of both males and females who were above 30 years of age. About 5 mL venous blood was drawn from each participant and divided into two tubes: 2 mL was drawn into ethylene diamine tetra acetic acid (EDTA) tubes to measure the HbA1c and 3 mL was drawn into a plain tube with no anticoagulant to measure the biochemical parameter CRP. All the samples are run on Mispa-i2 for HbA1c and CRP.

Data Collection

General patient data were collected, including age, sex, height, weight, Laboratory data were collected, including: glycosylated hemoglobin (HbA1c) and CRP. HbA1c $>$ 6.5% were used as the diagnosis criteria for Diabetes mellitus.

Statistical Analysis

Variables are expressed as mean and standard deviation. The independent sample t-test was used for normally distributed data. Categorical variables are expressed as frequencies (percentage) and

the chi-square test was used for statistical analysis. Statistical significance was set at a P value of <0.05.

Observation and Results

A total of 150 Diabetes mellitus patients and 150 Normal Control were involved in this study. Among Diabetes mellitus, 56.66% were males and 43.33% were females; while 52.66% males and 47.33% were females in Normal Control. The mean age of Diabetes mellitus participants was 51 with 9.1 SD. The mean age of Normal Control is 43 with 8.4 SD. The BMI value for the Diabetes mellitus was 28.2 with 4.7 SD and for Normal control was 22.7 with 2.3 SD. The P-value is less than 0.05 for Age and BMI, which is statistically significant. All these data are available in Table 1.

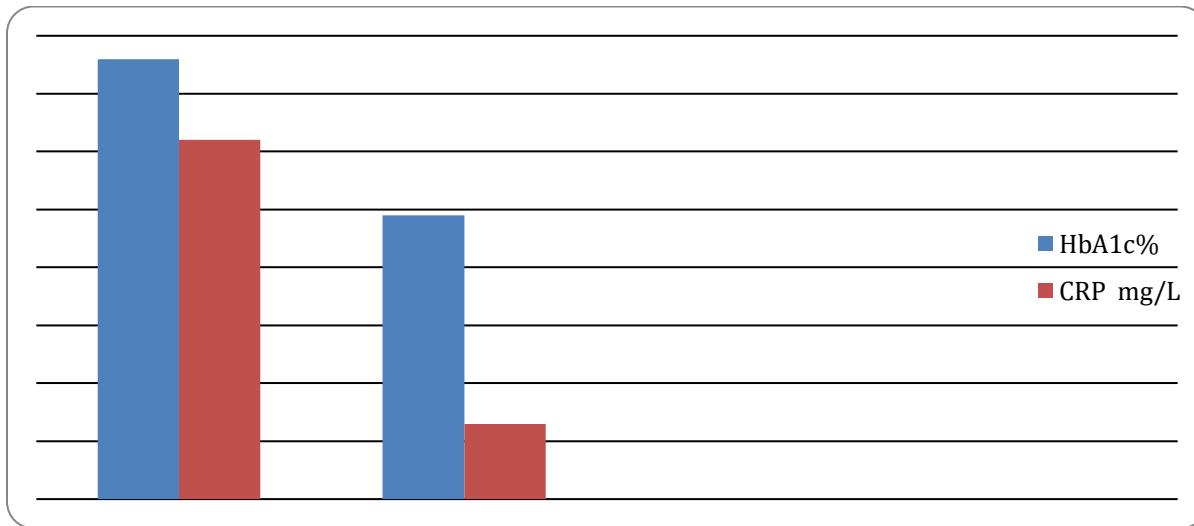
Table 1: Demographic details of the subjects

	T2DM(n=150)	Non-diabetic (n=150)	<i>P-value</i>
Age(years)	51±9.1	43±8.4	<0.05
Male (%)	85(56.66%)	79(52.66%)	---
Female (%)	65(43.33%)	71(47.33%)	---
BMI(kg/m ²)	28.2±4.7	22.7±2.3	<0.05

The CRP level in Diabetes mellitus was 6.2 mg/L and HbA1c 7.6% with SD 3.4 and 2.9 respectively. While CRP and HbA1c levels in Normal Control was 1.3 and 4.9 with SD 0.47 and 1.4 respectively. The P-value is less than 0.05 which is statistically significant. All these information are available in Table 2 and Graph.

Table 2 : Comparison of HbA1c and CRP

	T2DM(n=150)	Non-diabetic(n=150)	<i>P-value</i>
HbA1c(%)	7.6±2.9	4.9±1.4	<0.05
CRP(mg/L)	6.2±3.4	1.3±0.47	<0.05



Discussion:

The relationship of type 2 diabetes mellitus with inflammation has been studied since a few decades. Diabetes has been shown to be a risk factor for development of inflammation. Inflammation is a driving factor in the development of other secondary symptoms of DM. CRP is one of the important inflammatory markers and biomarker for predicting vascular events in several pathological conditions (14). In present study we found that patient having T2DM with high HbA1c and high levels of CRP when compared to the control group. CRP levels in our study were significantly higher in type 2 diabetes patients compared to the Non-diabetic group. Same type of results came out by King and others study (15). Our study showed that a rise in HbA1C levels significantly correlated with increasing values of CRP. Hu et al studied association of serum C-reactive protein level with sex-specific type 2 diabetes (16). Williams et al. showed that obesity was independently related to CRP, an increase in CRP is associated with an increase in BMI (17). In our study BMI is directly proportional to the severity of diabetes and inflammation, that is HbA1c and CRP proportionally increases. CRP may have certain significance in the occurrence, prognosis, and treatment assessment of T2DM patients. There are several biologically plausible pathways by which CRP could be associated with the neurodegeneration and capillary dropout seen in retinopathy. Systemically, CRP is synthesized by hepatocytes (and adipocytes) in response to interleukin-6, IL-1 β , and TNF- α (18). CRP can exert direct effects on endothelial cells and immune cells via Fc γ receptors (especially CD32) and complement activation. For example, CRP

has been shown to directly upregulate endothelial adhesion molecules such as ICAM-1, VCAM-1, and E-selectin. This upregulation facilitates leukocyte adherence to the retinal microvascular endothelium, leading to leukostasis and capillary occlusion. CRP also stimulates secretion of chemokines like MCP-1, further attracting inflammatory cells into the retinal tissue. Thus, elevated CRP can actively promote the low-grade endothelial dysfunction and leukocyte-mediated capillary damage (19). On a molecular signaling level, CRP binding to Fc γ R on endothelial and glial cells triggers intracellular pathways. However, CRP is a very non-specific marker of inflammation; it rises with infection, other vascular diseases, obesity, and metabolic syndrome. From a clinical perspective, CRP is potentially a low-cost and readily available biomarker for addressing the risk of inflammation in individuals with T2DM. In addition to CRP, other systemic or environmental stimuli that contribute to vascular inflammation may also play a role in DM pathogenesis. Endocrine-disrupting chemicals can cause mitochondrial dysfunction and exacerbate inflammatory damage in the microvascular complications of diabetes (20).

Conclusion:

Our findings reinforce that systemic inflammation, as reflected by CRP, is linked with more severe DM. This adds to a growing body of evidence that chronic inflammation contributes to the microvascular complications of diabetes. Considering inflammatory pathways alongside traditional risk factors may improve our understanding of DM pathogenesis. Future longitudinal and mechanistic studies, including prospective cohorts, genetic analyses, and interventional trials, are warranted to clarify whether CRP can serve as a useful biomarker and therapeutic target in diabetic disease. We concluded that the CRP may be a additional marker of better glycemic control and also correlates with the inflammation seen in type 2 diabetes mellitus.

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Conflicts of interest

There are no conflicts of interest

Use of AI-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript.

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