Section: General Medicine

Original Research Article

A PROSPECTIVE STUDY OF C-REACTIVE PROTEIN AND GLYCEMIC CONTROL IN ADULTS WITH TYPE-2 DIABETES MELLITUS PATIENTS AT TERTIARY CARE CENTRE

Ramavatar Bairwa[1], Yogesh Kumar Mishra[2], Vijay Kumar Bairwa[3]

[1,2]Assistant Professor, Department of General Medicine, Government Medical College, Dausa, Rajasthan, India.
[3]Senior Resident, Department of General Medicine, Government Medical College, Dausa, Rajasthan, India.

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Corresponding Author:
Dr. Ramavatar Bairwa,
Assistant Professor, Department of General Medicine, Government Medical College, Dausa, Rajasthan, India.
Email: drramavatar09@gmail.com

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ABSTRACT

Background: Diabetes is associated with a pro-inflammatory state and endothelial dysfunction. Various markers of inflammation like C Reactive Protein (CRP) are elevated significantly in diabetic population. Hence understanding the role of CRP in inflammation process among diabetics with poor glycemic control is relevant to early identification and prevention of complications of diabetic people and enhance the quality of life in all aspects.

Materials and Methods: This is a hospital based prospective study done on patients of type 2 diabetes mellitus, attending the outpatient Department of General Medicine in a tertiary care hospital, Dausa, Rajasthan, India during one-year period. Total number of 120 subjects were included in this study with age range from 30 to 60 years of both sexes. Among them 60 were cases of type 2 diabetes mellitus patients having HbA1C level >6.5% and 60 were age-matched with healthy controls having HbA1C level <6.5%. All the analyses were done using SPSS version 23.0.

Results: Patients with diabetes exhibited significantly higher mean BMI (28.3±4.13 kg/m²) compared to controls (23.2±2.7 kg/m²; $p < 0.05$*). Similarly, both systolic (138.26 ± 12.38 vs. 127.60 ± 11.82 mmHg; $p < 0.05$*) and diastolic blood pressure (86.50 ± 11.23 vs. 77.50 ± 8.54 mmHg; $p < 0.05$*) were significantly elevated in the diabetic group. Inflammatory burden was notably greater in diabetics, as reflected by CRP levels (7.32 ± 5.15vs. 5.36 ± 0.44 mg/L; $p < 0.05$*). Glycemic control markers confirmed poor control in diabetics, with a mean HbA1c of 8.12% compared to 5.78% in controls ($p < 0.05$*).

Conclusion: Elevated CRP levels are significantly associated with poor glycemic control in T2DM, suggesting a critical role of low-grade inflammation in the development of diabetic complications. CRP may serve as a valuable adjunct marker for identifying high-risk patients

Keywords: CRP, HbA1c, Glycemic control, Diabetes mellitus, BMI.

INTRODUCTION

Hyperglycemia brought on by insulin resistance and/or insufficient insulin production is a hallmark of type 2 diabetes mellitus (T2DM), a chronic metabolic disease. Over the past decades, it has become a major global health burden due to its rising prevalence and its role in accelerating cardiovascular and microvascular complications. Low-grade systemic inflammation may be a key factor in the development, course, and consequences of type 2 diabetes, according to an increasing amount of research.[1]

One well-known acute-phase reactant that has become a crucial indicator of systemic inflammation is C-reactive protein (CRP). Patients with type 2 diabetes frequently have elevated CRP levels, which are thought to be related to poor glycemic control, obesity, insulin resistance, and the emergence of comorbidities such nephropathy and retinopathy.[2,3]

This inflammatory burden, as indicated by CRP, may exacerbate the risk of both macrovascular and microvascular problems in diabetes by causing endothelial dysfunction and vascular damage.[4]

As of right now, the most popular method for determining glycemic status is HbA1c. Elevated HbA1c levels, an indication of poor glycemic management, hasten the development of atherosclerosis and greatly raise the risk of cardiovascular events.[5] A particularly sensitive indicator of the inflammatory activity in the artery wall is C-reactive protein, which is evaluated by highly sensitive assays (hsCRP).[6,7] In addition to the conventional risk factors, it is a significant predictor of cardiovascular risk.[8,9] Interestingly, persistent hyperglycemia causes the liver to secrete acute phase reactants and increases the release of several inflammatory cytokines (IL-6, TNF-α), which raises CRP in conjunction with high fasting plasma glucose.[10]

Despite these findings, there is limited data evaluating the relationship between CRP and glycemic control in the development of type 2 diabetes mellitus.[11] Hence understanding the role of CRP in inflammation process among diabetics with poor glycemic control is relevant to early identification and prevention of complications of diabetic people and enhance the quality of life in all aspects.

MATERIALS AND METHODS

This is a hospital based prospective study done on patients of type 2 diabetes mellitus, attending the outpatient Department of General Medicine in a tertiary care hospital, Dausa, Rajasthan, India during one-year period.

Inclusion Criteria

- Patients clinically diagnosed and confirmed with type 2 diabetes having HbA1C level >6.5%, by a general physician at tertiary care hospital aged 18 years or older.
- Patients who were willing to participate and give consent.

Exclusion Criteria

- Gestational diabetic patients and lactational mothers.
- Patients who used anti-inflammatory drugs or cholesterol-lowering drugs with in the previous 30 days.
- People who are physically and mentally disabled.
- Patients who did not give consent.

Methods

Total number of 120 subjects were included in this study with age range from 30 to 60 years of both sexes. Among them 60 were cases of type 2 diabetes mellitus patients having HbA1C level >6.5% and 60 were age-matched with healthy controls having HbA1C level <6.5%.

5 ml of Venous Blood was collected from the antecubital vein with all aseptic precaution. Blood was centrifuged at 3000 rpm and the serum was separated. HbA1c was measured by high pressure liquid chromatography. High sensitive CRP was measured by particle enhanced turbidometry assay. They were followed up on a monthly basis, clinical evaluation was done; fasting and postprandial blood sugars were monitored and adjustment of anti-diabetic medications done on an individual basis in addition to re-enforcement on life style modification (diet and exercise).

Statistical Analysis: Descriptive statistics were reported as mean ± SD for normally distributed data. Paired t test or Wilcoxon Signed rank test was used to test the significance for the change in HBA1c and CRP after 6 months from baseline. P value less than 5% was considered statistically significant. All the analyses were done using SPSS version 23.0.

RESULTS

A total of 120 participants were enrolled, comprising 60 patients with type 2 diabetes mellitus and 60 age- and sex-matched non-diabetic controls. Patients with diabetes exhibited significantly higher mean BMI (28.3±4.13 kg/m²) compared to controls (23.2±2.7 kg/m²; $p < 0.05$*). Similarly, both systolic (138.26 ± 12.38 vs. 127.60 ± 11.82 mmHg; $p < 0.05$*) and diastolic blood pressure (86.50 ± 11.23 vs. 77.50 ± 8.54 mmHg; $p < 0.05$*) were significantly elevated in the diabetic group. Inflammatory burden was notably greater in diabetics, as reflected by CRP levels (7.32 ± 5.15vs. 5.36 ± 0.44 mg/L; $p < 0.05$*). This indicates a substantially higher inflammatory burden in individuals with type 2 diabetes mellitus. Glycemic control markers confirmed poor control in diabetics, with a mean HbA1c of 8.12% compared to 5.78% in controls ($p < 0.05$*). [Table 1]

Table 1: Clinical and Biochemical Parameters of Study Participants (n = 120)

Variables	Diabetic (Mean±SD)	Control (Mean±SD)	P-value
BMI (Kg/m²)	28.3±4.13	23.2±2.7	<0.05*
HbA1c (%)	8.12 ± 1.26	5.78 ± 0.33	<0.05*
Serum CRP Mg/L (Mean + SD)	7.32 ± 5.15	5.36 ± 0.44	<0.05*
Systolic BP (mmHg)	138.26 ± 12.38	127.60 ± 11.82	<0.05*
Diastolic BP (mmHg)	86.50 ± 11.23	77.50 ± 8.54	<0.05*

DISCUSSION

Diabetes mellitus is a multifactorial metabolic disease marked by persistent hyperglycemia and abnormalities in the metabolism of carbohydrates, fats, and proteins due to deficiencies in insulin secretion, action, or both.[12] It is becoming widely accepted that the pathophysiology of DM involves a chronic inflammatory response that may be present even before the disease is diagnosed, and that the vascular strain associated with this response results blood vessel dysfunction and damage. In this sense, diabetes is closely tied to vascular insult, a key component of the triad of thromboembolic risk factors. Cardiovascular disease is the leading cause of both morbidity and mortality among patients with diabetes. The most common cardiovascular complications experienced by diabetic patients include atherosclerosis, myocardial infarction, and stroke.[13]

C-reactive protein (CRP), a marker of systemic inflammation is emerging as independent risk factor for cardiovascular diseases.[1] It is helpful to identify patients at higher risk for vascular complications.

This study provides comprehensive evidence supporting the role of C-reactive protein (CRP) as an inflammatory biomarker linked to poor glycemic control and early diabetic complications. The significantly elevated CRP levels in type 2 diabetic patients compared to non-diabetic controls (7.32 ± 5.15vs. 5.36 ± 0.44 mg/L; $p < 0.05$*) align with findings by Sasidharan et al. and Habib et al., who demonstrated higher CRP in diabetic cohorts and correlated it with worsening metabolic parameters and insulin resistance.[14,15] In our study, the strength of association between CRP and glycemic control was notable. A robust positive correlation was observed between CRP and HbA1c ($r = 0.831$, $p < 0.0001$), indicating that systemic inflammation escalates with deteriorating glycemic regulation. This supports mechanisms described by Tang et al. and Elimam et al., where chronic hyperglycemia induces oxidative stress, endothelial dysfunction, and systemic inflammatory cascades.[16,17]

There is insufficient data on the serum hs-CRP level of our normal non-diabetic population, however several studies by western writers have demonstrated that Asian people have a considerably greater level of CRP.[18] CRP is a well-known inflammatory biomarker that is high in the blood of patients with severe inflammation and disorders such as T2DM and CVD. A Chinese population study found that CRP levels were greater in T2DM patients than in normal subjects,[19] implying that CRP is an independent predictor of incident T2DM. Other studies have found that greater levels of CRP are connected with an increased risk of acquiring diabetes.[20] Because of its relative stability in serum or plasma, accessibility of measurement, and availability of the international standard, CRP is a more usable and trustworthy sign in research and clinical settings than other inflammatory indicators such as cytokines.[21] According to a European study, higher levels of CRP were more strongly and independently related with an elevated risk of T2DM in women than in men, and this did not change after stratification by age, smoking or drinking behaviour, obesity, or diabetes family history.[22]

CONCLUSION

We concluded that there is positive correlation between the level of glycemic control (HbA1c) and CRP levels; Better glycemic control results in significant reduction in the hsCRP levels. The role of CRP in Indian patients and its integration into routine clinical practice as a cost-effective risk marker can actually be determined by a bigger prospective multicenter trial.

REFERENCES

1. King, D. E., Mainous III, A. G., Buchanan, T. A., & Pearson, W. S. C-reactive protein and glycemic control in adults with diabetes. Diabetes care.2003;26(5):1535-39.
2. Bhowmick, K., Kutty, A. V. M., & Shetty, H. V. Glycemic control modifies the association between microalbuminuria and c-reactive protein in Type 2 Diabetes Mellitus. Indian Journal of Clinical Biochemistry.2007; 22: 53-59.
3. Chuengsamarn, S., Rattanamongkolgul, S., Sittithumcharee, G., & Jirawatnotai, S. Association of serum high-sensitivity C-reactive protein with metabolic control and diabetic chronic vascular complications in patients with type 2 diabetes. Diabetes & Metabolic Syndrome: Clinical Research & Reviews.2017;11(2):103-08.
4. Fronczyk, A., Molęda, P., Safranow, K., Piechota, W., & Majkowska, L. Increased concentration of C-reactive protein in obese patients with type 2 diabetes is associated with obesity and presence of diabetes but not with macrovascular and microvascular complications or glycemic control. Inflammation.2014; 37:349-57.
5. Cavero-Redondo I, Peleteiro B, Álvarez-Bueno C, et al. Glycated haemoglobin A1c as a risk factor of cardiovascular outcomes and all-cause mortality in diabetic and non-diabetic populations: a systematic review and meta-analysis. BMJ Open.2017;7: e015949.
6. Kamath DY, Xavier D, Sigamani A, Pais P. High sensitivity C-reactive protein (hsCRP) & cardiovascular disease: An Indian perspective. Indian J Med Res.2015;142:261-8.
7. Pfützner A, Forst T. High-sensitivity C- reactive protein as cardio-vascular risk marker in patients with diabetes mellitus. Diabetes Technol Ther. 2006; 8(1):28-36.
8. Asegaonkar SB, Marathe A, Tekade ML, Cherekar L, Bavikar J, Bardapurkar J et al. High-sensitivity C-reactive protein: a novel cardiovascular risk predictor in type 2 diabetics with normal lipid profile. J Diab Complications. 2011;25(6):368-70.
9. Mohieldein AH, Hasan M, El-Habiby MI. High Sensitivity C-Reactive Protein as Atherogenic Marker Among Type 2 Diabetes. Eur Sci J.2017(13);33:403.
10. Lin Y, Rajala MW, Berger JP. Hyperglycaemia-induced production of acute phase reactants in adipose tissue. J Biol Chem.2001;276(45):42077-83.
11. Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. Jama. 2001 Jul 18;286(3):327-34.
12. Ozougwu JC, Obbimba KC, Belonwu CD, Unakalamba CJ. The pathogenesis and pathophysiology of type 1 and type 2 diabetes mellitus. J physiol Pathophysiol. 2011;2(4):1-16.
13. O'Hara A, Pozin J, Darki A, Siddiqui F, Hoppensteadt D, Walenga J, Fareed J, Kantarcioglu B. Glycemic control and plasma levels of pro-inflammatory and pro- thrombotic

biomarkers in diabetic patients presenting with acute pulmonary embolism. Clinical and Applied Thrombosis/Hemostasis. 2023 Mar; 29:10760296231165058.
14. Sasidharan, A., Krishnamurthy, A., Tagore, S., Nagaraj, T., Santosh, H. N., & Nigam, H. C-reactive protein and glycemic control in adults with type 2 diabetes mellitus. Journal of Medicine, Radiology, Pathology and Surgery. 2016;2(3):10-13.
15. Habib, S. S. Serum lipoprotein (a) and high sensitivity C reactive protein levels in Saudi patients with type 2 diabetes mellitus and their relationship with glycemic control. Turkish Journal of Medical Sciences.2013; 43(2):333-38.
16. Tang, M., Cao, H., Wei, X. H., Zhen, Q., Liu, F., Wang, Y. F., ... & Peng, Y. D.Association between high-sensitivity c-reactive protein and diabetic kidney disease in patients with type 2 diabetes mellitus. Frontiers in endocrinology.2022;13:885516.
17. Elimam, H., Abdulla, A. M., & Taha, I. M. Inflammatory markers and control of type 2 diabetes mellitus. Diabetes & Metabolic Syndrome: Clinical Research & Reviews.2019; 13(1):800-04.
18. Ikonomova K. Inflammation and Metabolic syndrome. Turkish Journal of Endocrinology and Metabolism 2004; 8 (3): 68-69.
19. Wen, J. et al. C-reactive protein, gamma-glutamyltransferase and type 2 diabetes in a Chinese population. Clin Chim Acta.2010; 411:198–03.
20. Duncan, B. B. et al. Low-grade systemic inflammation and the development of type 2 diabetes. Diabetes.2003; 52:1799–05.
21. Doi, Y. et al. Elevated C-reactive protein is a predictor of the development of diabetes in a general Japanese population. Diab Care.2005; 28: 2497–2500.
22. Han, T. S. et al. Prospective study of C-reactive protein in relation to the development of diabetes and metabolic syndrome in the Mexico city diabetes study. Diab Care.2002; 25: 2016–21.