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Original Research Article

Assessment of the Association of Fasting & PP C- Peptide with Hba1c in Type-2 Diabetes Mellitus in Population

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Conflict of interest: Nil

Abstract

Aim: To provide correlation of Fasting & PP C-peptide with HbA1C in patients of T2 Diabetes Mellitus.

Material & Methods: 60 patients admitted in Department of General Medicine, Patna Medical College, Patna, Bihar, India. The study was conducted over a period of 15 months.

Results: Mean & SD for fasting C-Peptide for males was1.490 ± 1.182& for females 2.371 ± 2.389. Raised Mean & SD for fasting for males was 3.482 ± 1.789 & for females were 0.820 ± 0.552. Raised Mean & SD for HbA1c for males was 10.722 ± 2.30 & for females 5.792 ± 0.281. Both were statistically significant with a p value of ≤0.05.

Conclusion: Insulin secretion estimated by measurement of Fasting C-Peptide was either normal or raised in newly diagnosed T2DM subjects in my study indicating predominant role of insulin resistance in the etiology. Further research can explore the exact contribution of insulin resistance and insulin secretory defects in this area.

Keywords: PP C- peptide, Diabetes mellitus**,** insulin resistance

Introduction

Glycemic control is the most important aspect in management of diabetes mellitus. It is a cornerstone in reducing morbidity and mortality of the diseases [1, 2]. The chronic hyperglycemia of diabetes is associated with long term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, nerves, heart, and blood vessels [3]. Many large randomized clinical trials and observational studies in type 1 and 2 diabetes have clearly shown that achieving glycemic control or reducing hyperglycemia significantly decrease the microvascular and macrovascular complications of diabetes mellitus (DM) [4–6].

C-peptide is a useful and widely used method of assessing pancreatic beta cell function [7-8]. After cleavage of proinsulin, insulin and the31-amino-acid peptide c-peptide are produced in equal amounts [9-10]. So why is c-peptide testing preferable to insulin as a guide to beta cell function? The degradation rate of c-peptide in the body is slower than that of insulin (half-life of 20–30 min, compared

with the half-life of insulin of just 3–5 min), which affords a more stable test window of fluctuating beta cell response. In healthy individuals the plasma concentration of c-peptide in the fasting state is 0.3–0.6 nmol/l, with a postprandial increase to 1–3 nmol/l [10].

Hb undergoes non enzymatic glycosylation in persons with persistent hyperglycemia and designated as HbA1c. HbA1C represents the integrated values of glucose over preceding 6-8 weeks and provides an additional criterion for assessing glucose control.

Our aim was to study correlation of Fasting c-peptide &Postprandial C-peptide with T2 Diabetes Mellitus in patients coming to SMIH for evaluation and follow up for treatment of Type 2 Diabetes Mellitus.

Material & Methods:

60 patients admitted in Department of General Medicine, Patna Medical College, Patna, Bihar, India. The study was conducted over a period of 15 months.

Methodology

Serum samples taken for fasting & PP C-peptide and HbA1C for patients of T2 Diabetes Mellitus and run on VITROS 5600/7600 which is based on dry chemistry.

Results:

We took 60 patients who were T2DM then we did fasting C peptide & PP C-peptide and HbA1c.

Out of 60, 10 were females & 50 were males. Out of 60, 54 patients had raised HbA1C maximum around 8-10.

Mean & SD for fasting C-Peptide for males was1.490 ± 1.182& for females 2.371 ± 2.389. [Table 1] Mean & SD forHbA1c for males was 9.554 ± 3.281& for females were 10.563 ± 2.583. [Table 2] Raised Mean & SD for fasting for males was 3.482 ± 1.789 & for females was 0.820 ± 0.552. Raised Mean & SD forHbA1cfor males was10.722 ± 2.30& for females 5.792 ± 0.281. Both were statistically significant with a p value of ≤0.05. [Table 3].

Table 1: Mean values fasting and post-prandial values of C-peptide according to gender

Parameter	Male	Female			
	Mean±SD	Mean±SD	T value	P value	Significant
Fasting	1.490 ± 1.182	2.371 ± 2.389	2.228	0.0138	S(P≤0.05)
PP	4.772 ± 5.011	2.011 ± 2.163	0.789	0.4381	NS(P≥0.05)

Table 2: Mean values of HbA1c according to gender

Parameter	Male	Female			
	Mean±SD	Mean±SD	T value	P value	Significant
HbA1c	9.554 ± 3.281	10.563 ± 2.583	0.7948	0.4306	NS(P≥0.05)

Table 3: Mean values fasting C-peptide and HbA1c values

Parameter	Raised	Un raised			
	Mean±SD	Mean±SD	T value	P value	Significant
Fasting	3.482 ± 1.789	0.820 ± 0.552	7.992	0.001	S (P≤0.05)
HbA1c	10.722 ± 2.30	5.792 ± 0.281	2.639	0.001	S (P≤0.05)

Discussion:

C-peptide is the part of proinsulin which is cleaved prior to co-secretion with insulin from pancreatic beta cells. Produced in equimolar amounts to endogenous insulin, it is not a product of therapeutically administered exogenous insulin and has

been widely used as a measure of insulin secretion. This review of the literature will identify the main indications and rationale for c-peptide sampling in clinical practice and compare the available methods of c-peptide testing. [11]

C-peptide sampling as part of the OGTT has been found to significantly correlate with insulin secretion in type 2 diabetes mellitus (T2DM), when samples are taken at 0, 30, 60, 90, and 120 min (with the possibility of extending this to include sampling at 150, 180, 240, and 300 min) [12].

In patients with diabetes who were both insulin and non-insulin treated, GST demonstrated a 29% rise in c-peptide compared to 19% rise postprandially [13].

Growing body of evidence have also shown a strong association between PPG and cardiovascular risk and outcomes [14], oxidative stress, carotid intimal thickness and endothelial dysfunction [15]. A recent diabetes complications trial study concluded that PPG, but not FPG, was an independent predictor of mortality and cardiovascular complications in diabetes [14,16-17]. It is also plausible that humans spend half of their lives in postprandial states and thus, to achieve better long-term metabolic control (HbA1c) and minimize the risk of chronic diabetic complications, glucose monitoring in postprandial state will be indispensable.

There are different methods to measure B cell secretory function. Acute insulin response (AIR) or AIR max is the gold standard for assessment of B cell function but difficult to perform in clinical setting. [18] Assay of serum insulin as a measure of insulin has half-life 3-5 minutes and almost half of insulin secreted to pancreas is degraded by hepatic first pass metabolism. C-Peptide secreted in the equimolar amount of insulin has negligible extraction by the liver and constant peripheral clearance making half-life longer than insulin. For this reason, it is commonly used in preference to insulin measurement when assessing B cell function in clinical practice. [19]

Whilst c-peptide levels may be associated with complications in diabetes through a glycemic mechanism it may also have direct molecular effects. C-peptide is generally thought of as a biologically inert peptide of interest only as a surrogate marker for insulin levels, but this may be overly simplistic. C-peptide has been shown in vitro to inhibit endothelial cell reactive oxygen species (ROS) formation in the presence of hyperglycemia [20-22]. C-peptide also down regulates the expression of several hyperglycemia-induced adhesion molecules, including vascular cellular adhesion molecule 1 (VCAM1), reducing leukocyte adhesion to endothelial cell walls and preventing the early stages of atherosclerosis plaque formation [7].

Conclusion:

Insulin secretion estimated by measurement of Fasting C-Peptide was either normal or raised in newly diagnosed T2DM subjects in my study indicating predominant role of insulin resistance in the etiology. Further research can explore the exact contribution of insulin resistance and insulin secretory defects in this area.

References:

1. American Diabetes Association. Standards of medical care in diabetes. Diabetes Care. 2014;37: S14–80.
2. Sarah JB, Soma SN, Margaret J, Jeremy M, Shiva S, Charles M. Adequacy of glycemic, lipid, and blood pressure management for patients with diabetes in a managed care setting. Diabetes Care. 2004;27 :694–8.
3. VinodMahato R, Gyawali P, Raut PP, Regmi P, Singh KP, Raj Pandeya DP, et al. Association between glycaemic control and serum lipid profile in type 2 diabetic patients: Glycatedhaem-oglobin as a dual biomarker.

Biomedical Researchm. 2011;22(3): 375–80.
4. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet. 1998; 352:837–53.
5. The Diabetes Control and Complications Trial Research Group. The relationship of glycemic exposure (HbA1c) to the risk of development and progression of retinopathy in the Diabetes Control and Complications Trial. Diabetes. 1995; 44:968–83.
6. Swetha NK. Comparison of fasting blood glucose & post prandial blood glucose with HbA1c in assessing the glycemic control. International J of Healthcare and Biomedical Research. 2014;2(3):134–9.
7. Jones AG, Hattersley AT. The clinical utility of C-peptide measurement in the care of patients with diabetes. Diabet Med. 2013; 30:803–17.
8. Kulkarni CM, Patil S. Urinary C-peptide and urine C-peptide/creatinine ratio (UCPCR) are possible predictors of endogenous insulin secretion in T2DM subjects—a randomized study. Int J Pharma Bio Sci. 2016; 7:443–446.
9. Steiner DF, Cunningham D, Spigelman L, Aten B. Insulin biosynthesis: evidence for a precursor. Science. 1967; 157:697–700.
10. Yosten GLC, Maric-Bilkan C, Luppi P, Wahren J. Physiological effects and therapeutic potential of proinsulin C-peptide. Am J PhysiolEndocrinol Metab. 2014;307:E955–68.
11. Emma Leighton. Christopher AR Sainsbury. Gregory C. Jones. A Practical Review of C-Peptide Testing in Diabetes. Diabetes Ther. 2017: 8:475–487
12. Okuno Y, Komada H, Sakaguchi K, et al. Postprandial serum C-peptide to plasma glucose concentration ratio correlates with oral glucose tolerance testand glucose clamp-based disposition indexes.Metabolism. 2013; 62:1470–6.
13. Koskinen PJ, Viikari JS, Irjala KM. Glucagon-stimulated and postprandial plasma C-peptide values as measures of insulin secretory capacity. Diabetes Care. 1988; 11:318–22.
14. Haffner S. The importance of postprandial hyperglycaemia in development of cardiovascular disease in people with diabetes. Int J ClinPract. 2001;123(suppl):24–6.
15. International Diabetes Federation, 2011 Guideline for Management of PostMeal Glucose in Diabetes. 2011; 1–37.
16. Hanefeld M, Cagatay M, Petrowitsch T, Neuser D, Petzinna D, Rupp M. Acarbose reduces the risk for myocardial infarction in type 2 diabetic patients: meta-analysis of seven long-term studies. Eur Heart J. 2004;25 (1):10–6.
17. Kovatchev B, Cox DJ, Summers KH, Gonder-Frederick L, Clarke WL. Postprandial glucose dynamics and associated symptoms in type 2 diabetes mellitus. The Journal of Applied Research. 2003;3(4):449–58
18. Cobelli C, Toffolo GM, Man CD, Campioni M, Denti P, Caumo E. Assessment of B cell function in humans, simultaneously with insulin sensitivity and hepatic extraction from intravenous and oral glucose tests. Am J PhysiolEndocrinolMetab. 2007;293 (1):E1–15.
19. Leighton E, Sainsbury CAE, Jones GC. A Practical Review pfCPeptide Testing in Diabetes. Diabetes Ther. 2017;8(3):475–87.
20. Bunyan, W. S., Saadoon, A. A., Saeed, M. N., Flayh, Z. J., & Alrazzaq, H. A. abd. Prevalence and Determinants of Retinopathy among Patients with Diabetes Mellitus: Thiqar 2022. Clinical Medicine Insights, 2022:3(2), 304–311.

21. Cifarelli V, Geng X, Styche A, Lakomy R, Trucco M, Luppi P. C-peptide reduces high-glucose-induced apoptosis of endothelial cells and decreases NAD(P)H-oxidase reactive oxygen species generation in human aortic endothelial cells. Diabetologia. 2011; 54:2702–12.
22. Bhatt MP, Lim Y-C, Hwang J, Na S, Kim Y-M, Ha K-S. C-peptide prevents hyperglycemia-induced endothelial apoptosis through inhibition of reactive oxygen species-mediated transglutaminase 2 activations. Diabetes. 2013; 62:243–53.