Short Communication Article

Effect of Satiety over Blood Glucose levels : A Prospective Study

Sabitha K[1], Rajkumar[2], B Venugopal[3], Rafi[4]

[1, 2, 3] Asst. Professor
[4] Professor
Department of Biochemistry
Chalmeda Anand Rao
Institute of Medical Sciences
Karimnagar - 505 001.
Telangana State, India.

CORRESPONDENCE :

Sabitha K
Asst.Professor
Department of Biochemistry
Chalmeda Anand Rao
Institute of Medical Sciences
Karimnagar - 505 001.
Telangana State, India.
E-mail : tejaswani19@gmail.com

ABSTRACT

Aim: Fasting blood glucose reflects pancreatic islet ß- cell function and stands for secretory function of basic insulin and measurement of fasting blood sugar(FBS) is used to diagnose diabetes as it has low positive results. But high levels of postprandial blood sugar(PPBS) alone is the risk factor for mortality in cardio vascular disease(CVD). The present study aims to know the differences in the post prandial blood sample given after having breakfast and after having lunch.

Materials and Methods: 125 samples are collected from normal healthy individuals working in Chalmeda AnadaRao Institute of Medical Sciences (CAIMS) hospital. The samples are collected in fasting, postprandial after breakfast and postprandial after lunch and is analysed for blood glucose levels in semi auto analyzer by kinetic method.

Results and Conclusion: The mean and SD values of postprandial sample after having breakfast and after lunch are 100±5.93, $p<0.01$(significant). The Post prandial sample collected after having lunch is more appropriate as it gives satiety to stomach and insulin secretion will be according to the contents than postprandial collected after breakfast.

Keywords: Postprandial blood glucose, insulin, satiety

INTRODUCTION

The prevalence of impaired glucose tolerance and type 2 diabetes is increasing [1] and it is estimated that the number of individuals with the disease will double from year 2000 to 2030 [2]. Both high fasting glycemia and high post prandial glycemia increase the risk of morbidity and mortality [3,4]. Some studies suggest that post prandial glycemia may be the most important of the two [5,6]. Fasting blood sugar (FBS) reflects islet ß – cell function and stands for secretory function of basic insulin. The measurement of FBS is used to diagnose diabetes as it has low positive results. Compared to FBS post prandial blood sugar (PPBS) is more prone to false positive results hence FBS is more preferred in diagnosis of diabetes. But high PPBS alone is the risk factor for mortality in Cardiovascular disease (CVD). If PPBS is under control, better protection will be given to structure and function of vascular endothelial cells and thus can reduce CVD [7]. The present study aims to know the differences in the post prandial blood sample given after having breakfast and after having lunch.

MATERIALS AND METHODS

The present study is conducted on 125 healthy individuals of age group between 20 – 35 years at Chalmeda Anada Rao Institute of Medical Sciences, Karimnagar, Telangana, South India. The blood samples were collected from the subjects in fasting condition, then after breakfast and also after having lunch. 5ml of blood sample is collected into a test tube coated with sodium fluoride, allowed to clot for 30 minutes and then centrifuged at 3000 rpm the supernatant is collected for the estimation of blood sugar. The study was approved by Institutional ethical committee (IEC) and an informed and written consent was obtained from all subjects. The demographic details are given in Table1.

Table1: Demographic details of the samples

	Samples
Age	20 – 35 years
Diet	Mixed diet
H/O of DM in family	25
H/O of Hypertension in family	25
Habit of smoking	13
Habit of drinking	Most of them are occasional drinkers
Type of work	Moderate

The blood sugar is estimated by Nicholas 5010 semi auto analyzer by Glucose-Oxidase Peroxidase (GOD-POD) method.

STATISTICAL ANALYSIS

The statistical analysis is done by using paired t test.

RESULT

In the present study theMean ± SD values of PPBS after having breakfast are 97.95±16.88 and after having lunch are 93.45±15.36 . The mean ±SD values after comparison is 100± 5.93 with p<0.01(significant).

	PPBS Breakfast	PPBS After Lunch	(PPBS Breakfast) Vs (PPBS After Lunch)
Mean ± SD	97.95±16.88	93.45±15.36	100 ± 5.93
P value	-	-	<0.01

DISCUSSION

In normal healthy individual, an acute increase of glycemia is capable of electrophysiological changes, which is evident with prolonged QT curve [8,9,10]. The increase in post prandial blood glucose is an important factor in the development of atherosclerosis [11]. The post prandial hyperglycemia(PPH), increases LDL oxidation, reduces the carotid – intima meida thickness thus leading to atherosclerosis and coronary Heart Diseases(CHD) [12,13,14]. In diabetics, PPH causes an increased production of plasma IL [6 ,18] & tumor necrosis factor – a (TNF a), overproduces thrombin, decreases flow mediated vasodilation [15,16,17]. The PPH and post hypertrigly ceredimea causes endothelial dysfunction which is mediated by oxidative stress also. Thus, PPH amplifies the atherogenic lipid profile and is a true risk factor for CHD [18, 19, 20, 21]. The post prandial blood glucose levels are affected by many factors like first phase insulin secretion, glucagon secretion, muscle tissue, liver tissue and fat tissue sensitivity to insulin, blood sugar levels before a meal, food and time to have meals, digestion function and absorption functions [22].

In a study by Matthew Riddle et al[23] they specifically mentioned about the timings of glucose measurements irrespective of diabetic or suspected diabetics or taking anti hyperglycemic drugs or not. The timings of glucose measurement influences especially basal hyperglycemia or post prandial hyper glycemia. In this present study we found that post prandial blood glucose levels given after taking breakfast is slightly towards higher side than the post prandial sample given after lunch however, all the values are within normal limits (except 5).

Similarly, in Paris a study is conducted to assess risk factors in a large male population, they concluded that 2 hour post load insulin level was a major independent predictor of coronary heart disease death, whereas impaired glucose tolerance was not [24]. Protein and fat content of food has minimal effects in raising blood glucose [25].

The relationship between insulin, blood glucose levels and satiety are unclear. There are findings which states that any effects on insulin and glucose were related to the lunch meal but not the preceeding meals i.e., breakfast [26]. There are reports which states no relationship between insulin, blood glucose levels and satiety [27] and also there are studies which concludes a significant inverse relationships between satiety and glucose and insulin responses [28, 29].

CONCLUSION

The present study has revealed the importance of collection of blood sample for assessment of blood glucose levels and has indicated that a post prandial sample taken after lunch (satiety) rather than after taking breakfast will be more significant and better indicator of blood glucose levels.

This study emphasizes the importance of post prandial sample to be drawn after having a meal which gives satiety and the sample to be given exactly after 2 hours of time, and then the blood sugar measurement will be according to the levels of insulin secretion.

CONFLICT OF INTEREST
The authors declared no conflict of interest.

FUNDING: None

REFERENCES

1. Dunstan DW, Zimet PZ, Welborn TA, et al. The rising prevalence of diabetes and impaired glucose tolerance – the Australian diabetes, obesity and life style study. *Diabetes Care.* 2002 ;25(5): 829-34.

2. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes – estimates for the year 2000 and projections for 2030. *Diabetes care.* 2004; 27(5):1047-53.

3. DECODE study group. Is the current definition for diabetes relevant to mortality risk from alls causes and cardiovascular and non cardio vascular diseases? *Diabetes care.* 2003; 26(3): 688-96.

4. Levitan EB, Song Y, Ford ES, Liu S. Is non diabetic hyperglycemia a risk factor for cardiovascular disease? *Arch Intern Med.* 2004; 164:2147- 55.

5. Shaw JE, Hodge AM, de Coueten M, chitson P, Zinmet PZ. Isolated post challenge hyperglycemia confirmed as a risk factor for mortality. *Diabetologia.* 1999;42:1050-4.

6. Temelkova – Kurktschiev TS, Koehler C, Henkel E, Leonhardt W, Fuecker K, Hanefeld M. Post challenge plasma glucose and glycemic spikes are more strongly associated with atherosclerosis than fasting glucose or HbA1C level. *Diabetes Care.* 2000; 23(12):1830-4.

7. Cavalot F, Petrelli A, Traversa M, et al. Post prandial blood glucose is a stronger predictor of cardiovascular events than fasting blood glucose in type 2 diabetes mellitus, particularly in women: lessons from the San Luigi Gonzag Diabetes study. *J Clin Endocrinol Metab.* 2006; 91;813-9.

8. Marfella R, Nappo F, De Angelis L, Sinischalchi M, Rossi F, Giugliano D: The effect of acute hyperglycemia on QTc duration in healthy man. *Diabetologia.* 2000; 43 : 571 – 575

9. Gokhroo R, Mittal SR. Electrocardiographic correlates of hyperglycemia in acute myocardial infarction. *Int J Cardiol.* 1989; 22: 267 – 269.

10. Bonara E, Muggeo M. Post prandial blood glucose as a risk factor for cardiovascular disease in type II diabetes : the epidemiological evidence. *Diabetologia.* 2001; 44: 2107 – 2114.

11. Eposito K, Giugliano D, Nappo F, Marfella R. Regression of carotid atherosclerosis by control of post prandial hyperglycemia in type 2 diabetes mellitus. *Circulation.* 2004; 29: 2978 – 2984.

12. Diwadkar VA, Anderson JW, Bridges SR, Gowri MS, Oelgten PR. Post prandial low density lipoproteins in type 2 diabetes are oxidized more extensively than fasting diabetes and control samples. *Proc Soc Exp Biol Med.* 1999; 222: 178 – 184.

13. Ceriello A, Bortolotti N, Motz E, Pieri C, Marra M, Tonutti L, Lizzio S, Feletto F, Catone B, Taboga C. Meal – induced oxidative stress and low density lipoprotein oxidation in diabetes : the possible role of hyperglycemia. *Metabolism.* 1999; 48: 1503 – 1508.

14. Shige H, Ishikawa T, Suzukawa M, Ito T, Nakajima K, Higashi K,et.al. Endothelieum – dependent flow – mediated vasodilation in the postprandial state in type 2 diabetes mellitus. *Am J Cardiol.* 1999; 84: 1272 – 1274.

15. Ceriello A, Taboga C, Tonutti L, Giacomello R, Stel G, Motz E, et al. Post- meal coagulation activation in diabetes mellitus: the effect of acarbose. *Diabetologia.* 1996; 39: 469 – 473.

16. Nappo F, Esposito K, Cioffi M, Giugliano G, Molinari AM, Paolisso G et al. Post prandial endothelial activation in healthy subjects and in type 2 diabetic patients: role of fat and Carbohydrate meals. *J Am Coll Cardiol.* 2002; 39: 1145 – 1150.

17. Cavallero E, Dachet C, Neufcou D, Wirquin E, Mathe D, Jacotot B. Post prandial amplification of lipoprotein abnormalities in controlled type II diabetic subjects: relationship to post prandial lipemia and C – peptide / glucagon levels. *Metabolism.* 1994; 43: 270 – 278.

18. Heine RJ, Dekker JM. Beyond post prandial hyperglycemia: metabolic factors associated with cardiovascular disease. *Diabetologia.* 2002; 45: 461 – 475.

19. Ceriello A, Taboga C, Tonutti L, Quagliaro L, Piconi L, Bais B et al. Evidence for an independent and cumulative effect of post prandial hypertriglyceridemia and hyperglycemia on endothelial dysfunction and oxidative stress generation : effects of short and long term Simvastatin treatment. *Circulation.* 2002; 106: 1211 – 1218.

20. Ceriello A, Quagliaro L, Piconi L, Assaloni R, Da Ros R, Maier A et al. Effect of post prandial hypertriglyceridemia and hyperglycemia on circulating adhesion molecules and oxidative stress generation and the possible role of Simvastatin treatment. *Diabetes.* 2004; 53: 701- 710.

21. Diabetes diagnosis : Fasting or ost prandial blood glucose. *J Diabetes Sci Technol.* 2010; 4(2):236-243.

22. Matthew Riddle, Guillermo Umpierrez, Andres Digenio, Rong Zhu, Julio Rosenstock. Contribution of basal and post prandial hyper glycemis over a wide range of A1C levels before and after treatment intensification in type 2 Diabetes : *Diabetes care.* 2011; 34: 2508-2514.

23. Font bonne Em, Eschwege Em. Insulin and cardiovascular disease. Paris prospective study. *Diabetes care.* 1991;14(6): 461-469 [Pub med].

24. Wolever TM, Yand M, Zeng XY, Atkinson F, Brand-Miller JC. Food glycemic index, as given in glycemic index tables, is a significant determinant of glycemic responses elicited by composite breakfast meals. *Am J Clin Nutr.* 2006; 83(6):1306-1312. [Pub med].

25. JB Keogh, CWH Lau, M Noakes, J Bowen, PM Clifton: Effects of meals with high soluble fibre, high amylase barley variant on glucose, insulin, satiety and thermic effect of food in healthy lean women. *Eur J Clin Nutr.* 2007; 61 : 597 – 604.

26. Steward SL, Black RM, Wolever TMS, Anderson GH. The relationship between the glycaemic response to breakfast cereals and subjective appetite and food intake. *Nutr Res.* 1997; 17: 1249 – 1260.

27. Anderson Gh, Woodend D. Effect of glycemic carbohydrates on short – term satiety and food intake. *Nutr Res.* 2003; 61: S17-S26.

28. Holt S, Brand J, Soveny C, Hansky J. Relationship of satiety to post prandial glycaemic, insulin and cholecystokinin responses. *Appetite.* 1992; 18: 129 – 141.

29. Holt SHA, Brand – Miller J. Increase insulin responses to ingested foods are associated with lessened satiety. *Appetite.* 1995; 24: 43-54.