e-ISSN: 0975-5160, p-ISSN: 2820-2651

Available online on www.ijtpr.com

International Journal of Toxicological and Pharmacological Research 2022; 12(1); 170-174

Original Research Article

Study of Pancreatic Enzyme in Type 2 Diabetes Mellitus Patient

Bachchoo Singh[1], Laxman Prasad Gupta[2]

[1]Assistant Professor Dept. of General Medicine Krishna Mohan Medical College and Hospital, Pali Dungra, Sonkh Road, Mathura

[2]Associate Professor Dept. of General Medicine Krishna Mohan Medical College and Hospital, Pali Dungra, Sonkh Road, Mathura

Received: 10-11-2021 / Revised: 18-12-2021 / Accepted: 30-01-2022
Corresponding author: Dr. Laxman Prasad Gupta
Conflict of interest: Nil

Background:

Introduction: Type 1 and type 2 diabetes are frequently associated by exocrine pancreatic insufficiency, which is usually moderate. Despite its great prevalence, little is known about the clinical implications of exocrine pancreatic insufficiency and how best to treat it (nutritionally). Even little is known about whether and to what extent exocrine pancreatic insufficiency impacts diabetes glycemic management. This article attempts to summarize current clinical information on screening, diagnosis, and treatment of exocrine pancreatic insufficiency in diabetes, as well as provide an overview of the pathophysiology of exocrine pancreatic insufficiency.

Result: mean plasma fasting blood sugar level higher in study group (190.8±65.0) as compared to control group (80.4±11.70). Serum amylase level lower in study group (27.90±2.39) as compared to control group (47.39±22.90). Serum lipase level lower in study group (33.01±2.80) as compared to control group (68.90±32.95).

Conclusion: According to our findings, decreased serum amylase and serum lipase concentrations in type 2 diabetes mellitus might be connected to a disruption of the exocrine-endocrine axis, resulting in altered exocrine pancreatic function.

Keyword: Diabetes Mellitus, Pancreatic Function, Amylase, Lipase

Introduction

Diabetes mellitus (DM) is on the rise, with global prevalence expected to double by 2030, from 171 million in 2000 to 366 million, with India leading the way. It is anticipated that DM would affect 79.4 million individuals in India by 2030. [1]

Type 1 and type 2 diabetes are caused by insulin resistance and low or non-existent insulin production, respectively. Insulin resistance is described as a reduction in the target organ's sensitivity to insulin's biochemical actions. [2]

Exocrine and endocrine activities are shared by the pancreas. The digesting enzymes amylase, lipase, and trypsin are secreted by the exocrine pancreas, whereas the hormones insulin, glucagon, and somatostatin are secreted by the endocrine pancreas. Within the exocrine acinar cells, the pancreas has a

complicated structure with clusters of distinct endocrine cells distributed. [3]

The pancreas is a mixed exocrine–endocrine gland that has an exocrine portion that makes up the majority of its volume (84 percent).Around 4% of the volume is made up of ductal cells and blood vessels, whereas 2% is made up of the endocrine system. An extracellular matrix takes up the remaining 10% of the space. The islands are close to the tissues of the acinar pancreas. Due to its close anatomical affinity, active interactions between the exocrine and endocrine pancreas are possible in any disease affecting the organ. [4] An islet-exocrine portal system bathes the acinar cells anatomically with blood from the islets. [5]

Exocrine acinar cells in the pancreas create enzymes like amylase and lipase, which aid in the digestion of specific food particles. The enzyme amylase breaks down starch into maltose, maltotriose, and -limit dextrins during digestion. Lipase is a digestive enzyme that originates in the pancreas and proceeds to the stomach, where it aids in the breakdown of triglycerides into fatty acids and monoglycerides. Foods are not effectively digested due to a lack of pancreatic enzymes, resulting in poor digestion and malnutrition. [6, 7]

In diabetics with pancreatic exocrine insufficiency, GI symptoms such as loose bowel movements, stomach pain, and gas are common. Exocrine insufficiency in diabetes mellitus can lead to a macronutrient shortage, steatorrhoea, and eventually malnutrition. Exocrine pancreatic function has received minimal attention in our country's human diabetes research due to a paucity of published data. The majority of the research looked at insulin resistance and chronic hyperglycemia as possible causes of metabolic problems. [8]

Material Method:

This cross sectional study conducted at biochemistry department and medicine department at this medical college and hospital.

Sample size:

There are 100 subjects included this study 50 subjects for diabetes mellitus and 50 subjects for healthy individual.

All the subjects included in the study were in the age group of 35to 70 years.

Table 1: Inclusion and exclusion criteria.

Inclusion criteria:	Exclusion criteria:
• Fasting blood glucose level more than 110 mg/dl • Random blood glucose level more than 180 mg/dl	• Patients age below 25 years. • Hepatic, chronic renal failure, gastrointestinal, skeletal and endocrine diseases.

Sample Collection and Biochemical Analysis:

8 ml fasting blood sample was collected intravenously and centrifuged for 10-15 minutes in a centrifuge. The separated serum was used for analysis of plasma sugar, serum amylase and serum lipase level estimated by fully automated clinical chemistry analyzer (AU-480).

Statistical Analysis:

The SPSS (Statistical Package for the Social Sciences) application version-22 was used to conduct the statistical analysis. When $P< 0.05$, the significance was assessed. Microsoft Word version 2016 was used to chart and graph the statistical data.

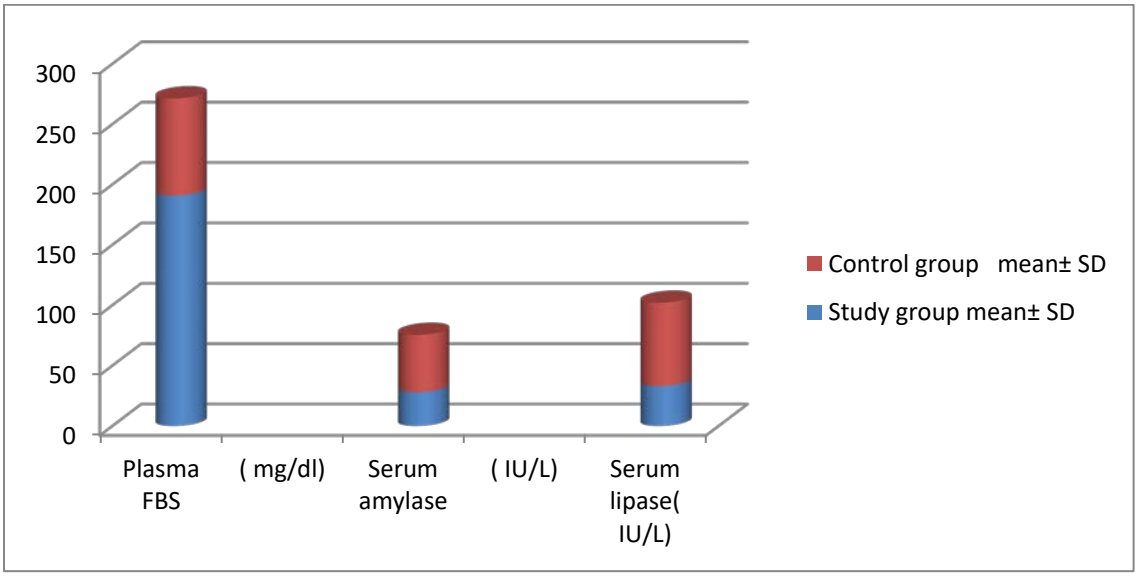
Result:

Table 2: Mean comparison of plasma glucose, serum amylase and serum lipase level in control group and study group.

Parameter	Study group mean± SD	Control group mean± SD	p- value
Plasma FBS (mg/dl)	190.8±65.0	80.4±11.70	P < 0.0001
Serum amylase (IU/L)	27.90±2.39	47.39±22.90	P < 0.0001
Serum lipase (IU/L)	33.01±2.80	68.90±32.95	P < 0.0001

Table 02 show the mean plasma fasting blood sugar level higher in study group (190.8±65.0) as compared to control group (80.4±11.70). Serum amylase level lower in study group (27.90±2.39) as compared to control group (47.39±22.90). Serum lipase level lower in study group (33.01±2.80) as compared to control group (68.90±32.95).

Graph no:1 show the level of fasting blood glucose level, serum amylase and lipase level in control group and study group.



Graph no 01 our study show that plasma fasting blood glucose level higher in study group as compared to control group. Serum amylase and serum lipase level lower in study group as compared to control group.

Discussion:

In disorders of the pancreas, the human exocrine pancreatic secretions are altered. The decreased exocrine-endocrine interactions of the pancreas may be reflected in reduced blood amylase levels in diabetes. Only a few clinical research, however, have looked into this complex link. [8]

I our study table no 01 showed that plasma fasting blood sugar when compared to the control group, there was a considerable rise in the study group. (190.8±65.0, 80.4±11.70) p-value P < 0.0001.

Serum amylase concentration significantly decreased in study group as compared to control group (27.90±2.39, 47.39±22.90) P < 0.0001. Serum lipase concentration significantly decreased in study group as compared to control group (33.01±2.80, 68.90±32.95) P < 0.0001.

Hyperglycemia in diabetes mellitus induces cellular damage to the exocrine pancreas and

affects the production of pancreatic digesting enzymes, according to a review of the literature. [9]

Hyperglycemia, according to some authors, can disrupt cellular signalling in the pancreas, which controls both transcription and protein metabolism, resulting in pancreatic exocrine insufficiency in DM. [10]

Insulin and glucagon, two hormones of the pancreas, have been identified to regulate enzyme synthesis and release in the exocrine pancreas. Exocrine production is inhibited by glucagon, and the sensitivity of pancreatic acini to diabetes is reduced. Insulin deficiency and excessive glucagon disrupt the natural environment of the pancreas in sugar, lowering total volume, amylase secretion, and bicarbonate content of exocrine secretion. [11]

About 50 percent of people with diabetes have pancreatic fibrosis, and pathological factors include atrophy, fat intake, and loss of exocrine acinar cells. [12]

Exocrine dysfunction and decreased entero-pancreatic reflexes are also common complications of diabetic neuropathy. Increased hormone and peptide concentrations may decrease exocrine function (glucagon, pancreatic polypeptide P, somatostatin). TGF-beta 1, TGF-alpha (growth mutants), TNF-alpha (tumor necrosis factor), gastrin, and insufficient genetic activity have recently been added to the list of cytokines that may interfere with and damage the functions of exocrine and endocrine. [13,14]

Conclusion:

According to our findings, decreased serum amylase and serum lipase concentrations in type 2 diabetes mellitus might be connected to a disruption of the exocrine-endocrine axis, resulting in altered exocrine pancreatic function.

Reference:

1. 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(3):1047-53.
2. Xu H, Huang X, Arnlov J, Cederholm T, Stenvinkel P, Lindholm B, et al. Clinical correlates of insulin sensitivity and its association with mortality among men with CKD stage 3 and 4. Clin J Am Soc Nephrol. 2014; 9:1–8.
3. Henderson JR, Daniel PM, Fraser PA. "The pancreas as a single organ: the influence of the endocrine upon the exocrine part of the gland"Gut**(**1981) 22,158-67.
4. Singh J, Yago MD, Adeghate E The role of insulin, glucagon, somatostatin, cholecystokinin, acetylcholine and nerve stimulation in the interactions between the endocrine and exocrine pancreas in normal and diabetic conditions in rats. Int J Diabetes. 1999; 7(1):114-19.
5. Bertelli E and Bendayan M. association between endocrine pancreas and ductal system. More than an epiphenomenon of endocrine differentiation and development. J Histochem Cytochem. 2005;53(9):1071-86.
6. Hayden MR et al. Attenuation of endocrine-exocrine pancreatic communication in type 2 diabetes: pancreatic extracellular matrix ultrastructural abnormalities. J Cardiometab Syndr. 2008; 3:234–43.
7. Barrett KE, Barman SM, Boitano S, Brooks HL. Ganong's review of medical physiology. 24thed. New Delhi: Tata McGraw-Hill. Chapter 26, Digestion, Absorption and Nutritional Principles; 2012:477-495.
8. Noor-E-Jannat Tanvi1, Qazi Shamima Akhter et al. Serum amylase and lipase levels in type 2 diabetes mellitus J Bangladesh Soc Physiol. 2017, December; 12(2): 52-56.

9. Ata N, Dal K, Kucukazman M, Yeniova AO, Karakaya S, Unsal O et al. The effect of glycemic control on CEA, CA 19-9, amylase and lipase levels. Open Med. 2015; 10: 8-13.
10. Patel R, Atherton P, Wackerhage H, Singh J. Signaling protein associated with diabetic-induced exocrine pancreatic insufficiency in rats. Ann N Y Acad Sci 2006 Nov; 1084:490-502.
11. Singh J, Yago MD, Adeghate E The role of insulin, glucagon, somatostatin, cholecystokinin, acetylcholine and nerve stimulation in the interactions between the endocrine and exocrine pancreas in normal and diabetic conditions in rats. Int J Diabetes. 1999;7(1):114-19.
12. Hayden MR et al. Attenuation of endocrine-exocrine pancreatic communication in type 2 diabetes: pancreatic extracellular matrix ultrastructural abnormalities. J Cardiometab Syndr. 2008; 3:234–43.
13. Rakhee Yadav et al., the Evaluation of Serum Amylase in the Patients of Type 2 Diabetes Mellitus. Journal of Clinical and Diagnostic Research. 2013 ;7(7): 1291-1294
14. Margute, T. G., Ferreira , P. C., Almeida, I. M. M., Denardin, C., Silva , T. Q. M. da, Margute , T. G., Maione, M. S., Rossato, A. R., & Santos, I. F. dos. Use of tricyclic antidepressants in trigeminal neuralgia. Journal of Medical Research and Health Sciences, 2022:5(5), 2008–2012.