

A Study of Serum Amylase and Lipase Levels in Type 2 Diabetic Subjects**Hariprasath G.¹, Yogapriya V.², Ramana Sai P.³, Ashwin Raj H.⁴, Jyothirmayi B.⁵**¹Assistant Professor, Department of Biochemistry, Madha Medical College & Research Institute, Chennai, Tamil Nadu, India²Associate Professor, Department of Biochemistry, Madha Medical College & Research Institute, Chennai, Tamil Nadu, India³Tutor, Department of Biochemistry, Madha Medical College & Research Institute, Chennai, Tamil Nadu, India⁴Assistant Professor, Department of Biochemistry, Madha Medical College & Research Institute, Chennai, Tamil Nadu, India⁵Professor & Head, Department of Biochemistry, Madha Medical College & Research Institute, Chennai, Tamil Nadu, India

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Conflict of interest: Nil

Abstract:**Background:** Type 2 Diabetes Mellitus is a chronic metabolic disorder characterized by insulin resistance and pancreatic β cell dysfunction. Amylase and Lipase are digestive enzymes secreted by the acinar cells of exocrine pancreas. Pancreatic β -cell dysfunction in Diabetes can interfere with the exocrine function of the pancreas. In the present study we assessed serum Amylase and Lipase levels in subjects with Type 2 Diabetes Mellitus.**Materials and Methods:** The study included 50 Type 2 Diabetic Subjects and 50 age-matched healthy individuals with no history of Diabetes. Fasting and Post Prandial plasma glucose, HbA1c, Serum Amylase and Serum Lipase were measured in all these subjects.**Results:** Fasting plasma glucose, Post Prandial plasma glucose and HbA1c were significantly higher in subjects with Type 2 Diabetic subjects compared to controls ($p < 0.01$). A significant elevation of Serum Amylase and Lipase ($p < 0.01$) levels that are within the normal reference range is observed in Type 2 Diabetic subjects when compared to controls.**Conclusion:** The compensatory hyperinsulinemia in Type 2 Diabetes may increase the synthesis of serum Amylase and Lipase via stimulation of islet acinar axis. Elevated Serum Amylase and Lipase levels within normal range may be an early sign of pancreatic impairment in Type 2 Diabetes Mellitus and may be useful in monitoring the progression of the disease.**Keywords:** Pancreatic β -cell Dysfunction, Islet Acinar Axis, Hyperinsulinemia, Pancreatic Impairment.This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.**Introduction**

Type 2 Diabetes Mellitus is a chronic metabolic disorder characterized by insulin resistance and pancreatic β cell dysfunction [1]. Though the exact pathogenesis is not clear, insulin resistance and pancreatic β -cell dysfunction are considered to play a central role in the development of Type 2 Diabetes Mellitus [2]. The pancreas is a dual gland with an exocrine function that synthesizes and secretes digestive enzymes and an endocrine function which produces hormones that regulate blood glucose [3]. The exocrine and endocrine parts of pancreas are in close proximity to each other anatomically and functionally and they work together to achieve metabolic balance [4]. Hence impairment in one part can have implications over the other part in pancreas.

Amylase and Lipase are digestive enzymes produced by the acinar cells of exocrine pancreas [5]. Amylase is involved in the breakdown of dietary starch to glucose, maltose and maltotriose [6] and Lipase catalyses the hydrolysis of dietary fat into free fatty acids and glycerol [7]. Studies suggest that serum Amylase and serum Lipase can be used as biochemical markers for the assessment of the pancreatic exocrine function [8]. In the present study we assessed serum Amylase and Lipase activities in subjects with Type 2 Diabetes Mellitus.

Material & Methods

This study was conducted in the Department of Biochemistry, Madha Medical College & Research Institute, Chennai. 50 Type 2 Diabetic subjects in

the age group of 35–60 years were selected for this study and 50 age-matched healthy individuals were selected as a control group. The study was approved by the institutional ethical committee. Informed consent was obtained from all the subjects. The Subjects and Controls voluntarily participated in this study. All the Type 2 Diabetic subjects were on oral hypoglycemic drugs. The subjects who were smokers, known alcoholics, on insulin treatment, having complications of Diabetes Mellitus, with history of Type 1 Diabetes Mellitus, liver diseases and renal diseases were all excluded from the study. Fasting Plasma Glucose, Post Prandial Plasma Glucose, HbA1c, Serum Amylase and Serum Lipase were all measured in these subjects.

Statistical Analysis: The statistical analysis was performed using SPSS statistics version 20.0. The results are expressed as mean \pm SD. Statistical difference between the control and Type 2 Diabetic

Subjects were carried out using Student “t” test and p value of < 0.05 was considered to be statistically significant.

Results

Table I shows the Age and Biochemical characteristics of healthy controls and Type 2 Diabetic Subjects.

The controls and Type 2 Diabetic Subjects were of same age group and no significant difference was observed between the groups.

Fasting plasma glucose, Post Prandial plasma glucose & HbA1c levels were significantly higher in Type 2 Diabetic Subjects than the control group ($p < 0.01$). A significant elevation of Serum Amylase and Serum Lipase ($p < 0.01$) that are within the normal range is observed in Type 2 Diabetic subjects when compared to controls.

Table 1: Comparison of Biochemical characteristics between Type 2 Diabetic Subjects & Controls.

Parameters	Controls (n=50) Mean \pm SD	Type 2 Diabetic Subjects (n=50) Mean \pm SD	p-Value
Age (years)	47.18 \pm 4.78	48.36 \pm 7.35	0.34
Fasting Plasma Glucose (mg/dl)	93.5 \pm 7.1	179.8 \pm 68.8	$p < 0.001$
Post Prandial Plasma Glucose (mg/dl)	109.9 \pm 12.12	259.3 \pm 78.9	$p < 0.001$
HbA1c (%)	5.79 \pm 0.38	8.64 \pm 1.37	$p < 0.01$
Serum Amylase (IU/L)	15.4 \pm 3.9	38.25 \pm 12.56	$p < 0.01$
Serum Lipase (IU/L)	25.8 \pm 3.5	40.75 \pm 11.2	$p < 0.01$

Discussion

The present study was done to assess Serum Amylase and Serum Lipase levels in Type 2 Diabetic Subjects. The participants in both the study groups were of same age group (47.18 \pm 4.78 Vs 48.36 \pm 7.35) and no significant difference between the groups ($p > 0.05$). The mean Fasting plasma glucose, Post Prandial plasma glucose and HbA1c levels were all significantly elevated in Type 2 Diabetic subjects than healthy controls. This indicates the poor control of blood glucose among diabetic subjects. Insulin resistance can induce an imbalance in glucose metabolism causing generation of chronic hyperglycemia in Type 2 Diabetics [9].

The present study shows that serum Amylase and Lipase are significantly increased in subjects with Type 2 Diabetes than healthy controls ($p < 0.01$). But this increase was within normal limits. This may indicate early stage in impairment of pancreatic function. This finding is consistent with the observation of Bastyr et al (2009) [10] and Steinberg et al (2014) [11] who also reported elevation of Amylase and Lipase activity in subjects with Type 2 Diabetes. However, Rakhee Yadav et al (2013) [12] and Mahesh Basavaraj Madole et al (2016) [13] in their study reported

decreased Amylase and Lipase activity in Type 2 Diabetes. This discrepancy might be due to the difference in duration of diabetes among the study subjects. Persistent hyperglycemia in long standing diabetes under poor glycemic control might cause acinar cell damage resulting in pancreatic fibrosis and atrophy thereby leading to insufficiency of exocrine acinar cells [15], which would have resulted in reduced Amylase and Lipase activity in their study.

The cause of increase in pancreatic enzymes in the Type 2 Diabetes is still unknown and requires further investigation. The possible mechanism for the increased Amylase and Lipase levels may be due to the presence of insulin resistance in Diabetes which is compensated by secondary hyperinsulinemia which may increase the pancreatic exocrine function via stimulating islet acinar axis leading to increased production of pancreatic enzymes [16].

Conclusion

Elevated levels of Amylase and Lipase within normal range may be an early sign of pancreatic impairment in Type 2 Diabetes Mellitus and may be useful in monitoring the progression of the disease.

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