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Study of Serum Amylase Levels in Patients with Type 2 Diabetes Mellitus in a Tertiary Care Hospital

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Abstract

Background: Diabetes mellitus is an endocrine disorder of the pancreas, due to inadequate synthesis of insulin and /or resistance to the action of it. There is a close anatomical and functional relationship between its endocrine and exocrine portions both directly and in directly. So we are studying the Serum amylase in Type2 Diabetes mellitus (DM). The aims and objective is to study the Serum Amylase levels in patients of Type-2 Diabetes Mellitus and to correlate Serum Amylase level with the HbA1c level in patients with type-2 DM. **Subjects and Methods**: Patients with Type-2 Diabetes Mellitus aged more than 30 years were taken for the study. After obtaining consent from patient, a detailed history was taken and a proper clinical examination was done. Relevant clinical investigations were done. Serum Amylase was measured. Then Serum Amylase level was compared with the HbA1c level. **Results**: In this study, serum amylase level was statistically significantly lower in Type 2 DM group compared to non diabetics group with p value <0.01. **Conclusion**: The study revealed that patients with Type 2 DM the exocrine functions of pancreas are also affected. Patients of Type 2 DM have low serum amylase levels. Serum amylase levels was negatively correlated with HbA1c levels in patients with type 2 DM, indicating the exocrine dysfunction which needs further evaluation to rule out chronic exocrine pancreatic insufficiency in type 2 DM patients .

Keywords: Serum Amylase, Hba1c, Type 2 Diabetes Mellitus.

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Introduction

Diabetes Mellitus (DM) is a metabolic disease occurring either due to absolute insulin deficiency or reduced sensitivity of tissues to insulin.[1] Diagnostic criteria for Diabetes Mellitus are Fasting blood Sugar level (FBS)≥126mg/dl, Postprandial blood sugar (PPBS) ≥200mg/dl, Glycosylated Haemoglobin (HbA1C) ≥6.5% or Random blood sugar (RBS) ≥200mg/dl.[2]

Diabetes is a danger to global health and will remain so. In the United Kingdom, at least 2% of the population (over 1 million) is diabetic and 5 to 10% of the overall health budget is consumed by the disease.[3] Given the high prevalence of diabetes among Indians with over 20 million diabetics, and the number projected to grow to 57 million by 2025, this could put a huge burden on the country's health budget.[4]

The pancreas is a mixed gland, with 84% of the gland made up of exocrine portion while the endocrine part comprises 2% and remaining portion is connective tissue.[5] Since there is a near anatomical and functional association between these two sections of the pancreas, one portion of the disease can affect the other.[6,7] The exocrine acinar cells are exposed to high islet hormone concentrations as they receive blood passes from the surrounding islets.[8]

Several types of enzymes, including amylase and lipase, are produced by the pancreatic exocrine acinar cells that aid in the digestion of specific food particles. In the digestion process, amylase is the main enzyme responsible for cleaving starch into maltose, maltriose, and alpha-limit dextrin. Lipase comes predominantly from the pancreas, moves to the intestine all the way down, and facilitates triglyceride breakdown into fatty acids and monoglycerides. Foods are not properly digested due to deficiency of pancreatic enzymes and the effects are maldigestion and malnutrition.[9] For many years, it has been thought that low serum amylase levels due to advanced pancreatic disease reflect diffuse pancreatic harm. But several studies have now shown that this low serum amylase level

is also associated with metabolic syndrome and diabetes mellitus.[10]

Research investigating the impact of hyperglycaemia on pancreatic exocrine functions in type 2 diabetic patients reported a substantial increase in serum amylase and lipase levels with glycemic control regulation, but still lower than control levels. A strong negative association between serum amylase and lipase and basal FBS and HbA1c was found.[11]

Insulin resistance results in increased activity of anti-insulin hormones and atrophy of exocrine acinar cells in diabetic patients. Thus the synthesis and secretion of the exocrine pancreatic enzyme is reduced.[12] With the onset of type 2 diabetes mellitus, coordination between the endocrine islets and the exocrine acinar cells was also lost. The exocrine pancreas displays a reaction to hormonal stimulation in the form of fibrotic changes on long term exposure.[13] In diabetes mellitus, insulin inactivity and/or hyperglycaemia due to insulin resistance or low insulin may induce pancreatic exocrine dysfunction and the development of pancreatic-exocrine insufficiency.[14]

A deficiency of exocrine pancreatic enzymes resulting in an inability to maintain normal digestion is known as pancreatic exocrine insufficiency (PEI).[15] The most common etiology of PEI is chronic pancreatitis. Prevalence of Pancreatic Exocrine Insufficiency (PEI) in Diabetes Mellitus: In recent decades, there have been several studies on PEI in patients with diabetes mellitus. Pancreatic exocrine function was tested in early studies with the gold-standard direct pancreatic function test tool (pancreozymin-secretin test). In 52.4 percent of the cases.[16–21] However since direct pancreatic function tests are invasive, time-consuming and costly, these trials have only been limited to a small number of patients. This exocrine insufficiency in diabetes mellitus can also lead to a deficiency of macronutrients, steatorrhoea and consequent malnutrition.[8]

In the human diabetic research of our country very small concern has been given to pancreatic exocrine function, as less published data is available regarding this topic. Majority of the studies focus on the metabolic derangement due to impaired insulin action and persistent hyperglycaemia. Therefore, the current study has been designed for evaluating pancreatic exocrine function by checking serum amylase and lipase in Type 2 DM.

Aims

Study the Serum Amylase level in patients of Type-2 Diabetes mellitus and Compared with non diabetics control group and to study the correlation of Serum Amylase with HbA1c level of Type 2 Diabetes mellitus.

Subjects and Methods

This prospective observational study was done on Type 2 DM patients admitted from November 2018 to October 2020 in the Department of Medicine at Karnataka Institute of Medical Sciences, Hubballi. Total of 100 cases and 100 control patients were included in the study. The inclusion criteria for cases: Type 2 Diabetes Mellitus cases above the age of 30 years admitted to KIMS Hospital in Department of General Medicine and for control: non-Diabetics subjects of similar age and sex matching for cases coming to KIMS hospital. The exclusion criteria were patients with Acute and Chronic pancreatitis, any case of Acute Abdomen, Type-1 DM patients, patients with Liver diseases, patients with deranged renal function, patients with parotid related problem and patients who did not give consent for the study.

The study was carried out in patients with type 2 diabetes mellitus aged more than 30 years. Detailed history and clinical review was conducted after taking authorisation from the patient. The levels of serum amylase were assessed. The levels of serum amylase in the diabetic group were then compared with that of the non-diabetic control group. The following investigations were done**:** Complete blood count (CBC), rental function tests, liver function tests, Glycosylated haemoglobin (HbA1c), Fasting blood sugar (FBS), Postprandial blood sugar (PPBS), Serum Amylase and USG abdomen and pelvis. CT abdomen and C Peptide if needed.

Statistical analysis:

A statistical analysis of the serum amylase levels and their correlation to HbA1c levels to assess significance in the values obtained. The data was represented in Microsoft Excel and word were used to obtain various types of graphs such as Pie diagram, bar diagram and Scatter plots. A ‘p’ value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests. Data was analysed by using MS Excel, SPSS version 22 (IBM SPSS Statistics, Somers NY, USA).

Results

In the study both in case and control groups 58% were males and 42% were females. [Table 1] shows the age distribution of cases and control group. In the study age and sex matching controls for the cases were taken. In the case group 8% were in the age group <40 years, 32% were in the age group 40-49 years, 29% were in the age group 50-59 years, 23% were in the age group 60-69 years, 8% were in the age group $\geq$70 years which was similar for control group also. [Table 2] shows distribution of mean S. Amylase among cases and control group. In our study mean S. Amylase levels showed a statistically significant difference between the control and

Table 1: Age distribution of cases and control group.

Age in yrs	Cases	Controls	In Percentage
<40	8	8	8%
40-49	32	32	32%
50-59	29	29	29%
60-69	23	23	23%
≥70	8	8	8%
Total	100	100	100%

case group with p value <0.001. [Table 3] shows HbA1C distribution among cases and controls. In our study mean HbA1c showed a statistically significant difference between the control and case group with p value <0.001. [Table 4] shows Mean S. Amylase with respect to HbA1c among case group. In this study among the case group, patients who had an HbA1c level below 7 had a mean S. Amylase level of 41.85±15.45 which was higher compared to the patients who had an HbA1C level ≥ 7, mean amylase level was 31.87±14.49. The P value 0.004 we obtained, this indicated that the patients with good glycemic control had a lesser effect on exocrine function of pancreas, when compared to patients with uncontrolled glycemic index.

Discussion

In our study 200 subjects were enrolled, out of which 100 who had type 2 DM were included under the case group and 100 were taken as control group who were non diabetic. They underwent a detailed history, clinical examination, hematological investigation, FBS, PPBS, HbA1c and serum amylase and lipase levels were done. Serum amylase level was compared between the case and control groups and amylase was studied with respect toHbA1c levels in the case group.

In our study patients with type 2 DM were included as the case group. Age and sex matching subjects were taken as the control group. Known cases of parotid disorder, pain abdomen patients and chronic pain abdomen patients were excluded since these conditions were known to alter the levels of serum amylase level. Known cases of nephropathy and liver disease were also excluded from the study since these conditions are known to alter the blood sugar and HbA1c levels.

200 patients were enrolled in the study, 100 were in the case group and 100 were in the control group. In the study the mean age among case group was 53.84±10.21 and in the control group was 53.84±10.21. The mean age of patients in other various studies were Reenajain et al,[22] in case group 53.41±10.4 and control group 50.15±5.5, Tanvi et al,[23] in case group 47.06±0.65 and in control group 45.64±0.59, Basavaraj et al,[24] in case group 54.33±3.5 and in control group 35.73±4.5, Kalitha et al,[25] in case group 45.73±6.13 and in control group 44.54±7.14, Vishwanath et al,[26] in case group and in control group 55±8.6 56.1±8.3.It was observed that our study mean age distribution was almost similar to Reenajain et al,[22] and Vishwanath et al.[26]

In our study among the case group out of 100, 58% were males and 42% were females. In other various studies sex distribution in case group was Reenajain et al,[22]70% were males,30% were females, in Tanvi et al,[23] 56% were males and 44% were females, in Basavaraj et al,[24] 50% males and 50% females, Kalitha et al,[25] 61.33% were males and 38.66% were females, in Vishwanath et al,[26] 63.33% were males and 36.66% were females.

In our study among the control group out of 100, 58% were males and 42% were females. In other various studies sex distribution in control group was Reenajain et al,[22] 68% were males and 32% were females, in Tanvi et al,[23] 52% were males and 48% were females, in Basavaraj et al,[24] 40% males and 60% females, Kalitha et al,[25] 58% were males and 42% were females, in Vishwanath et al 70% were males and 30% were females.

In our study male patients with T2DM among the case group were higher compare to females, this difference was also observed in other studies as seen above except in Basavaraj et al,[24] and our study sex distribution almost similar to Tanvi et al,[23] Kalitha et al.[25]

In our study the mean Serum amylase levels (U/L) among the case and control group respectively was 34.96±17.6 and 71.14±24.57. In other various studies mean Serum amylase(U/L) in case and control group respectively were Reenajain et al,[22] 48.13±9.16 and 73.9±23.1, in Tanvi et al,[23] in 33.37±2.36 and 56.37±1.84 Basavaraj et al,[24]56.37±23.48 and 80.83±20.26, in Kalitha et al,[25] 47.95±23.63 and in 107.1±37.97, in Vishwanath et al.[26] 56.13±40.50 and 98.66±0.93.

Our study showed that there was a significant difference in the mean amylase levels between the case and control groups with a p value <0.001 and this was almost similar to Tanvi et al,[23] and Reenajain et al.[22] This shows that patients with type 2 DM have significantly reduced serum amylase levels compared to non diabetic patients, suggesting that diabetes

Table 2: Distribution of mean S. Amylase among cases and control group

	S. Amylase U/L	
	Value	**SD**
Cases	34.96	17.60
Controls	71.14	24.83
P-Value	<0.001	

Table 3: HbA1C distribution among cases and controls

	Cases	**Controls**	**P-Value**
Mean HbA1C %	7.52	5.19	<0.001
SD	1.04	0.61	

Table 4: Mean S. Amylase with respect to HbA1c among case group

	Mean S. Amylase U/L	
	Value	**SD**
HbA1C<7	41.85	15.45
HbA1C≥7	31.87	14.49
P-Value	0.004	

Table 5: Comparison of mean S. Amylase levels of patients

Study	**S. Amylase of Cases (U/L)**	**S. Amylase of Controls (U/L)**
Present study	34.96±17.6	71.14±24.57
Reenajain et al, [22]	48.13±9.16	73.9±23.1
Tanvi et al, [23]	33.37±2.36	56.37±1.84
Basavaraj et al, [24]	56.37±23.48	80.83±20.26
Kalitha et al, [25]	47.95±23.63	107.1±37.97
H.L.Vishwanath, [26]	56.13±40.50	98.66±0.93

affects the exocrine pancreas also.

Our study showed that there were reduced serum amylase and lipase levels in the case group compared to the control group with a p value <0.0001 which is statistically significant. The same observation was also seen in other studies such as Reena jain et al, [22] where the study showed low serum amylase levels in the case groups compared to the control group with a p value of < 0.001, Basavaraj madole et al, [24] studied exocrine function of the pancreas by measuring the serum amylase and lipase levels in DM patients, which showed that patients in both Type 1 and Type 2 DM groups had low serum amylase and lipase levels compared to the control groups with a p value <0.001 both for amyalse and lipase levels .Kei nakajima, Swislocki A et al, Snehankar K et al.,also showed reduced serum amylase level. [27–29] The same observation was also seen in Vishwanath et al, [26] with a p value <0.0001 when serum amylase levels were compared among the two groups and <0.006 when serum lipase levels were compared among the case and control group both the p values being statitically significant. [26]

It was observed that hormones like insulin and glucagon secreted by the pancreas influence the synthesis of enzymes and their release from the exocrine pancreas. Insulin has a trophic/ stimulatory effect on the acinar cells, whereas glucagon has an inhibitory influence on the exocrine secretions. These lead to a decrease in the sensitivity of the diabetic pancreatic acini to secretagogues. So, the deficiency of insulin and the excess of glucagon in diabetes affect the internal milieu of the pancreas. This leads to a decrease in the total volume, the amylase and the lipase secretion in its exocrine sections. [30] Similarly diabetic neuropathy may lead to impaired enteropancreatic reflexes and exocrine dysfunction. [31] A few studies in recent time have observed that cytokines such as TNF-alpha (tumor necrosis factor), TGF-alpha, TGF-beta 1 (transforming growth factors), gastrin and low regulatory gene functions may interact and impair the exocrine and endocrine functions. [32–34]

We observed low serum amylase level in Type 2 DM group compared to non diabetic group. This indicates that the endocrine functions of the pancreas have an influence on the exocrine functions. We observed from our study that there was a negative correlation between HbA1c of diabetes and serum amylase level. However, we need more studies for the same to know better.

Conclusion

The study revealed that patients with Type 2 DM the exocrine functions of pancreas are also affected. Patients of Type 2 DM have low serum amylase levels. Serum amylase levels wasnegatively correlated with HbA1c levels in patients with type 2 DM, indicating the exocrine dysfunction which needs further evaluation to rule out chronic exocrine pancreatic insufficiency in type 2 DM patients.

References

1. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. Diabetes Care . 2009;32:62–67. Available from: https://dx.doi.org/10.2337/dc09-S062.
2. Classification and diagnosis of diabetes: Standards of Medical Care in Diabetesd2020. Diabetes Care. 2020;43:14–31. Available from: https://doi.org/10.2337/dc20-s002.
3. Khan MAB, Hashim MJ, King JK, Govender RD, Mustafa H, Kaabi JA. Epidemiology of Type 2 Diabetes – Global Burden of Disease and Forecasted Trends. J Epidemiol Glob Health. 2020;10(1):107–111. Available from: https://dx.doi.org/10.2991/jegh.k.191028.001.
4. Ramachandran A, Snehalatha C, Latha E. Rising prevalence of NIDDM in an urban population in India. Diabetologia. 1997;40(2):232–237. Available from: https://doi.org/10.1007/s001250050668.
5. Singh J, Yago MD, E A. 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):114–133.
6. Pap A. Effect of insulin and glucose metabolism on pancreatic exocrine function. Int J Diabetes and Metabolism. 2004;12(3):30–34.
7. Jain R, Jain PK, Mangukiya K. Study of serum amylase in the patients of type 2 diabetes mellitus. IJSN. 2014;5(3):553–559.
8. Chakraborty PP, Chowdhury S. A Look Inside the Pancreas: The "Endocrine-Exocrine Cross-talk". Endocrinol Metab Synd. 2015;4(1):1–4.
9. Hackert T, Schütte K, Malfertheiner P. The Pancreas: Causes for Malabsorption. Viszeralmedizin. 2014;30(3):190–197. Available from: https://dx.doi.org/10.1159/000363778.
10. Nakajima K, Nemoto T, Muneyuki T, Kakei M, Fuchigami H, Munakata H. Low serum amylase in association with metabolic syndrome and diabetes: A community-based study. Cardiovasc Diabetol. 2011;10:34–34.
11. Ata N, Dal K, Kucukazman M, Yeniova AO, Karakaya S, Unsal O. The effect of glycemic control on CEA, CA 19-9, amylase and lipase levels. Open Med. 2015;10:8–13. Available from: https://dx.doi.org/10.1515/med-2015-0002.
12. Demir K. Pancreatic dyspepsia: a place for pancreatic insufficiency in dyspepsia. Eur J Surg Sci. 2012;3(1):1–4.
13. Hayden MR, Patel K, Habibi J. Attenuation of endocrine-exocrine pancreatic communication in type 2 diabetes: pancreatic extracellular matrix ultrastructural abnormalities. J Cardiometab Syndr. 2008;3(4):234–243. Available from: https://dx.doi.org/10.1111/j.1559-4572.2008.00024.x.
14. Nakajima K, Oshida H, Muneyuki T, Kakei M. Pancrelipase: an evidence-based review of its use for treating pancreatic exocrine insufficiency. Core Evid. 2012;7:77–91. Available from: https://doi.org/10.2147/ce.s26705.
15. Struyvenberg MR, Martin CR, Freedman SD. Practical guide to exocrine pancreatic insufficiency - breaking the myths. BMC Med. 2017;15(1):29. Available from: https://doi.org/10.1186/s12916-017-0783-y.
16. Hahn JU, Kerner W, Maisonneuve P, Lowenfels AB, Lankisch PG. Low fecal elastase 1 levels do not indicate exocrine pancreatic insufficiency in type-1 DM. Pancreas. 2008;36(3):274–278. Available from: https://doi.org/10.1097/mpa.0b013e3181656f8.
17. Pollard HM, Miller L, Brewer WA. External secretion of the pancreas and diabetes. Am J Dig Dis. 1943;10(1):20–23.
18. Cheywy SH, Shuman CR. External pancreatic secretion in diabetes mellitus. Ann Intern Med. 1953;59:812–833.
19. Vacca JB, Henke WJ, Knight WA. The exocrine pancreas in diabetes mellitus. Ann Intern Med. 1964;61:242–247. Available from: https://doi.org/10.7326/0003-4819-61-2-242.
20. Frier BM, Saunders J, Wormsley KG, Bouchier IA. Exocrine pancreatic function in juvenile-onset diabetes mellitus. Gut. 1976;17(9):685–91. Available from: https://doi.org/10.1136/gut.17.9.685.
21. Harano Y, Kim CI, Kang M, Shichiri M, Shimizu Y, Li H. External pancreatic dysfunction associated with diabetes mellitus. J Lab Clin Med. 1978;91(5):780–90.
22. Jain R, Jain PK, Mangukiya K. Study of serum amylase level in the patients of Type-2 Diabetes Mellitus. IJSN. 2014;5(3):553–556.
23. Tanvi NEJ, Akhter QS, Nahar S, Sumi MN, Hosen M. Serum amylase and lipase levels in type 2 diabetes mellitus. JBSP. 2017;12(2):52–56. Available from: https://doi.org/10.3329/jbsp.v12i2.35422.
24. Madole MB, Iyer CM, Madivalar MT, Wadde SK, Howale DS. Evaluation of Biochemical Markers Serum Amylase and Serum Lipase for the Assessment of Pancreatic Exocrine Function in Diabetes Mellitus. J Clin Diagn Res. 2016;10(11):1–04. Available from: https://doi.org/10.7860/jcdr/2016/23787.8900.
25. Kalita S, Deori R. Serum amylase and lipase activities in newly diagnosed patients with type 2 diabetes mellitus. Int J Adv Res. 2016;4(7):1476–1483. Available from: http://dx.doi.org/10.21474/IJAR01/996.
26. Vishwanath H, N S, C L. Biochemical analysis of serum amylase and lipase in patients with type 2 diabetes mellitus. Int J Clin Biochem Res. 2019;6(1):121–125. Available from: https://doi.org/10.18231/2394-6377.2019.0029.

27. Nakajima K. Low serum amylase and obesity, diabetes and metabolic syndrome: A novel interpretation. World J Diabetes. 2016;7(6):112–21. Available from: https://doi.org/10.4239/wjd.v7.i6.112.
28. Swislocki A, Noth R, Hallstone A, Kyger E, Triadafilopoulos G. Secretin-stimulated amylase release into blood is impaired in type 1 diabetes mellitus. Horm Metab Res. 2005;37(5):326–330. Available from: https://doi.org/10.1055/s-2005-861478.
29. Snehankar K, Rumi D, Risha G, Kailash B. Serum amylase and lipase activities in newly diagnosed patients with type 2 diabetes mellitus. Int J Adv Res. 2016;4(7):1476–83. Available from: http://dx.doi.org/10.21474/IJAR01/996.
30. Rakhee Y, Jaiprakash B, Sunilkumar V, Manojkumar N. The evaluation of serum amylase in the patients of type 2 diabetes mellitus, with a possible correlation with the pancreatic functions. J Clin Diagn Res. 2013;7(7):1291–4. Available from: https://doi.org/10.7860/jcdr/2013/6016.3120.
31. Newihi H, Dooley CP, Saad C, Staples J, Zeidler A, Valenzuela JE. Impaired exocrine pancreatic function in diabetics with diarrhea and peripheral neuropathy. Dig Dis Sci. 1988;33(6):705–710. Available from: https://doi.org/10.1007/bf01540434.
32. Sanvito F, Nichols A, Herrera PL, Huarte J, Wohlwend A, Vassalli JD. TGF- beta 1 overexpression in murine pancreas induces chronic pancreatitis and, together with TNF-alpha, triggers insulin-dependent diabetes. Biochem Biophys Res Commun. 1995;217(3):1279–86. Available from: https://doi.org/10.1006/bbrc.1995.2906.
33. Kobayashi T, Nakanishi K, Kajio H, Morinaga S, Sugimoto T, Murase T. Pancreatic cytokeratin: an antigen of pancreatic exocrine cell autoantibodies in type 1 (insulin-dependent) diabetes mellitus. Diabetologia. 1990;33(6):363–70. Available from: https://doi.org/10.1007/bf00404641.
34. Perfetti R, Wang Y, Shuldiner AR, Egan JM. Molecular investigation of age- related changes in mouse endocrine pancreas. J Gerontol A BiolSci Med Sci. 1996;51(5):331–367. Available from: https://doi.org/10.1093/gerona/51a.5.b331.

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