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Case-control study of serum amylase and serum lipase levels in type II diabetes mellitus

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Abstract

Background: Type II 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 the exocrine pancreas. Pancreatic β-cell dysfunction in diabetes can impair exocrine pancreatic function. In the present study, we examined the correlation between serum amylase and lipase levels in subjects with type II diabetes mellitus. As lipase is a specific marker of pancreatic inflammation, its correlation with amylase may indicate specific inflammation of pancreatic cells in type II diabetes mellitus.

Methods: A cross-sectional, observational, and analytical study was conducted among patients who visited the medicine OPD. The study included 100 subjects, comprising healthy controls (n = 50) and patients with diabetes (n = 50). Random Blood Sugar (RBS), serum amylase, and serum lipase levels were measured enzymatically. The collected data were analyzed using Student's t-test and Pearson's correlation in SPSS version 29.

Results: Serum amylase levels were significantly higher in the type II diabetes mellitus case group (149.63 ± 99.97 IU/L) than in the healthy control group (56.39±19.49) ($p < 0.001$). Serum lipase levels were also significantly higher in the type II diabetes mellitus group (163.22 ± 127.57 IU/L) than in the healthy control group (48.08 ± 9.31) ($p < 0.001$). A strong positive correlation was observed between serum amylase and lipase levels in the type II diabetes mellitus case group ($r = +0.22$), whereas the healthy control group showed almost no correlation ($r = +0.02$).

Conclusion: The study suggests that elevated serum amylase and lipase levels in patients with Type II diabetes mellitus indicate pancreatic exocrine dysfunction and are associated with pancreatic β-cell endocrine function. Monitoring pancreatic exocrine function in patients with type II diabetes is crucial for predicting, preventing, assessing, and managing associated complications.

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Highlights

What is current knowledge?

All studies have shown an association between pancreatic exocrine dysfunction and type II diabetes mellitus. However, these studies have also shown an association with the long-term complications of pancreatic cell inflammation caused by hyperglycemia.

What is new here?

As lipase is a specific marker of pancreatic inflammation, its correlation with amylase may indicate specific inflammation of pancreatic cells in type II diabetes mellitus. Therefore, we studied the correlation between serum amylase and lipase levels in Type II Diabetes Mellitus.

Introduction

Diabetes Mellitus (DM) is a metabolic disease caused by either a complete lack of insulin or reduced insulin sensitivity (1). Diabetes poses a threat to global health and will continue to do so. In the United Kingdom, at least 2% of the population (More than 1 million people) has diabetes, and the disease accounts for 5-10% of the overall healthcare budget (2). Given the high prevalence of diabetes among Indians, with over 20 million individuals affected-and a projected increase to 57 million by 2025-the disease could place a substantial burden on the country's healthcare system (3).

The pancreas is a mixed organ composed of approximately 84% exocrine tissue, 2% endocrine tissue, and 14% connective tissue (4). Because these two functional components are anatomically and physiologically interconnected, diseases affecting one compartment frequently affect the other (5,6).

The exocrine acinar cells are exposed to high concentrations of islet hormones because they receive blood from the surrounding islets (7). Several enzymes, including amylase and lipase, are produced by pancreatic exocrine acinar cells and facilitate the digestion of specific food components. During digestion, amylase is the primary enzyme that hydrolyzes starch into maltose, maltotriose, and alpha-limit dextrin. Lipase originates primarily from the pancreas, travels to the intestine, and facilitates the breakdown of triglycerides into fatty acids and monoglycerides. A deficiency of pancreatic enzymes prevents adequate food digestion, resulting in maldigestion and malnutrition (8).

For many years, low serum amylase levels associated with advanced pancreatic disease have been considered indicative of diffuse pancreatic damage. However, several studies have now shown that low serum amylase levels are also associated with metabolic syndrome and diabetes mellitus (9). A research study investigating the impact of hyperglycemia on pancreatic exocrine function in type 2 diabetic patients reported a substantial increase in serum amylase and lipase levels following improved glycemic control. Nevertheless, these levels remained lower than those of the control group. A strong negative association was identified between serum amylase and lipase levels and basal FBS and HbA1c (10).

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Insulin resistance results in increased activity of anti-insulin hormones and atrophy of exocrine acinar cells in diabetic patients. Consequently, the synthesis and secretion of exocrine pancreatic enzymes are reduced (11). With the onset of type 2 diabetes mellitus, coordination between the endocrine islets and the exocrine acinar cells also becomes disrupted. Over the long term, the exocrine pancreas responds to hormonal stimulation through fibrotic changes (12).

In diabetes mellitus, insulin resistance and/or hyperglycemia may lead to pancreatic exocrine dysfunction and pancreatic exocrine insufficiency (13). Pancreatic exocrine insufficiency (PEI) is defined as a deficiency of exocrine pancreatic enzymes that results in an inability to maintain normal digestion (14). The most common etiology of PEI is chronic pancreatitis. Prevalence of Pancreatic Exocrine Insufficiency (PEI) in Diabetes Mellitus: In recent decades, several studies have examined PEI in patients with diabetes mellitus.

In early studies, pancreatic exocrine function was assessed using the gold-standard direct pancreatic function test (Pancreozymin-secretin test) in 52.4 percent of cases. However, because direct pancreatic function tests are invasive, time-consuming, and costly, these trials have been limited to only a small number of patients. Exocrine insufficiency in diabetes mellitus can also lead to macronutrient deficiencies, steatorrhoea, and consequent malnutrition (15).

All studies have demonstrated an association between pancreatic exocrine dysfunction and type II diabetes mellitus. However, these studies have also shown an association with the long-term complications of pancreatic cell inflammation caused by hyperglycemia. As lipase is a specific marker of pancreatic inflammation, its correlation with amylase may indicate specific inflammation of pancreatic cells in type II diabetes mellitus. Therefore, we studied the correlation between serum amylase and lipase levels in Type II Diabetes Mellitus.

Methods

The present study was conducted in the OPD of the Department of Medicine and the Department of Biochemistry at D. Y. Patil Medical College, Kolhapur. The study was conducted over 3 months, from September 2025 to November 2025. Participants were randomly selected according to the inclusion and exclusion criteria. The study was explained to all participants, and written informed consent was obtained. Patient histories were recorded using the case record form. Under aseptic precautions, 10 ml of venous blood was collected from the antecubital vein into a 5 ml fluoride tube and a 5 ml plain tube. The samples were centrifuged at 3000 rpm for 15 min. The separated plasma was analyzed for blood sugar levels. The biochemical parameters, serum amylase and lipase, were measured using samples from the plain serum tubes and were analyzed immediately on a fully automated analyzer, Erba EM 200. The subjects were divided into two groups based on their Random Blood Sugar (RBS) levels: those with RBS greater than 200 mg/dl were classified as cases, and those with RBS less than 200 mg/dl were classified as controls.

A. Group I (Cases): 50 Type II diabetic patients with a disease duration of 5-10 years.
B. Group II (Healthy controls): 50 non-diabetic healthy controls.

Study design

In the present study, 50 patients with Type II diabetes were compared with 50 age-matched healthy control subjects. The subjects' ages ranged from 35 to 65 years.

Inclusion criteria

1. Patients with clinically diagnosed type 2 diabetes mellitus who were receiving oral antidiabetic drugs and had a disease duration of more than 5 years were included.
2. Patients who were not receiving insulin.
3. Participants who provided written consent were included in the study.

Exclusion criteria

1. Known cases of insulin-dependent diabetes mellitus (IDDM).
2. Patients with a known history of chronic illness, pregnancy, or chronic pancreatitis were excluded.

Plasma was collected to estimate Random Blood Sugar (RBS), and serum was collected to estimate serum amylase and lipase levels. The following clinical methods were used to measure the biochemical parameters:

1. Estimation of blood sugar by the GOD-POD method.
2. Estimation of serum amylase by the CNPG3, kinetic method.
3. Estimation of serum lipase by the Methyl resorufin, kinetic method.

Statistical analysis

Statistical analysis was performed using an unpaired Student's t-test in SPSS version 29.0. The results were expressed as mean ± SD. A p-value of less than 0.05 ($P<0.05$) was considered significant.

Results

Various parameters were evaluated in group I and group II (Table 1). The results showed that serum amylase and lipase levels were higher in group I than in group II.

Table 1. Comparison of demographic and laboratory variables in people with type II diabetes and healthy controls

Characteristics	Groups		P-value
	Type II diabetic patients (n=50)	Healthy controls (n=50)	
	Mean ± SD	Mean ± SD	
Age, Years	42.46±12.90	43.34±12.10	0.11
RBS	175.75±23.53	98.49.2±18.26	< 0.05
Amylase	149.63±99.97	56.39±19.49	<0.001
Lipase	163.22±127.57	48.08±9.31	< 0.001

Values were expressed as Mean±SD

The correlation between serum amylase and serum lipase levels in the healthy and Type II diabetes groups, as depicted in Table 2, indicates that the healthy group showed a correlation of (r= +0.02), whereas the Type II diabetes group showed a correlation of (r= +0.22; Figure 1).

Table 2. Correlation between serum amylase and serum lipase in the healthy and Type II diabetes groups

Enzymes	Control (N=50)	Type II diabetes (N=50)
Serum amylase	1	1
Serum lipase	0.02	0.22

*P < 0.001 - Highly Significant.
r= +0.02 in the healthy group; r= +0.22 in the Type II diabetes group

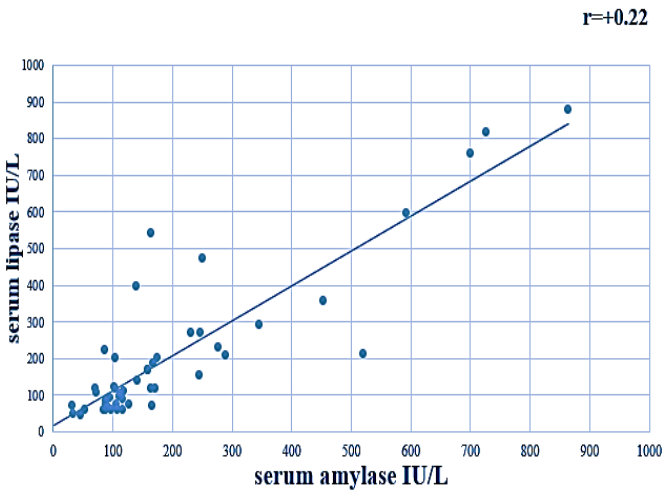

Figure 1. Correlation between serum amylase and serum lipase in group I (Type II diabetes)

Discussion

In this study, 50 subjects with Type II diabetes (Group I) and 50 healthy controls (Group II) were included. Table 1 shows that age, RBS, amylase, and lipase levels differed between the groups. Age did not differ significantly between the groups, as the p-value for group I and group II were $p = 0.11$ ($p > 0.05$). Random Blood Sugar levels were significantly elevated in subjects with Type II diabetes compared with healthy controls ($p < 0.05$). In group I, subjects with type II diabetes had insulin resistance, resulting in decreased glucose tolerance and increased blood sugar levels, thereby leading to a hyperglycemic state (16).

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Serum amylase levels were significantly higher in subjects with Type II diabetes than in healthy controls ($p < 0.001$). This may indicate an early stage of impaired pancreatic function. This finding was consistent with the observations of Bastyr et al. (2009) (17) and Steinberg et al. (2014) (18), who also reported elevated amylase and lipase activity in subjects with Type II diabetes mellitus. However, Rakhee Yadav et al. (2013) (19) and Mahesh Basavaraj Madole et al. (2016) (20) reported decreased amylase and lipase activity. This discrepancy might be attributable to differences in the duration of diabetes among the study subjects. Persistent hyperglycemia in long-standing diabetes with poor glycemic control may cause acinar cell damage, leading to pancreatic fibrosis and atrophy and, consequently, exocrine acinar cell insufficiency (21), which may have resulted in reduced amylase and lipase activity in their studies. The cause of increased pancreatic enzyme levels in type II diabetes mellitus remains unknown and requires further investigation.

Our findings (Table 2) showed that serum amylase and serum lipase levels were weakly positively correlated in the healthy control group ($r = +0.02$), whereas these levels (Table 2 and Figure 1) were significantly higher in the type II diabetic subject groups than in the controls. A positive correlation was observed between serum amylase and lipase levels in the group of subjects with type II diabetes ($r = +0.22$). A possible mechanism underlying the increased amylase and lipase levels may be insulin resistance in diabetes, characterized by increased BSL and compensatory hyperinsulinemia. This condition may also enhance pancreatic exocrine function by stimulating the islet-acinar axis, thereby increasing pancreatic enzyme production (22).

In our study, we did not consider age, sex, BMI, or the duration of diabetes, and glycated hemoglobin (HbA1c), insulin levels, and C-peptide were not measured. Future researchers are advised to consider the above parameters in their studies.

Conclusion

The study revealed elevated serum amylase and lipase levels in subjects with Type II diabetes mellitus, indicating that these elevated levels may reflect enzyme leakage, mild inflammation, or metabolic stress affecting true exocrine dysfunction. This is likely attributable to the close relationship between pancreatic β-cell function and exocrine function. Hyperglycemia and insulin resistance may contribute to pancreatic inflammation and damage, leading to increased enzyme leakage into the bloodstream. These findings suggest that pancreatic exocrine function should be monitored regularly in patients with Type II diabetes to prevent and manage associated complications.

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Ethical Statement

This study was approved by the Institutional Ethics Committee of D. Y. Patil Medical College and Hospital.

Conflicts of Interest

None declared.

Author Contributions

The first author and corresponding authors contributed to the conception and design of the study, data collection, and manuscript preparation. The second author contributed to data analysis and interpretation.

Data Availability Statement

All data generated or analyzed during this study are included in this published article and its supplementary information files.

Use of Artificial Intelligence

We declare that no generative AI or AI-assisted technologies were used in the writing process of this manuscript.

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