

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Study of Serum Pancreatic Enzymes in Patients with Type 2 Diabetes Mellitus.

P Ravisekar*, VS Kalai Selvi , AJ Manjula Devi, and B Shanthi.

Sree Balaji Medical College & Hospital, Bharath University, Chrompet, Chennai – 44, Tamil Nadu, India.

ABSTRACT

Many researches in Diabetes give more focus on dyslipidemia as a major factor for cardiac, cerebral and renal complications. A few clinical studies explain the impaired exocrine-endocrine relationship in the pancreas. Serum pancreatic enzyme levels may reflect the impaired exocrine-endocrine relationship in the pancreas. This study is planned to investigate pancreatic enzymes level in association with the pathogenesis of impaired insulin action. To investigate the pancreatic exocrine insufficiency in patient with diabetes mellitus by estimating serum pancreatic amylase and lipase enzymes in healthy subjects and in type 2 diabetic patients. The present study was conducted on 50 participants, out of which 30 were diabetic 3-5years duration ,attending the O.P.D. of Sree Balaji Medical college and Hospital & 20 healthy age & sex matched controls. After obtaining the informed consent from the subjects, the fasting venous blood samples were collected by standard aseptic techniques. Serum was separated for the various assays. Fasting blood sugar (FBS), Glycosylated hemoglobin (HbA1C), pancreatic amylase and lipase enzymes were analysed in mind ray fully automated analyser. Data collected were analysed using SPSS package. The FBS and HbA1C estimates were consistently higher in type 2 diabetic patient. Decreased serum pancreatic amylase and Lipase was recorded in type 2 diabetes. Correlation of the decrease in serum amylase and lipase levels in type 2 diabetes was higher in patients with longer duration of illness. The present study clearly explains decrease in serum amylase and lipase levels in type 2 diabetes .We suggest that analysis of serum pancreatic enzymes could be an additional informative parameter for the assessment of chronicity and progress of the illness as well as the response to therapy.

Keywords: FBS, HBA1C, Pancreatic Enzymes, Type2 Diabetes Mellitus.



*Corresponding author



INTRODUCTION

Most of researches in Diabetes give more focus on dyslipidemia as a major factor for cardiac, cerebral and renal complications [1,2]. A few clinical studies explain the impaired exocrine-endocrine relationship in the pancreas. Serum pancreatic enzyme levels may reflect the impaired exocrine-endocrine relationship in the pancreas [3-5]. This study is planned to investigate pancreatic enzymes level in association with the pathogenesis of impaired insulin action.

Aim and Objective

To investigate the pancreatic exocrine insufficiency in patient with diabetes mellitus by estimating serum pancreatic amylase and lipase enzymes in healthy subjects and in type 2 diabetic patients.

MATERIALS AND METHODS

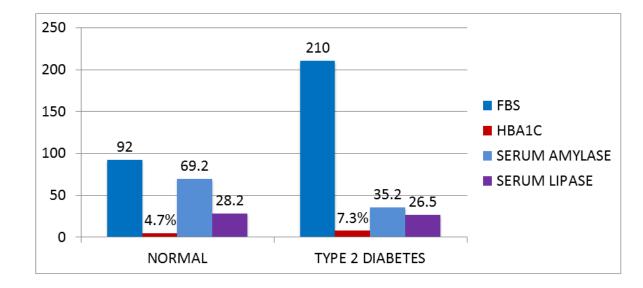
The present study was conducted on 50 participants, out of which 30 were diabetic 3-5years duration, attending the O.P.D. of Sree Balaji Medical College and Hospital & 20 healthy age & sex matched controls. After obtaining the informed consent from the subjects, the fasting venous blood samples were collected by standard aseptic techniques. Serum was separated for the various assays. Fasting blood sugar (FBS), Glycosylated hemoglobin (HbA1C), pancreatic amylase and lipase enzymes were analysed in mind ray fully automated analyser .Data collected were analysed using SPSS package.

RESULTS

The mean values of FBS and HBA1C in Type 2 DM patients were 210 ± 15 mg/dl and $7.3\pm1.5\%$ respectively. Their Serum Amylase and lipase levels were 35.2 ± 2.3 IU/L and 26.5 ± 1.2 U/L.The mean values of FBS and HBA1C in control were 92 ± 12 mg/dl and $4.7\pm1.2\%$ respectively. Their Serum Amylase and lipase levels were 69.2 ± 3.1 IU/L and 28.2 ± 2.1 U/L respectively as shown in the table 1. On comparison with control group, the Serum amylase and lipase levels show significant decrease type 2 Diabetic patients. Significantly low serum Amylase and Lipase levels were found in the diabetic patients as compared to those in the healthy controls (p value <0.001). Correlation of the decrease in serum Amylase and Lipase levels in type 2 diabetes was higher in patients with longer duration of illness.

	FBS(mg/dl)	HBA1C%	SERUM AMYLASE(IU/L)	SERUM LIPASE(U/L)
CONTROL	92±12	4.7±1.2	69.2±3.1	28.2±2.1
TYPE 2 DIABETES	210±15	7.3±1.5	35.2±2.3	26.5±1.2

Table 1: pancreatic enzmes and HbA1c levels in type2 DM and controls.





DISCUSSION

Mechanisms linking exocrine pancreatic insufficiency might be the impairment of entero-pancreatic reflexes or changes in gastrointestinal peptides. Most of epidemiological studies indicate that serum amylase levels are inversely associated with most cardio metabolic risk factors, especially those associated to obesity. the serum amylase level decreased with worsening of metabolic abnormalities and these declines were independent of smoking, a strong factor for increasing insulin resistance. Regarding the cause-effect relationship, low serum amylase levels were believed to be due to deficient insulin activity. The finding of decreased enzyme level thought to be due to reduced acinar cell function in the vicinity of insulin-depleted islets [6, 7]. Similar reductions in the pancreatic amylase, lipase, trypsin and elastase have been recorded by Lorini et al [8], Yajnik et al [9] and Kim et al [10]. Besides these putative mechanisms, there are multiple defects in insulin secretion and signaling in type 2 diabetes, which might be associated with the low amylase secretion from the pancreas.

CONCLUSION

The present study clearly explains decrease in serum amylase and lipase levels in type 2 diabetes . We suggest that analysis of serum pancreatic enzymes could be an additional informative parameter for the assessment of chronicity and progress of the illness as well as the response to therapy.

REFERENCES

- [1] Thomas GN, Critchley JA, Tomlinson B, Cockram CS, Chan JC. Diabet Med 2003; 20: 988-995.
- [2] Hadjadj S, Duly-Bouhanick B, Bekherraz A, Brldoux F,Gallois Y, Mauco G, et al. Diabetes Metab 2004; 30: 43-51.
- [3] Di Magno EP, Go VL, Summerskill WH. N Engl J Med 1973;288(16):813-5.
- [4] Fieker A, Philpott J, Armand M. Clin Exp Gastroenterol 2011;4:55-73.
- [5] Leeds JS, Oppong K, Sanders DS. Nat Rev Gastroenterol Hepatol 2011;8(7):405-15.
- [6] Korc M, Owerbach D, Quinto C, Rutter WJ. Science 1981; 213:351-353.
- [7] Adler G, Kern HF. Horm Metab Res 1975; 7: 290-296.
- [8] Lorini R, Cortona L, Scotta MS, Melzi d'Eril GV, Severi F. Diabetes Res Clin Pract 1990; 8: 263-267.
- [9] Yajnik CS, Sahasrabudhe RA, Nail SS, Katrak A, Shelgikar KM, Kanitkar SV, et al. Pancreas 1990;5: 631-638.
- [10] Kim KH, Lee HS, Kim CD, Chun HJ, Song CW, Um SH, et al. J Clin Gastroenterol 2000; 31: 51-54.