International Journal of Sciences: Basic and Applied Research (IJSBAR)

ISSN 2307-4531
(Print & Online)

http://gssrr.org/index.php?journal=JournalOfBasicAndApplied

Lipoprotein Lipase, Amylase and Triglyceride Alteration: Lean Diabetic and Obese (Non-Diabetic) Patients

Dr. Kamal Eldin Ahmed Abdelsalam*

Department of CLS, Shaqra University, KSA

kamaleldin55@yahoo.com

Abstract

Obesity, as well as type 2 diabetes mellitus, is a significant issue for health policy because it is so widespread in the population as a whole, and because of the high risk of complications it carries. Serum lipase and pancreatic amylase concentrations are used in conjunction with clinical findings to diagnose pancreatic disorders. The aim of this study was to differentiate the alterations in fasting dyslipidemia and serum levels of pancreatic enzymes in lean diabetics and non-diabetic obese. Triglyceride, lipase and amylase were measured in the serum collected from overnight fasting 300 volunteers those were separated into 3 groups; control group (non-diabetic and with normal weight), diabetics (normal weight) group and obese (non-diabetic) group. In this study, serum levels of triglyceride, amylase and lipase were significantly changed in obese group when compared to control group. But, only of triglyceride and amylase were increased significantly in diabetic group when compared to control group. On the other hand, serum lipase showed significant increase diabetic group when compared to obese group. In conclusion, serum triglyceride and amylase concentrations were higher in obese subjects than those with normal weight. The results of this study illustrated that serum lipase concentrations were decreased in obese subjects but increased in diabetic patients compared to the normal healthy subjects.

Keywords: Lipase; amylase; triglyceride; obese; diabetes mellitus

---

* Corresponding author.

E-mail address: kamaleldin55@yahoo.com.

1. Introduction

Obesity was not seen until the 20thcentury that it became common. People become obese for several reasons, including eating of much more than they used to. This used to be the case just in developed nations - however, the trend has spread worldwide. Also with the arrival of televisions, computers, video games, remote controls, washing machines, dish washers and other modern convenience devices, the majority of people are leading a much more sedentary lifestyle compared to their parents and grandparents [1]. In 1997 the World Health Organization (WHO) formally recognized obesity as a global epidemic. In June of 2013 the American Medical Association classified obesity as a disease with much controversy [2]. The rise in obesity has led to widespread calls for regular monitoring of changes in overweight and obesity prevalence in all populations. Comparable, up-to-date information about levels and trends is essential to quantify population health effects and to prompt decision makers to prioritize action [3]. Obesity is difficult to treat and has a high relapse rate. Most people who lose weight regain the weight within five years. Obesity was recently described as a risk factor of pancreatic cancer in combination with metabolic abnormalities [4].

Type 2 diabetes mellitus consists of an array of dysfunctions characterized by hyperglycemia and resulting from the combination of resistance to insulin action, inadequate insulin secretion, and excessive or inappropriate glucagon secretion. Poorly controlled type 2 diabetes is associated with an array of micro-vascular, macro-vascular and neuropathic complications [5].

Amylase (a-I. 4-glucan 4-glucanohydrolase, EC 3.2.1.1) is an enzyme that catalyzes the hydrolysis of starch into sugars. Amylase is present in the saliva of humans and some other mammals, where it begins the chemical process of digestion. The pancreas and salivary gland make amylase (alpha amylase) to hydrolyze dietary starch into disaccharides and polysaccharides which are converted by other enzymes to glucose to supply the body with energy [6].

A lipase (glycerol ester hydrolase, EC 3.1.1.3) is an enzyme that catalyzes the hydrolysis of fats (lipids). Lipases perform essential roles in the digestion, transport and processing of dietary lipids. Human pancreatic lipase (HPL) is the main enzyme that breaks down dietary fats in the human digestive system, converts ingested triglyceride to mono-glycerides and two fatty acids. Some lipase activities are confined to specific compartments within cells while others work in extracellular spaces [7].

Amylase and lipase are enzymes produced by the pancreas that help to digest food. Determination of serum lipoprotein lipase activity has been advocated as a tool for diagnosing pancreatitis for several decades[6], but serum amylase activity determination continues to be more popular for this purpose because of confusion in the literature on the meaning of the term, “lipase,” and on the diagnostic usefulness of various types of “lipase” data[6, 7].The interpretation of a high amylase or lipase can be complex. The clinical history is critical. Asymptomatic patients should be thoroughly screened for conditions like chronic alcohol abuse, renal failure, pancreatic cancer, and other non-pancreatic conditions [8].

2. Materials and Methods

2.1. Study design:

It was a cross-sectional study.

2.2. Study area:

This study was conducted in Khartoum state, Sudan. The samples for this study were collected from Dr. Isam diabetes center, Al-Ajyal hospital, Al-Afaf Specialized Clinics and Dr. Amel Hospital for Obstetrics and Gynaecology, and Alribat hospital.

2.3. Study period

The study was commenced in November, 2012 and ended in July, 2014.

2.4. Sample size and study population

The study included 100diabetic patients with normal weight (50 males and 50 females) with mean age of 52.5± 8.3 years, and 100 obese (non-diabetic) volunteers (50 males and 50 females) with mean age of 49.5 ± 9.1 years. The control group consisted of 100 healthy non-diabetic volunteers with normal weight (50 males and 50 females) whose mean ages were matched (50.5 ± 9.8 years).

2.5. Sampling

Venous blood samples were withdrawn from all subjects after 10 hours overnight fasting. 10 ml venous blood was obtained from antecubital vein by standard venipuncture techniques without venous stasis[9], in SST gel separated serum tubes. Following collection, samples were left to clot and were centrifuged for 5 min at 15000 RPM and analyzed immediately after separation. Some serum was separated and stored at -20°C for further use.

2.6. Methods of estimation of serum amylase and serum lipoprotein lipase levels

In this study, chemicals and reagents used for estimation were obtained from Mindray Company China, and the measurement was carried by chemistry auto analyzer (Mindray BS-200E Chemistry Analyzer).Serum amylase was measured using ELISA methods as described by [10]; while serum lipoprotein lipase was measured using a sandwich ELISA following the methods described by Yang *and his colleagues* [11].

2.7. Methods of body mass index (BMI) estimation [12]:

The BMI calculates a value indicative of the fat content of the body by dividing the body weight by the square of body height.

2.8. Ethical clearance

An ethical clearance of this study was approved by the ethical committee of Omdurman Islamic University. Informed consent was obtained from each participant before taking the samples.

2.9. Statistical analyses of data

The data obtained were expressed as mean values ± SD. Statistical analyses were performed using SPSS (Statistical Package for Social Sciences) version 11.5. Differences in mean values between groups were evaluated by a Student's t-test. P-value was statistically significant at $P<0.05$.

Table 5: Criteria for classification of participants based on BMI

Categories	***BMI***
Underweight	Less than 18.5
Normal weight	18.5 - 24.9
Overweight	25 - 29.9
Obese	30 or higher

3. Results

This study included three groups, those were obese, diabetic (DM) and the control groups, each group comprised of 100 volunteers. All groups were in matched ages. Also, there were matches in duration between obese and diabetic groups. And also, there was a matching between control and diabetic groups, where both groups were in the normal weight (table 1).

Table 1: Demographic and clinical features of subjects

		Control	Obese	DM
Number		100	100	100
Gender	Males	50	50	50
	Females	50	50	50
Age		50.5 ± 9.8	49.5 ± 9.1	52.5± 8.3[a]
Duration (years)		0	6 ± 3.9	6.5 ± 4[b]
BMI (kg/m 2)		22.3 ± 4.1	34.7 ± 6.8	24.7 ± 9.6[c]

[a] insignificant variation between all groups; [b] insignificant variation between obese and diabetic groups; [c] insignificant variation between control and diabetic groups.

As in table 2, the serum levels of triglyceride, amylase and lipase were significantly changed in obese group when compared to control group.

Table 3 showed that serum levels of triglyceride and amylase were significantly increased in diabetic group when compared to control group.

In table 4, serum lipase in diabetic group showed significant increase when compared to obese group.

Table 2: comparison of triglyceride, amylase and lipase levels between obese and control groups

Groups	Triglycerides(mg/dl)	Amylase (U/L)	Lipase (U/L)
Control	92.5 ± 10.1	42.1 ± 12.7	83 ± 12.7
Obese	173 ± 11.1	110 ± 18.1	56.1 ± 18.5
P value	0.000	0.000	0.046

Table 3: comparison of triglyceride, amylase and lipase levels between diabetic and control groups

Groups	Triglycerides(mg/dl)	Amylase (U/L)	Lipase (U/L)
Control	92.5 ± 10.1	42.1 ± 12.7	83 ± 12.7
Diabetes	169 ± 10.3	89.6 ± 9.5	95.3 ± 54
P value	0.010	0.000	0.088

Table 4: comparison of triglyceride, amylase and lipase levels between obese and diabetic groups

Groups	Triglycerides(mg/dl)	Amylase (U/L)	Lipase (U/L)
Obese	173 ± 11.1	110 ± 18.1	56.1 ± 18.5
Diabetes	169 ± 10.3	89.6 ± 9.5	95.3 ± 54
P value	0.104	0.091	0.002

4. Discussion

Most of the metabolic abnormalities associated with obesity have been shown to normalize with weight loss and thus are likely a consequence of the obese state, not a cause [1]. While diabetes mellitus is known to have a

deleterious metabolic abnormalities, that eventually contribute to diabetes-associated vascular complications [5]. There is a close association between obesity and type 2 diabetes. The likelihood and severity of type 2 diabetes are closely linked with body mass index (BMI) [3].

In the present study, the results of serum triglyceride and serum amylase were increased significantly in obese group when compared to control group (p value = 0.000); while serum lipase level was decreased significantly in obese group comparing to control (p value<0.05). In a previous study, Malloy *and his colleagues* [13] reported that results of amylase and lipase increased in the serum of obese persons may be due obesity and elevated triglyceride in the blood.

The results of the present study also showed that the serum lipase levels were insignificantly increased in diabetic group than those in control group (p value>0.05). On the other hand, in diabetic group, the serum results of triglyceride and amylase were increased significantly (p value<0.05). These results were consistent with report of Tirosh *and his colleagues* [14] showing that elevated triglyceride levels are a common dyslipidemic feature accompanying type 2 diabetes and pre-diabetic states. Also, Yang *and his colleagues* [11] reported that the type 2 diabetic population is likely to have pancreatic enzyme elevations.

Our results suggest that relatively higher LPL may be associated with type 2 diabetes in lean patients in the background of elevated triglyceride and serum amylase. The explanation of this could be related to low leptin level in lean patients, which related to distribution of body fat. For a given BMI, central rather than lower body fat distribution, confers greater risk of metabolic and cardiovascular complications of obesity [15]. Intra-abdominal fat tissues secrete less leptin than subcutaneous fat tissue [16]. In addition, Sayeed *and his colleagues* [17] stated that leptin is reduced in lean subjects with type 2 diabetes in Bangladesh. Furthermore, Obesity is typically associated with high leptin levels and not leptin deficiency [18]. Studies investigated effect of leptin on diabetes concluded that long-term leptin replacement therapy improves glycemic control [19-21].

Serum triglyceride and amylase levels showed insignificant increase in obese group more than diabetic patient in this study (p value>0.05); while serum lipase level was decreased significantly in obese group when compared to diabetics (p value<0.05). Gajda *and his colleagues* [22] published that triglyceride was increased significantly in obese subjects more than in diabetic patients. Such results were contrary to what found in this study about triglycerides. Also, the results of the present study showed that amylase concentration was insignificantly increased in obese subjects more than in diabetic subjects (p value>0.05). In contrast, the lipase enzyme concentration was found to be increased significantly in diabetic group more than obese group (p value<0.05). Bojadzievski *and his colleagues* [23] reported that although the direct mechanism by which diabetes increases the susceptibility to higher pancreatic enzyme concentrations is unknown, insulin resistance and hyperglycemia appear to be important factors linked to the increase in pancreatitis in individuals with type 2 diabetes.

So, this research is recommended to maintain the ideal weight due to problems facing obese persons; on the other hand, the diabetic patients should keep the blood glucose within the normal rate and care to reduce weight as much as possible.

5. Conclusion

Overall, obese subjects had higher serum triglyceride and amylase concentrations than those with normal weight. The results of this study displayed that serum lipase concentrations were decreased in obese subjects but increased in diabetic patients comparing to the control.

Conflict Of Interest

We declare that we have no conflict of interest.

Acknowledgement

Authors are grateful to the V R Center to provide automated analyzer and its reagents. Authors acknowledge the immense help received from the scholars whose articles are cited and included in references of this manuscript. The authors are also grateful to authors / editors / publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed.

References

[1] M. I. Yasawy, A. A. Al-Quorain, A. M. Hussameddin, Z. M. Yasawy, and R. M. Al-Sulaiman, "Obesity and gastric balloon," *J Family Community Med*, vol. 21, pp. 196-9, Sep 2014.

[2] D. Haslam, "Obesity: a medical history," *Obesity Reviews*, vol. 8, pp. 31-36, 2007.

[3] A. Abdullah, A. Peeters, M. de Courten, and J. Stoelwinder, "The magnitude of association between overweight and obesity and the risk of diabetes: a meta-analysis of prospective cohort studies," *Diabetes Res Clin Pract*, vol. 89, pp. 309-19, Sep 2010.

[4] V. Rebours, S. Gaujoux, G. D, A. Sauvanet, P. Ruszniewski, P. Levy, *and his colleagues* ., "1044 Obesity Is a Risk Factor for Pancreatic Precancerous Lesions," *Gastrointestinal Endoscopy*, vol. 79, p. AB191.

[5] A. L. Schneider, M. Lazo, C. E. Ndumele, J. S. Pankow, J. Coresh, J. M. Clark, *and his colleagues* ., "Liver enzymes, race, gender and diabetes risk: the Atherosclerosis Risk in Communities (ARIC) Study," *Diabet Med*, vol. 30, pp. 926-33, Aug 2013.

[6] S. Oppliger, S. Hartnack, B. Riond, C. E. Reusch, and P. H. Kook, "Agreement of the serum Spec fPL and 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6'-methylresorufin) ester lipase assay for the determination of serum lipase in cats with suspicion of pancreatitis," *J Vet Intern Med*, vol. 27, pp. 1077-82, Sep-Oct 2013.

[7] A. Mahajan, R. Kadavigere, S. Sripathi, G. S. Rodrigues, V. R. Rao, and P. Koteshwar, "Utility of serum pancreatic enzyme levels in diagnosing blunt trauma to the pancreas: A prospective study with

systematic review," *Injury,* vol. 45, pp. 1384-93, Sep 2014.

[8] M. F. Philippe, S. Benabadji, L. Barbot-Trystram, D. Vadrot, C. Boitard, and E. Larger, "Pancreatic volume and endocrine and exocrine functions in patients with diabetes," *Pancreas,* vol. 40, pp. 359-63, Apr 2011.

[9] S. M. Grundy, H. B. Brewer, Jr., J. I. Cleeman, S. C. Smith, Jr., and C. Lenfant, "Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition," *Arterioscler Thromb Vasc Biol,* vol. 24, pp. e13-8, Feb 2004.

[10] M. Nassar, N. Hiraishi, M. S. Islam, M. Otsuki, and J. Tagami, "Age-related changes in salivary biomarkers," *Journal of Dental Sciences,* vol. 9, pp. 85-90.

[11] L. Yang, S. J. Chen, G. Y. Yuan, L. B. Zhou, D. Wang, X. Z. Wang, *and his colleagues* ., "Association of serum adipose triglyceride lipase levels with obesity and diabetes," *Genet Mol Res,* vol. 13, pp. 6746-51, 2014.

[12] N. J. MacKay, "Scaling of human body mass with height: the body mass index revisited," *J Biomech,* vol. 43, pp. 764-6, Mar 3 2010.

[13] J. Malloy, K. Gurney, K. Shan, P. Yan, and S. Chen, "Increased variability and abnormalities in pancreatic enzyme concentrations in otherwise asymptomatic subjects with type 2 diabetes," *Diabetes Metab Syndr Obes,* vol. 5, pp. 419-24, 2012.

[14] A. Tirosh, I. Shai, R. Bitzur, I. Kochba, D. Tekes-Manova, E. Israeli, *and his colleagues* ., "Changes in triglyceride levels over time and risk of type 2 diabetes in young men," *Diabetes Care,* vol. 31, pp. 2032-7, Oct 2008.

[15] N. Freemantle, J. Holmes, A. Hockey, and S. Kumar, "How strong is the association between abdominal obesity and the incidence of type 2 diabetes?," *Int J Clin Pract,* vol. 62, pp. 1391-6, Sep 2008.

[16] V. Van Harmelen, S. Reynisdottir, P. Eriksson, A. Thorne, J. Hoffstedt, F. Lonnqvist, *and his colleagues* ., "Leptin secretion from subcutaneous and visceral adipose tissue in women," *Diabetes,* vol. 47, pp. 913-7, Jun 1998.

[17] M. A. Sayeed, A. K. Azad Khan, H. Mahtab, K. A. Ahsan, A. Banu, P. A. Khanam, *and his colleagues* ., "Leptin is reduced in lean subjects with type 2 diabetes in bangladesh," *Diabetes Care,* vol. 26, p. 547, Feb 2003.

[18] R. S. Ahima, "Revisiting leptin's role in obesity and weight loss," *J Clin Invest,* vol. 118, pp. 2380-3, Jul 2008.

[19] R. Coppari and C. Bjorbaek, "Leptin revisited: its mechanism of action and potential for treating diabetes," *Nat Rev Drug Discov*, vol. 11, pp. 692-708, Sep 2012.

[20] M. Naito, J. Fujikura, K. Ebihara, F. Miyanaga, H. Yokoi, T. Kusakabe, *and his colleagues* ., "Therapeutic impact of leptin on diabetes, diabetic complications, and longevity in insulin-deficient diabetic mice," *Diabetes*, vol. 60, pp. 2265-73, Sep 2011.

[21] F. Picard, D. Richard, Q. Huang, and Y. Deshaies, "Effects of leptin adipose tissue lipoprotein lipase in the obese ob/ob mouse," *Int J Obes Relat Metab Disord*, vol. 22, pp. 1088-95, Nov 1998.

[22] K. Gajda, A. Sulich, J. Hamulka, and A. Bialkowska, "Comparing diabetic with non-diabetic overweight subjects through assessing dietary intakes and key parameters of blood biochemistry and haematology," *Rocz Panstw Zakl Hig*, vol. 65, pp. 133-8, 2014.

[23] T. Bojadzievski and R. A. Gabbay, "Patient-centered medical home and diabetes," *Diabetes Care*, vol. 34, pp. 1047-53, Apr 2011.