

## **Assessment of Gamma glutamyl Transferase Activity among Type-2 Diabetic patient in Sulaimani Governorate**

*Hamid Ghaffoori Hasan<sup>1</sup> and Fenk Bakir Maarouf<sup>2</sup>*

*1 . Baghdad University, Ibn Al-Haitham college, Baghdad – IRAQ.*

*Email : [gaforiiq@yahoo.co.uk](mailto:gaforiiq@yahoo.co.uk)*

*2.Ministry of Health, General Hospital, Central Lab, Sulaimani – IRAQ.*

---

### **Abstract**

Increased levels of the liver enzyme, gamma glutamyl transferase (GGT) has been found to be associated with diabetes. Anthropometric measurement concerning GGT activity as well as body mass index and family history of the volunteers were investigated in sera of 115 (58 male and 57 female) patients with type-2 diabetes mellitus against 60 (34 male and 26 female) as controls. A questionnaire form was used for collection characteristics of the patients and SPSS (version-16) as statistic tool analysis was applied to analyze the data obtained. The results obtained revealed a significant elevation ( $P<0.05$ ) in GGT activity among diabetics ( $14.2\pm 11.1$  IU/L) as compared with control ( $10.5\pm 8.0$ ). The results were also showed a significant elevation ( $P<0.05$ ) in diabetic patients with positive family history when compared with control. Gamma glutamyl transferase activity was found to be associated significantly with body mass index factor among diabetics. The study was conducted in Sulaimani Center for Diabetics and Endocrine Diseases from June through December 2010.

---

Keywords: Serum GGT, BMI, Family history, Type-2 DM.

### **Introduction**

Diabetes mellitus (DM) is a group of metabolic diseases, characterized by hyperglycemia resulting from defects in insulin secretion,

insulin action, or both,[ADA, 2010]. Type 2 DM is caused by a combination of resistance to insulin action and an inadequate compensatory insulin secretory response. This form of DM,

accounts for approximately 90 - 95% of those with DM and was previously referred to as non-insulin dependent diabetes mellitus (NIDDM), or adult-onset DM [ADA, 2010]. Some studies linked oxidative stress (OS) and inflammation to  $\beta$ -cell dysfunction resulting from chronic exposure to hyperglycemia, free fatty acid, or a combination of the two [Warram *et al*, 1990; Ceriello, 2005]. A growing body of data reinforces the concept that inflammation also plays an important role in the pathogenesis of type 2 DM [Ford, 2003; Nakanishi *et al*, 2003]. Gamma-glutamyl transferase (GGT) (E.C.2.3.2.2) activity, normally found in serum as well as in the plasma membrane of virtually all cells except erythrocytes, catalyzes the first step in the degradation of extracellular glutathione (GSH), allowing for precursor amino acids to be assimilated and reutilized for intracellular GSH synthesis [Whitfield, 2001]. Thus, GGT activity favors the cellular supply of GSH, the most important non-protein antioxidant of the cell. However, there is also clear evidence that the degradation of GSH can play a pro-oxidant role [Emdin *et al*, 2005]. Gamma-glutamyl transferase is therefore thought to have a role in oxidative mechanisms and is regarded as an early and sensitive marker of oxidative stress [Lim *et al*, 2004; Onat, 2006; Sharma, 2010]). Increased levels of the liver enzymes, Gamma-glutamyl transferase (GGT), Alanine aminotransferase (ALT) and Aspartate aminotransferase (AST) are known to be

markers of NAFLD [Perry *et al*, 1998; Wannamethee *et al*, 2005]. They have been found to be associated with diabetes, cardiovascular risk factors and insulin resistance syndrome, even within normal reference intervals [Nilssen and Forde, 1994; Sakugawa *et al*, 2004; Liu *et al*, 2005]. In many prospective studies, strong relationships between GGT or ALT concentrations and incident diabetes have also been observed in non-drinkers, in individuals with normal levels of liver enzymes, independently of classical cardiovascular risk factors [Vozarova *et al*, 2002; Sattar *et al*, 2004, Hanley *et al*, 2004]. However, a strong interaction between body mass index (BMI) and GGT has been described in diabetes [Lee *et al*, 2003a; Nakanishi *et al*, 2003]. A family history of diabetes mellitus (FHDM) is an established risk factor for type 2 diabetes, and is used as a convenient, first-line screening tool for diabetes [Harrison *et al*, 2003]. Several researchers have reported that increased GGT is independently associated with increased risk of type 2 diabetes in Asian and Caucasian populations [Lee *et al*, 2004a; Andre *et al*, 2005; Wannamethee *et al*, 2005 ]. Up to researcher's knowledge, no previous study had been conducted about GGT among diabetic patients neither in Sulaimani nor in Kurdistan region and the rest of Iraq. The aims of the present study are to measure the levels of serum liver enzyme GGT among known type 2 diabetic subjects, to evaluate the effect of

gender, BMI, on the GGT among the studied sample, and find the relation of GGT with family history of diabetes.

### Materials and Methods

This is a case control study that has been conducted between June and December 2010 at the Sulaimani Centre for Diabetes and Endocrine diseases and the Central laboratory of Directory of health. Ethical Consideration of the study was approved by the scientific committee of the Directorate of Health. Permission was obtained from administrative authorities of Sulaimani Diabetic Center and Central laboratory prior to data collection. The detail of the work was explained for the participant and a verbal consent was taken from each person.

### Chemicals

Commercial available kit was used to measure GGT (Biolabo, SA, Maizy, Franc). Materials used (not included in the kit) are all other chemicals and they were of analar grades.

### Specimen collection

One hundred and fifteen diabetic patients (115) distributed as 58 males and 57 females, their age ranged between 32-78 years, were recruited in the study during their visit to the Sulaimani Centre for Diabetes and Endocrine disease. They were selected by random selection. They were diagnosed by consultants. For the comparative purpose, a comparable group (60

samples) which constitute of 34 males and 26 females, apparently healthy, non diabetic subjects from health workers, their age ranged between 31-68 years, were voluntarily included in the study. Samples included were characterized by the followings:

1. Adult aged 30 years and above. ( both Diabetics and Control).
2. Patients diagnosed as diabetics by physician or have booklet.

(According to ADA-2010 criteria).

3. Overnight fasting, about 8 hours before taking the blood sample.

Samples excluded from the study were of:

1. History of alcohol consumption.
2. Patients taking drugs affecting liver enzymes, (Contraceptive pills, Statins).
3. Pregnant women.
4. Subjects with Positive HBsAg, Anti HCV.

### Blood Collection

Blood sample were drawn using 5 ml syringes with steel needles, 5ml of venous blood was drawn from each patient and healthy controls, and immediately transferred to plain tube and were allowed to stand at room temperature for 20 min. for blood clotting. After centrifugation for 15 min. at 3000 rpm (revolution per minute) at room temperature for 5 min., the serum was immediately transferred to a second tube using a micro pipette and analyzed at the same day. The rest of the samples were stored at  $-26^{\circ}\text{C}$  in the Central lab.

## Methods

The study consisted of two parts: (1) a questionnaire and (2) Laboratory evaluation. The questionnaires included a socio-demographic characteristics like; Name, age, gender, residence. It also included smoking, duration of diabetes, hypertension, family history of diabetes, and drug history. Diabetes mellitus was defined as either clinical diagnosis of diabetes verified by participant's medical records or according to the criteria set by the report on expert committee on the diagnosis and classification of diabetes [ADA, 2010]. Weight had been measured while the patient minimally clothed without shoes using digital weight scale ,While height was measured in

standing position without shoes using tape measure while the shoulder in normal state. BMI was calculated by dividing the weight (Kg) by the height squared ( $m^2$ ) and categorized on the basis of the World Health Organization classification [WHO, 1998] are:

Normal weight (BMI 18.5 – 24.9 Kg/ $m^2$ ),

Overweight (BMI  $\geq$  25 – 29.9 kg/ $m^2$ ) and

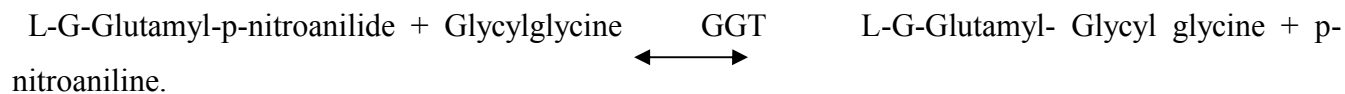
Obesity (BMI  $\geq$  30 kg/ $m^2$ ).

## Gamma glutamyl transferase (GGT) activity determination:

Blood tests including fasting GGT, were measured.

## Principle

This GGT activity protocol adopted was that of Szasz et al ( 1976), in which:



The rate of formation of P-nitroaniline, is directly proportional to the GGT activity in the sample, is measured at 405 nm.

## RESULTS

Demographic data and other categorical variables like smoking, body mass index and hypertension among the studied groups are described in Table-1.

**Table (1): Frequency distributions of categorical variables among studied sample.**

| Variables                     | Control (60) |         | Diabetics (115) |         |
|-------------------------------|--------------|---------|-----------------|---------|
|                               | No. of total | %       | No. of total    | %       |
| <b>Body mass index groups</b> |              |         |                 |         |
| Less than 25                  | 16           | (26.7)  | 23              | (20.0)  |
| 25-29.9                       | 26           | (43.3)  | 49              | (42.6)  |
| 30 and more                   | 18           | (30.0)  | 43              | (37.4)  |
| <b>Family History of DM</b>   |              |         |                 |         |
| Yes                           | 23           | (38.3 ) | 59              | (51.3 ) |
| NO                            | 37           | (61.7 ) | 56              | (48.7 ) |

The mean age  $\pm$  SD and Mean BMI of the diabetic and control groups are illustrated in Table-2

**Table (2): Age and BMI among Control and Diabetics.**

| Variables                           | Range      | Mean $\pm$ SD   |
|-------------------------------------|------------|-----------------|
| Age of Control (year)               | 31 – 68    | 47.6 $\pm$ 9.1  |
| Age of diabetic (year)              | 32- 78     | 52.5 $\pm$ 10.5 |
| BMI diabetic (kg/m <sup>2</sup> )   | 20.0- 48   | 28.8 $\pm$ 4.6  |
| BMI of Control (kg/m <sup>2</sup> ) | 21.5- 38.3 | 27.8 $\pm$ 3.7  |

The laboratory results show significantly higher mean GGT among diabetics as compared with control (14.2 $\pm$ 11.1 IU/L vs. 10.5 $\pm$ 8.0 IU/L)  $p=0.024$ , Fig-1.

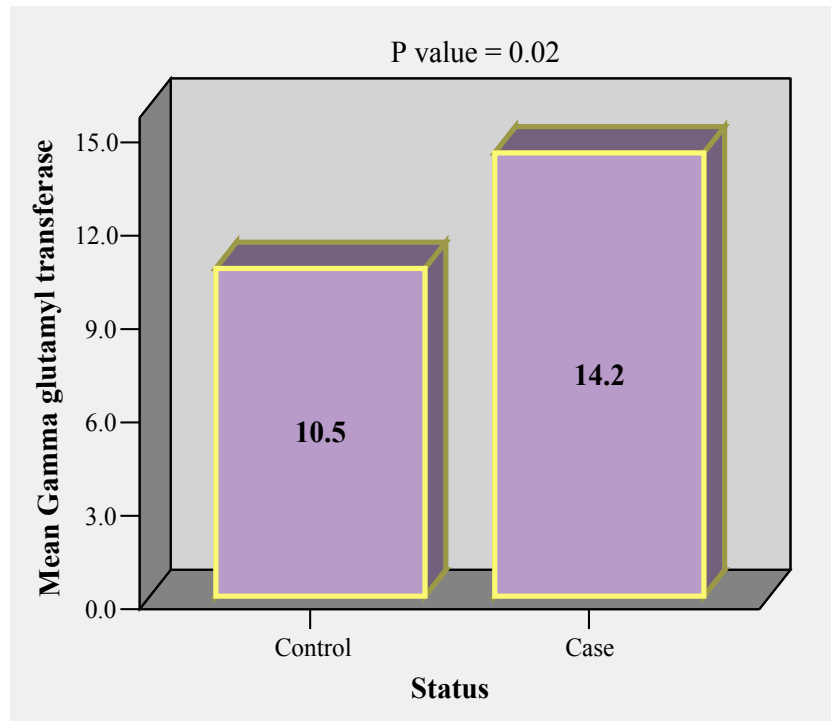


Figure (1): Mean GGT (IU/L) among Diabetic and Control group.

Family history of diabetes among first degree relatives, in both diabetic and control group, was significantly associated with higher mean GGT level. Table-3

Table (3): Relation of GGT with family history of Diabetes among Diabetics

| Family history of diabetes among DM | % of total 115 | GGT(IU/L) Mean ±SD | P value      |
|-------------------------------------|----------------|--------------------|--------------|
| Negative                            | 48.7           | 11.4 ± 6.8         | <b>0.009</b> |
| Positive                            | 51.3           | 16.8 ± 13.6        |              |
| Total                               | 100.0          | 14.2 ± 12.6        |              |

GGT activity was found to have statistically significant association with body mass index with ( $P < 0.05$ ) among diabetics (Fig-2), but not among control ( $p > 0.05$ )

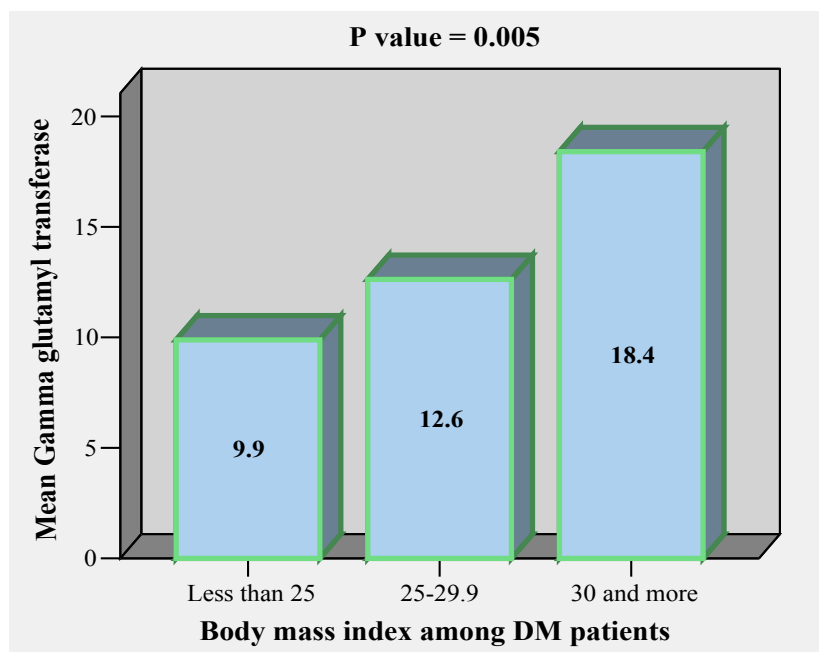


Fig (2) : BMI and GGT activity among diabetics.

## Discussion

Gamma glutamyl transferase was first used as a test in the evaluation of liver diseases. It reaches extremely high levels in patients with biliary obstruction and is a good marker for chronic alcohol consumption [Lee *et al*, 2006a]. Many scientists think that GGT plays an important role in protecting against oxidative stress by maintaining an adequate supply of intracellular glutathione, which protects cells against oxidants produced by normal metabolism [Lim *et al*, 2004; Meisinger *et al*, 2005; Bo *et al*, 2005; Zhang *et al*, 2005]. However, other has noticed that increased levels of serum GGT do not seem to reduce oxidative stress, implying that increased GGT is not a protective mechanism against oxidative stress [Lim *et al*, 2004]. Numerous studies

found that GGT is not just a marker of alcohol consumption, but is an independent predictor of many diseases, including cardiovascular diseases, type 2 diabetes, inflammation, and, possibly, underlying oxidative stress [Warram *et al*, 1990; Emdin *et al*, 2005; Sakuta *et al*, 2005; Wannamethee *et al*, 2005; Whitfield, 2001; Yamada *et al*, 2006]. The distribution of samples according to BMI, family history, and age were shown in table-1,2. In the current study, GGT level was significantly elevated ( $p < 0.05$ ) among diabetics than controls (fig-1), this result is in agreement With studies obtained previously [Balogun *et al*, 2008; Sharma *et al*, 2010]. The elevation in GGT activity may reflect an increased hepatic insulin resistance or oxidative stress. We can infer that much of GGT's association with disease is as a marker of preclinical effects that progress in time to

overt disease. Increase in GGT activity over time, even within the normal range, is associated with a change of insulin resistance markers and with a higher incidence of type 2 diabetes in both sexes [Philippe *et al*, 2006]. The GGT activity was found to have significant association ( $P < 0.001$ ) with first degree positive family history of diabetes in both diabetic and control group (table-3). The results were found to be in agreement with Inoue *et al.*, (2008) and Yue *et al.*, (2010), studies. A suggested explanation is that, elevated GGT was related to FHDM, independent of the other variables and also there is an additive interaction of family history and BMI on diabetes. In addition, GGT activity was found to associated significantly ( $P < 0.005$ ) with overweight, and obesity among the diabetics (fig-2). Similar results have been reported [Jousilahti *et al*, 2000; Al-Sultan AI, 2008; Azhar *et al*, 2009; Botton *et al*, 2007; Kasapoglu *et al*, 2010]. Association of Overweight, Obesity and abdominal fat distribution with increased GGT activity can be related to oxidative stress resulting from NAFLD, has been suggested in the mechanisms of insulin resistance,  $\beta$ -cell dysfunction, poorly-controlled type 2 diabetes, and subsequent complications [Kasapoglu *et al*, 2005; Bloomgarden, 2005; Wright *et al*, 2006]. Obesity particularly visceral or central (as evidence by the hip-waist ratio), is very common in type-2 DM. Adipocytes secrete a number of biologic products (leptin, TNF- $\alpha$ , free fatty acids,

resistin, and adiponectin) that modulate insulin secretion, insulin action and body weight and may contribute to the insulin resistance. Results from recent studies using data from the third NHANES study showed that all levels of GGT are strongly associated with C-reactive protein [Han *et al*, 2002; Sharma *et al*, 2010; Maryam *et al*, 2008]. This significant association might be a result of inflammation and oxidative stress in diabetes mellitus and that inflammatory markers, via their ability to enhance de novo hepatic fatty acid synthesis and fat accumulation, may contribute to both elevated liver enzymes and diabetes. These results strongly suggest that GGT is involved in the inflammatory pathway. Surprisingly, no any relations were found between GGT activity and cases of gender.

#### Reference

1. ADA (2010) American Diabetes Association.
2. Warram JH, Martin BC, Krolewski AS *et al.* (1990). Slow glucose removal rate and hyperinsulinemia precede the development of type-II diabetes in the offspring of diabetic parents. *Ann Intern Med*; 113: 909-15.
3. Ceriello A. (2005) Post-prandial hyperglycemia and diabetes complications: is it time to treat? *Diabetes*; 54 (1): 1-7.
4. Ford ES. (2003). the metabolic syndrome and C-reactive protein,



- fibrinogen and leucocytes count: findings from the Third National Health and Nutrition Examination Survey. *Atherosclerosis*; 168: 351-8.
5. Nakanishi N, Nishina K, Li W, Sato M, Suzuki K, Tatara K. (2003) Serum gamma-glutamyl transferase and development of impaired fasting glucose or type 2 diabetes in middle-aged Japanese men. *J Intern Med*; 254: 287-295
  6. Whitfield JB. (2001).Gamma-glutamyl transferase. *Crit Rev Clin Lab Sci*; 38:263-355
  7. Emdin M, Pompella A, Paolicchi A. (2005) Gamma-glutamyl transferase, atherosclerosis, and cardiovascular disease: triggering oxidative stress within the plaque. *Circulation*; 112:2078–2080.
  8. Lim JS, Yang JH, Chun BY, Kam S, Jacobs DR Jr, Lee DH.( 2004). Is serum gamma-glutamyl transferase inversely associated with serum antioxidants as a marker of oxidative stress? *Free Radic Biol Med*; 37: 1018-1023.
  9. Onat A. (2006).Serum gamma glutamyl transferase as a marker of metabolic syndrome and coronary disease likelihood in nondiabetic middle-aged and elderly adults. *Preventive Medicine*; 43, 136–139.
  10. Sharma R, Sharma S, Kaushik GG. (2010). Gamma-glutamyltransferase (GGT) –a Novel Marker of Endothelial Dysfunction? *JACM*; 11(1): 26-30.
  11. Perry IJ, Wannamethee SG, Shaper AG. (1998). Prospective study of serum gamma-glutamyl transferase and risk of NIDDM. *Diabetes Care*; 21:732-737.
  12. Wannamethee SG, Lennon L, Shaper AG, Whincup PH. (2005) Hepatic enzymes, the metabolic syndrome, and the risk of type 2 diabetes in older men. *Diabetes Care*; 28:2913–2918.
  13. Nilssen O and Forde OH. (1994). Seven-year longitudinal population study of change in gamma-glutamyl transferase: the Tromso Study. *Am J Epidemiol*; 139: 787-792
  14. Sakugawa H, Nakayoshi T, Kobashigawa K, Nakasone H, Kawakami Y, Yamashiro T, Maeshiro T, Tomimori K, Miyagi S, Kinjo F, Saito A. (2004). (Metabolic syndrome is directly associated with gamma glutamyl transpeptidase elevation in Japanese women. *World J Gastroenterol*; 10: 1052-1055.
  15. Liu CM, Tung TH, Liu JH, Chen VT, Lin CH, Hsu CT, Chou P. (2005).A community-based epidemiological study

- of elevated serum alanine aminotransferase levels in Kinmen, Taiwan. *World J Gastroenterol*; 11: 1616-1622.
16. Vozarova B, Stefan N, Lindsay RS, Saremi A, Pratley RE, Bogardus C, Tataranni PA. (2002). High alanine aminotransferase is associated with decreased hepatic insulin sensitivity and predicts the development of type 2 diabetes. *Diabetes*; 51:1889-1895.
  17. Sattar N, Scherbakova O, Ford I, O'Reilly DS, Stanley A, Forrest E, Macfarlane PW, Packard CJ, Cobbe SM, Shepherd J.(2004) Elevated alanine amino transferase predicts new-onset type 2 diabetes independently of classical risk factors, metabolic syndrome, and C-reactive protein in the west of Scotland coronary prevention study. *Diabetes*; 53: 2855-2860.
  18. Hanley AJ, Williams K, Festa A, Wagenknecht LE, D'Agostino RB Jr, Kempf J, Zinman B, Haffner SM.(2004). Elevations in markers of liver injury and risk of type 2 diabetes: the insulin resistance atherosclerosis study. *Diabetes*; 53: 2623-2632.
  19. Lee DH, Jacobs DR Jr, Gross M, Kiefe CI, Roseman J, Lewis CE, Steffes M. (2003-a). Gamma-glutamyl transferase is a predictor of incident diabetes and hypertension: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Clin Chem*; 49: 1358-1366.
  20. Nakanishi N, Nishina K, Li W, Sato M, Suzuki K, Tataru K. (2003) Serum gamma-glutamyl transferase and development of impaired fasting glucose or type 2 diabetes in middle-aged Japanese men. *J Intern Med*; 254: 287-295.
  21. Harrison TA, Hindorff LA, Kim H, Wines RCM, Bowen DJ, Mcgrath BB. (2003). Family history of diabetes as a potential public health tool. *Am J Prev Med.*; 24:152–159.
  22. Lee, D., Blomhoff, R., & Jacobs, D. Jr. (2004-a). Is serum gamma glutamyl transferase a marker of oxidative stress: *Free Radical Research*, 38(6), 535-539.
  23. Andre P, Balkau B, Born C, Royer B, Wilpart E, Charles MA, Eschwege E. (2005). Hepatic markers and development of type 2 diabetes in middle aged men and women: a three-year follow-up study. The D.E.S.I.R. Study (Data from an Epidemiological Study on the Insulin Resistance syndrome). *Diabetes Metab* 31:542-550.
  24. World Health Organization. (1998). Obesity—Preventing and Managing the Global Epidemic: Report of a WHO Expert Committee. *World Health Organization*: Geneva.

25. Szasz G, Gruber W , Bernt, E (1976). Reaction-rate method for gamma-glutamyl transferase activity in serum *Clinical chemistry*, 22: 650-656.
26. Lee DH, Silventoinen K, Hu G, Jacobs DR, Jr, Jousilahti P, Sundvall J, et al.(2006-a) Serum gamma-glutamyl transferase predicts non-fatal myocardial infarction and fatal coronary heart disease among 28,838 middle-aged men and women. *Eur Heart J*; 27:2170- 2176.
27. Meisinger C, Lowel H, Heier M, Schneider A, Thorand B. (2005). KORA Study Group: Serum gamma-glutamyl transferase and risk of type 2 diabetes mellitus in men and women from the general population. *J Intern Med* 258:527-535.
28. Zhang, H., Forman, H. J., & Choi, J. (2005), Gamma-glutamyl transpeptidase in glutathione biosynthesis. *Methods in Enzymology*, 401, 468-83.
29. Bo S., Gambino, R., Durazzo, M., Guidi, S., Tiozzo. E., Ghione, F., Gentile, L., Cassader, M., & Pagano, G. F. (2005). Associations between gamma-glutamyl transferase, metabolic abnormalities and inflammation in healthy subjects from a population based cohort: A possible implication for oxidative stress. *World Journal of Gastroenterology*, 11(45), 7109-7117.
30. Sakuta, H., Suzuki, T., Yasuda, H., & Ito, T. (2005). Gamma-glutamyl transferase and metabolic risk factors for cardiovascular disease, *Internal Medicine*; 44(6): 538-541.
31. Yamada, J., Tomiyama, H., Yambe, M., Koji, Y., Motobe, K., Shiina, K., Yamamoto, Y., & Yamashina, A. (2006). Elevated serum levels of alanine aminotransferase and gamma glutamyl transferase are markers of inflammation and oxidative stress independent of the metabolic syndrome *Atherosclerosis*, 189, 198-205.
32. Balogun WO, Adeleye JO, Akinlade KS, Adedapo KS, Kuti M. (2008). Frequent occurrence of high gamma-glutamyltransferase and alanine amino transferase among Nigerian patients with type 2 diabetes. *Afr J Med Med Sci*; 37: 177-83.
33. Sharma R, Sharma S, Kaushik GG. (2010). Gamma-glutamyltransferase (GGT) –a Novel Marker of Endothelial Dysfunction? *JLACM*; 11(1): 26-30
34. Philippe A, Beverley B, Catherine B, Marie-A Charles, and Eveline(

- 2006). Three-year increase of gamma-glutamyl transferase level and development of type 2 diabetes in middle-aged men and women: the D.E.S.I.R. cohort. *Diabetologia*; 49(11): 2599–2603.
35. Inoue K, Matsumoto M, Miyoshi Y, Kobayashi Y. (2008). Elevated liver enzymes in women with a family history of diabetes. *Diabetes Res Clin Pract*; 79(3): 4-7.
36. Yue Chen, Donna C Rennie and James A Dosman. (2010). Synergy of BMI and family history on diabetes: the Humboldt Study, *Public Health Nutrition* 13: 461-465.
37. Jousilahti P, Rastenyte D, Tuomilehto J. (2000). Serum gamma-glutamyl transferase, self-reported alcohol drinking, and the risk of stroke. *Stroke*; 31:1851-1855.
38. Al-Sultan AI. (2008). Assessment of the Relationship of Hepatic Enzymes with Obesity and Insulin Resistance in Adults in Saudi Arabia. *Sultan Qaboos University Medical Journal*, July, Volume 8, Issue 2, P. 185-192
39. Azhar I., Akbar K, Uzma I and Mehjabeen. (2009). A Comparison of the effect of Gamma Glutamyl transferase on age and obesity among normal, hypertensive and type 2 diabetics, *Biomedica* Vol.25, Jul. – Dec/Bio-7.Doc P. 123 – 127
40. Botton J, Heude B, Andre P, Bresson JL, Ducimetiere P, Charles MA (2007) Relationship between gamma-glutamyl transferase and fat mass in a general population of 8-17 years old children. The FLVS II study. *Diabetes Metab*. Nov; 33(5):354-9.
41. Kasapoglu B., Turkay C. Bayram Y. & Koca C. (2010) Role of GGT in diagnosis of metabolic syndrome: A clinic-based cross-sectional survey *Indian J Med Res* 132, July, pp56-61.
42. Kasapoglu C, Tschritter O, Haap M, Shirkavand F, Machann J, Fritsche A, Schick F, Haring H, Stumvoll M. (2005). Elevated serum GGT concentrations predict reduced insulin sensitivity and increased intrahepatic lipids. *Horm Metab Res* 37:246-251.
43. Bloomgarden, Z. T. (2005). Insulin resistance syndrome and nonalcoholic fatty liver disease. Second world congress on the insulin resistance syndrome. *Diabetes Care*, 28(6):1518-1523.
44. Wright E., Scism-Bacon, J. L., & Glass, L. C. (2006). Oxidative stress in type 2 diabetes: The role of fasting and postprandial glycaemia. *International*

- Journal of Clinical Practice*, 60(3), 308-314.
45. Han TS, Sattar N, Williams K, Gonzalez-Villalpando C, Lean ME, Haffner SM: (2002).Prospective study of C-reactive protein in relation to the development of diabetes and metabolic syndrome in the Mexico City Diabetes Study. *Diabetes Care* 25:2016-2021.
46. Maryam T, Hadi H, Farzad H, Yadollah M and Fereidoun A. (2008).Association of liver enzymes with incident type 2 diabetes: A nested case control study in an Iranian population. *BMC Endocrine Disorders*, 8:5.

## تقييم نشاط الكاما كلوتاميل ترانسفيريز في مرضى الداء السكري من النوع الثاني في محافظة السليمانية

حامد غفوري حسن. 2. فينك باقر معروف 1.

1. جامعة بغداد كلية ابن الهيثم 2. وزارة الصحة المستشفى الجمهوري

### الخلاصة

ان زيادة مستويات الانزيم الكبدي كاما كلوتاميل ترانسفيريز (GGT) تكون مرتبطة بداء السكري. تم قياس نشاط كاما كلوتاميل ترانسفيريز و محتوى كتلة الجسم وكذلك تاريخ الاسرة المرضي للمتطوعين وذلك في 115 نموذجا لمرضى السكري وبواقع 58 ذكور و 57 اناث مقابل 60 نموذجا من الاصحاء وبواقع 34 ذكور و 26 اناث. تم جمع المعلومات الشخصية للمرضى وطبق SPSS 16 في تحليل النتائج. أظهرت النتائج ارتفاعا معنويا ( $P < 0.05$ ) (GGT) المرضى ( $14.2 \pm 11.1$  IU/L) مقارنة بنشاطه عند الاصحاء ( $10.5 \pm 8.0$  IU/L) كما اظهرت النتائج ارتفاعا معنويا ( $P < 0.05$ ) في نشاط الانزيم في النماذج المرضية التي لها تاريخا اسريا في المرض مقارنة بالاصحاء. أتضح كذلك ان لانزيم كاما كلوتاميل ترانسفيريز علاقة ارتباطية مع محتوى كتلة الجسم. اجريت الدراسة في مركز السليمانية لامراض السكري غدد الصماء للفترة من حزيران والى كانون الاول 2010.