International Journal of Current Advanced Research
ISSN: O: 2319-6475, ISSN: P: 2319-6505, Impact Factor: 6.614
Available Online at www.journalijcar.org
Volume 7; Issue 5(B); May 2018; Page No. 12361-12366
DOI: http://dx.doi.org/10.24327/ijcar.2018.12366.2170

Research Article

SERUM GAMMA GLUTAMYL TRANSFERASE AND SIALIC ACID LEVELS IN TYPE 2 DIABETIC PATIENTS AND ITS ASSOCIATION WITH GLYCEMIC CONTROL

Kishan Prasad HL*[1]., Sabari Das.[1], Suchetha Kumari[2] and Jayaprakash Shetty K*[1]

[1]Department of Pathology, K S Hegde Medical Academy, Mangalore
[2]Department of Biochemistry, K S Hegde Medical Academy, Mangalore

A R T I C L E I N F O

Article History:

Received 6th February, 2018
Received in revised form 20th March, 2018 Accepted 8th April, 2018
Published online 28th May, 2018

Key words:

Type 2 Diabetes, Serum sialic acid, Gamma glutamyl tranferase,
Hb A1C, Glycaemic control

A B S T R A C T

Purpose: To analyse the association of oxidative stress (GGT activity) and inflammatory response (Sialic acid levels) with glycemic control (HbA1C) in patients with type 2 diabetes mellitus.
Methods: This non-Interventional, cross-sectional study comprised of 60 study subjects with 30 type 2 diabetes patients (the case group) and 30 healthy individuals (control group). From the venous blood, serum GGT was measured spectrophotometrically using commercially available kits. Total sialic acid was measured by using diphenylamine method and glycated haemoglobin (HbA1C) was measured using ion-exchange resin method on a spectrophotometer.
Results: The results showed a significant increase in the MDA levels in the elderly population (1.851 ± 0.583 μM/L) in comparison with the young adult population (0.963 ± 0.302 μM/L). There was also a significant reduction in the TAC the elderly, individuals (1.189 ± 0.411 μM/L) as compared to younger adults (1.342 ±0. 575 μM/L; $p<0.001$). But we did not find any significant correlation between the TAC and MDA levels in the study population.
Conclusion: In conclusion, the present study suggests that serum GGT and Sialic acid concentration is significantly increased in type 2 diabetes mellitus as compared to healthy control. There is a significant positive correlation between GGT and HbA1C. All these findings suggest a link between oxidative stress, inflammation and glycemic control in patients with type 2 diabetes mellitus.

INTRODUCTION

Diabetes mellitus (DM) refers to a group of metabolic disorders that share the common phenotype of hyperglycaemia [1]. At 2014, the global prevalence of diabetes was estimated to be 9% among adults aged 18 years and above [2]. According to WHO, diabetes will be the 7th leading cause of death in 2030 [3]. Recent research studies suggest the association of oxidative stress and inflammation to β-cell dysfunction in the pathogenesis of DM [4].

Type 2 diabetes (formerly called non-insulin dependent or adult onset) results from the body's ineffective use of insulin. Type 2 diabetes mellitus comprises 90% of people with diabetes around the world [5] and is largely the result of excess body weight and physical inactivity. Symptoms are similar to those with type 1 diabetes like excessive excretion of urine (polyuria), thirst (polydipsia), constant hunger, weight loss, vision changes and fatigue, but are often less marked. As a result, the disease may be diagnosed several years after onset, once complications have already risen.

Corresponding author:* **Kishan Prasad HL
Department of Pathology, K S Hegde Medical Academy, Mangalore

Until recently, this type of diabetes was seen only in adults but now it is also occurring in children. Gamma glutamyl transferase (GGT) is a plasma membrane bound enzyme which plays a pivotal role in maintaining the intracellular antioxidant defences through its mediation of extracellular glutathione (GSH) transport into most of the cells [6]. GGT can reflect the metabolic alterations and can serve as a marker of insulin resistance syndrome. Studies also suggest that GGT plays an important role in antioxidant systems [6, 7].

Sialic acid is an acetylated derivative of neuraminic acid and is attached to the non-reducing residues of carbohydrate chains of glycoproteins and glycolipids [8]. Sialic acid can be used as a measurement of acute phase response [9, 10]. Several population-based studies round the globe have clearly demonstrated the potential role of elevated serum Sialic acid as an inflammatory biomarker for cardiovascular disease in type 2 DM [11].

As the development of complications in DM is linked to the accumulation of glycation adducts in tissue proteins, it is the need of the hour to constantly monitor the glycemic control. Evaluation of plasma glucose measurements (fasting and post prandial blood sugar) provides a picture of short term glycemic control, whereas the glycated haemoglobin (HbA_1C) levels

reflect the average glycaemic control over the previous 3 months [1].

The rationale behind our study is to analyze the association of oxidative stress (GGT activity) and inflammatory response (Sialic acid levels) with glycemic control (HbA_1C) in patients with Type 2 DM.

Aims and Objectives
The purpose of this study is

- To estimate the Fasting blood sugar (FBS) level and glycated hemoglobin (HbA_1C) in Type 2 diabetes mellitus patients (the case group) and in the control group.
- To evaluate the serum GGT and Sialic acid levels in the case and control groups.
- To compare the serum GGT activity and Sialic acid levels with Glycated hemoglobin (HbA_1C) in the study population.

MATERIALS AND METHODS

Study Design

Type of Study : Prospective case-control study
Study duration : 2 months
Sample size : Minimum of 60subjects (30 case + 30 controls)

Ethical considerations

Institutional Ethical Clearance was obtained. A total of 60 study participants of both the sexes fulfilling the inclusion criteria was recruited.

Study population
The study population comprised of

- **Case group** : 30 cases of type 2 DM patients of either sex
- **Control group** : 30 non-diabetic individuals of either sex.

Inclusion criteria

30 cases of type 2 DM confirmed by biochemical investigations as per WHO criteria. The following table summarizes the 2006 WHO recommendations for the diagnostic criteria of diabetes (Table 1)

Table 1 2006 WHO recommendations for the diagnostic criteria of diabetes

	Fasting Blood Glucose (FBS) levels (mg/dl)	2 hour glucose (mg/dl)
Normal	**<110**	**<140**
DM	**≥126**	**≥200**

Exclusion criteria

- Patients with type 1 DM, acute complications such as severe infections.
- History of previous surgery, severe cardiovascular or respiratory distress.
- Smokers, alcoholics and patients under any nutritional supplements etc.

Sample Collection

5ml of blood sample was collected after an overnight fasting from the recruited subjects. Serum separated and wasstoredat -20^{o}C until analysis.

Procedure

1. Fasting plasma glucose was measured by glucose-oxidase method.
2. Glycated hemoglobin (HbA_1C) was measured using ion-exchange resin method on Spectrophotometer.
3. GGT was measured spectrophotometrically using commercially available kits.
4. Total serum sialic acid was measured by using diphenylamine method [27].

Statistical Analysis

Descriptive statistics like mean and standard deviation of the collected data was computed along with confidence intervals. Comparison of the different parameters under study in the different groups was tested by using the chi-square test. 'p' value of <0.05 was considered significant. SPSS software was used for the analysis of the data.

OBSERVATIONS AND RESULTS

The study involves the estimation of gamma-glutamyl transferase (GGT) and Sialic acid levels in diabetic as well as healthy individuals and its correlation with glycated haemoglobin (HbA_1C). In our study, we found out that there is a significant increase in GGT levels in diabetic individuals (26.8 ± 2.19) as compared to the control population (21.6 ± 0.873).

Table 2 GGT concentration (U/L) in diabetic population and control

Study group	GGT in U/L	
	Mean	Standard Deviation
Type 2 diabetic patients	26.8	2.19
Control	21.6	0.873

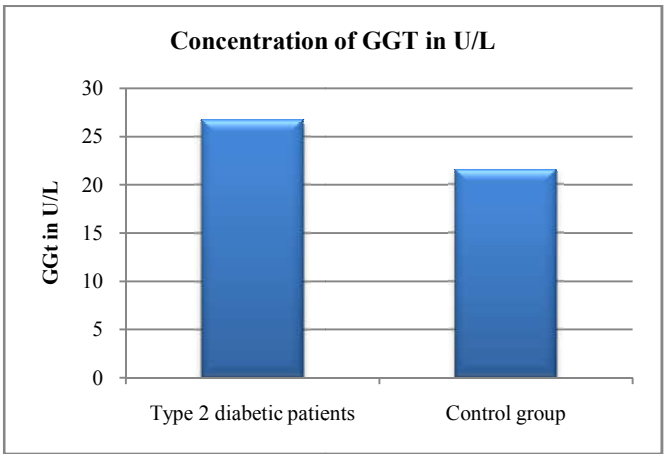

Figure 1 Concentration of GGT in type 2 diabetic patients and control group.

The study also showed an increase in Sialic acid level in type 2 diabetic patients (82.90 ± 1.542) in comparison with normal healthy individuals (58.43 ± 1.135).

Table 2 Sialic acid (mg/dL) levels in diabetic patients and control.

Study group	Sialic acid in mg/dL	
	Mean	Standard Deviation
Type 2 diabetic patients	82.90	1.542
Control	58.43	1.135

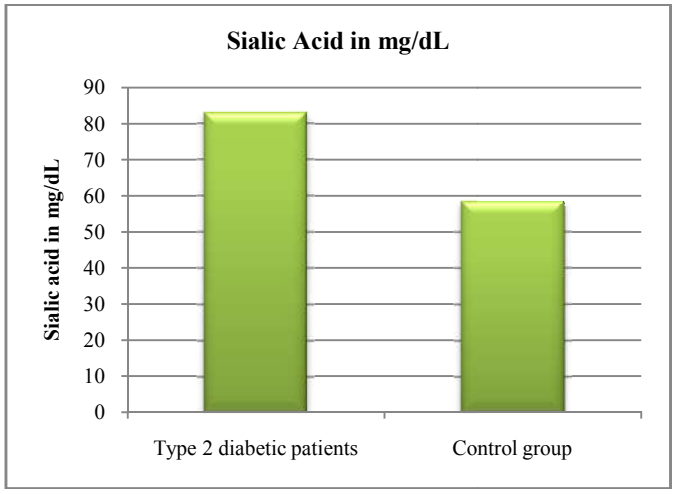

Figure 2 Sialic acid (mg/dL) levels in type 2 diabetic patients and control

Table 2 Showing mean and standard deviation of various parameters studied.

Parameter	Control (mean±SEM)	Diabetes(mean±SEM)	p- Value	Significance
FBS in mg/L	98.2 ± 2.18	146 ± 5.74	P<0.0001	Yes
HbA1C %	3.91 ± 0.125	6.94 ± 0.31	P<0.0001	Yes
GGT in U/L	21.6 ± 0.873	26.8 ± 2.19	P<0.05	Yes
Sialic Acid in mg/dL	58.43 ± 1.135	82.90 ± 1.542	P<0.0001	Yes

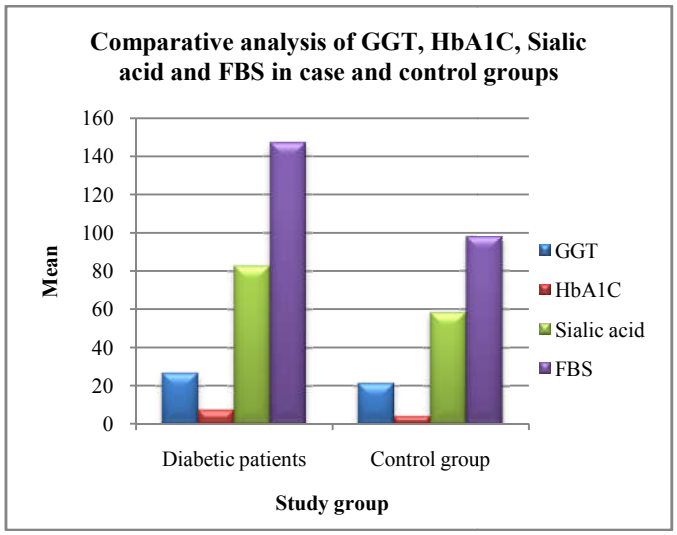

Figure 1 Comparative analysis of GGT, HbA_1C, Sialic acid and FBS in case and control groups.

We could also find a significant positive correlation between GGT and HbA_1C in patients with type 2 diabetes mellitus.

Table 3 Pearson's correlation analysis between serum GGT with HbA_1C

	Correlation coefficient r value	Two tailed p value
Serum GGT with HbA_1C	0.452	<0.05

DISCUSSION

The exact cause of type 2 diabetes mellitus which affects at least 100 million people throughout the world is not known. Though insulin resistance seems to be the central abnormality, the origin of the impaired insulin action and how it explains the complications of diabetes mellitus is not known.

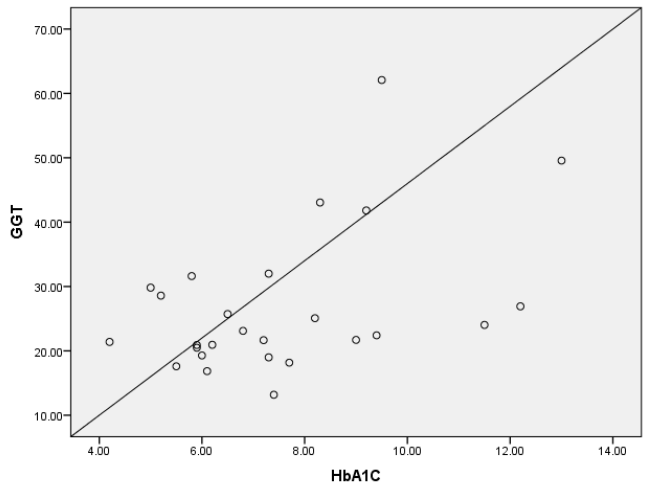

Figure 2 Showing correlation between serum GGT with HbA_1C in type 2 diabetic patients.

Our study showed statistically significantly increased concentration of GGT and Sialic acid in serum in patients with type 2 diabetes mellitus as compared to normal healthy individuals. Also, we found a significant positive correlation between GGT and HbA_1C in type diabetic patients. These findings suggest a link between oxidative stress as indicated by serum GGT concentration) and inflammation (Sialic acid) with glycemic control (HbA_1C). This could suggest the probable role of oxidative stress and chronic low grade inflammation in the pathogenesis of type 2 diabetes mellitus.

Elevation of serum GGT could be the expression of an excess fat deposition in the liver, termed non-alcoholic fatty liver disease. Fatty liver is thought to cause hepatic insulin resistance and to contribute to the development of systemic insulin resistance and hyperinsulinemia. Thus, GGT could serve as a marker of insulin resistance in the pathogenesis of diabetes. [28,29].

There is also strong supportive evidence to suggest that GGT is not only a marker of insulin resistance but also a marker of oxidative stress. Various studies have reported that GGT has a central role in the maintenance of intracellular antioxidant defences through its mediation of extracellular glutathione transport into most types of cells [6]. It is an ectoenzyme normally present at the outer side of the cell membrane that has the primary function of maintaining intracellular concentrations of glutathione (GSH), a critical antioxidant defence for the cell. Increase in GGT activity can be a response to oxidative stress, facilitating increased transport of GSH precursors into cells. In addition, GGT is leaked into the serum possibly because of normal cell turnover and cellular stresses. Several mechanisms for GGT leakage are possible and include increase in oxidative stress, proteolysis, glycosylation, GGT synthesis and endothelial cell damage [30]. Thus, increased serum concentrations of GGT could serve as a marker of increased oxidative and other cellular stresses. Oxidative stress is currently suggested as a patho-mechanism underlying diabetes and its complications. Of late, much attention has been focused on the role of oxidative stress and it has been reported that oxidative stress may constitute the key and common event in the pathogenesis of secondary diabetic complications. Implications of oxidative stress in the pathogenesis of diabetes is suggested, not only by oxygen free-radical generation, but also due to non-enzymatic protein glycosylation, auto-oxidation of glucose, impaired glutathione

metabolism, alteration in antioxidant enzymes, lipid peroxides formation and decreased ascorbic acid levels. In addition to GSH, there are other defence mechanisms against free radicals like enzymes superoxide dismutase (SOD), glutathione peroxidase (GPx) and catalase (CAT) whose activities contribute to eliminate superoxide, hydrogen peroxide and hydroxyl radicals [31]. Raised GGT concentrations could be a marker of oxidative stress, which might also play, a role in the cause and development of diabetes and its complications [6,7]. We also observed a statistically significant increase in total sialic acid concentration in the type DM patients studied as compared to controls. As the increased serum sialic acid concentration is due to an increase in acute phase reaction proteins, we confirm the increased acute phase reactant protein response in diabetics. This is in consistent with earlier reports [32, 33,34] showing increased acute phase response in type 2 diabetics even in the absence of complications which might themselves evoke that response.

Type 2 diabetes is an acute phase disease in which increased concentrations of cytokines are secreted from many cells such as macrophages, adipose tissue and endothelium, under the influence of stimuli such as over nutrition, perhaps in those predisposing to having an unregulated response because of increasing age, genetic,foetal metabolic pre-programming. Cytokines mainly IL-1, IL-6, and TNF α act on the liver to produce the characteristic dyslipidaemia of type 2 diabetes. They also promote the release from the liver of acute phase proteins which are atherosclerotic risk factors, stimulate leptin release from adipose tissue and act on the brain to release ACTH and then cortisol. The latter may contribute to obesity, hypertension and insulin resistance. TNF α also is a major factor in causing insulin resistance, long term hyper secretion of cytokines may impair beta cell insulin secretion [35].

Our study also suggests a positive correlation between GGT concentration and HbA_1C levels in patients with type 2 diabetes mellitus. There are several lines of evidence to support a relationship between elevated serum GGT and poor glycaemic control. Higher GGT levels are accompanied by more insulin resistance and greater risk for developing type diabetes mellitus and poor glycaemic control [36-40]. The strong association of serum GGT activity with some diabetes related metabolic disorders, such as atherogenic dyslipidaemia and poor glycaemic control, may be explained by underlying, not mutually exclusive, biological mechanisms such as fatty liver, insulin resistance and enhanced oxidative stress [36, 41-45]. It is possible that that the occurrence of GGT-mediated redox reactions plays a direct role in the pathogenesis of atherogenic dyslipidaemia and poor glycaemic control, independently of the presence of fatty liver, possibly through the induction of chronic inflammation and insulin resistance [46].

It is true that relatively small sample size is considered as a potential limitation of the present study and further intensive evaluation is need encompassing larger sample size considering other parameters of oxidative stress, dietary habit, genetic predisposition, fasting insulin concentration and lifestyle of the study population

CONCLUSIONS

In conclusion, the present study suggests that serum GGT and Sialic acid concentration is significantly increased in type 2 diabetes mellitus as compared to healthy control. There is a significant positive correlation between GGT and HbA_1C. All these findings suggest a link between oxidative stress, inflammation and glycaemic control in patients with type 2 diabetes mellitus. Thus, we propose that insulin resistance due to increase in GGT and persistent low grade inflammation as suggested by elevated Sialic acid levels has a significant role in the genesis of type 2 diabetes mellitus. Further, GGT and its correlation with glycated haemoglobin (HbA_1C) could highlight the probable clinical use of GGT evaluation in future as a marker of diabetic complications.

References

1. Alvin C. Diabetes Mellitus. In: Harrison's Principle of Internal Medicine. 19th ed. New York: McGraHill; 2015. p. 2399-406
2. WHO | Global status report on non-communicable diseases 2014 [Internet]. WHO. [cited 2016 Jan 22]. Available from: http://www.who.int/nmh/publications/ncd-status-report-2014/en/
3. Mathers CD, Loncar D. Projections of Global Mortality and Burden of Disease from 2002 to 2030. PLoS Med. 2006 Nov 28;3(11):e442.
4. Ford ES. The metabolic syndrome and C-reactive protein, fibrinogen, and leukocyte count: findings from the Third National Health and Nutrition Examination Survey. *Atherosclerosis*. 2003 Jun;168(2):351-8.
5. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: Diagnosis and classification of diabetes mellitus. Geneva, World Health Organization, 1999 (WHO/NCD/NCS/99.2)
6. Karp DR, Shimooku K, Lipsky PE. Expression of γ-glutamyl transpeptidase protects ramos B cells from oxidation-induced cell death. *Journal of Biological Chemistry*. 2001;276(6):3798-804.
7. Lee DH, Ha MH, Kim JH, Christiani DC, Gross MD, Steffes M, *et al*. Gamma-glutamyl transferase and diabetes—a 4-year follow-up study. *Diabetologia*. 2003;46(3):359–64.
8. Sudha R, Toora BD, Sarkar G, Sarkar M. Serum sialic acid in relation to erythrocyte sedimentation rate and hba of type 2 diabetic patients. *National Journal of Basic Medical Sciences*. 2(4):316-9.
9. Pickup JC. Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes. *Diabetes Care*. 2004 Mar;27(3):813-23.
10. Kugelman A, Choy HA, Liu R, Shi MM, Gozal E, Forman HJ. gamma-Glutamyl transpeptidase is increased by oxidative stress in rat alveolar L2 epithelial cells. *Am J Respir Cell Mol Biol*. 1994 Nov;11(5):586–92.
11. Crook M, Lumb P, Andrews V, Swaminathan R. Serum total sialic acid, a reputed cardiovascular risk factor, and its relationship to lipids, plasma fasting insulin, blood pressure and body mass index in normal individuals. *Clin Sci.* 1998 Jul;95(1):53-7.
12. Shahid SM, Mahboob T. Clinical correlation between frequent risk factors of diabetic nephropathy and serum sialic acid. *International Journal of Diabetes and Metabolism*. 2006;14(3):138-42.
13. Bennett CM, Guo M, Dharmage SC. HbA(1c) as a screening tool for detection of Type 2 diabetes: a systematic review. *Diabet Med*. 2007 Apr;24(4):333–43.

14. Kaushik GG, Sharm S, Sharma R, Mittal P. Association between gamma glutamyl transferase and insulin resistance markers in healthy obese children. *J Assoc Physicians India.* 2009 Oct;57:695-8.
15. Cho HC. The Association between Serum GGT Concentration and Diabetic Peripheral Polyneuropathy in Type 2 Diabetic Patients. *Korean Diabetes J*. 2010 Apr;34(2):111-8.
16. Kasapoglu B, Turkay C, Bayram Y, Koca C. Role of GGT in diagnosis of metabolic syndrome: a clinic-based cross-sectional survey. *Indian J Med Res*. 2010 Jul;132:56-61.
17. Zoungas S, Chalmers J, Ninomiya T, Li Q, Cooper ME, Colagiuri S, *et al*. Association of HbA1c levels with vascular complications and death in patients with type 2 diabetes: evidence of glycaemic thresholds. *Diabetologia*. 2012 Mar;55(3):636-43.
18. K. P, Kumar J. A, Rai S, Shetty SK, Rai T, Shrinidhi, *et al*. Predictive Value of Serum Sialic Acid in Type-2 Diabetes Mellitus and Its Complication (Nephropathy). *J ClinDiagn Res*. 2013 Nov;7(11):2435-7.
19. Gohel MG, Chacko AN. Serum GGT activity and hsCRP level in patients with type 2 diabetes mellitus with good and poor glycemic control: An evidence linking oxidative stress, inflammation and glycemic control. *J Diabetes MetabDisord.* 2013;12(1):56.
20. Khan SA, Rao R. Sialic acid a marker of diabetic complication. *IJPBS*. 2014;4(3):70-7.
21. Kunutsor SK, Abbasi A, Adler AI. Gamma-glutamyltransferase and risk of type II diabetes: an updated systematic review and dose-response meta-analysis. *Ann Epidemiol*. 2014 Nov; 24(11):809-16.
22. Jarcuska P, Janicko M, Drazilová S, Senajová G, Veselíny E, Fedacko J, *et al*. Gamma-glutamyltranspeptidase level associated with metabolic syndrome and proinflammatory parameters in the young Roma population in eastern Slovakia: a population-based study. *Cent Eur J Public Health*. 2014 Mar;22 Suppl:S43-50.
23. Nanditha A, Jagannathan R, Sundaram S, Susairaj P, Shetty AS, Snehalatha C, *et al*. Combining Fasting Plasma Glucose with Gamma-glutamylTransferase Improves the Sensitivity to Predict Incident Diabetes in Asian Indian Men with Impaired Glucose Tolerance. *J Assoc Physicians India.* 2014 Nov; 62(11):18-22.
24. Bhattacharjee D, Bhattacharya GC, Chakroborti G, Ravi BV. Study of Serum Sialic Acid and Copper as Inflammatory Markers in Type 2 Diabetes Mellitus. *Indian Medical Gazette*. 2015; 13-8.
25. Varghese A, Asha NS, Celine TM, Prasanna D. Inflammatory markers in Type II Diabetes mellitus. The *Pharma Innovation Journal*. 2015; 4(7):64-6.
26. Bahijri SM, Ahmed M, Al-Shali K, Bokhari S, Alhozali A, Borai A, *et al*. The relationship of management modality in Saudi patients with type 2 diabetes to components of metabolic syndrome, γ glutamyltransferase and highly sensitive C-reactive protein. *TherAdv Chronic Dis*. 2016 Sep; 7(5):246-54.
27. Winzler RJ. Determination of serum glycoproteins. *Methods Biochem Anal*. 1955; 2:279-311.
28. Marchesini G, Brizi M, Bianchi G, Tomassetti S, Bugianesi E, Lenzi M, *et al*. Nonalcoholic fatty liver disease: a feature of the metabolic syndrome. *Diabetes*. 2001 Aug;50(8):1844-50.
29. Chitturi S, Abeygunasekera S, Farrell GC, Holmes-Walker J, Hui JM, Fung C, *et al*. NASH and insulin resistance: Insulin hypersecretion and specific association with the insulin resistance syndrome. *Hepatology*. 2002 Feb;35(2):373-9.
30. Ikeda Y, Fujii J, Taniguchi N, Meister A. Expression of an active glycosylated human gamma-glutamyltranspeptidase mutant that lacks a membrane anchor domain. ProcNatlAcadSci USA. 1995 Jan 3;92(1):126-3
31. Moussa SA. Oxidative stress in diabetes mellitus. *Romanian J Biophys*. 2008;18(3):225-236.
32. Abdella N, Akanji AO, Mojiminiyi OA, Al Assoussi A, Moussa M. Relation of serum total sialic acid concentrations with diabetic complications and cardiovascular risk factors in Kuwaiti Type 2 diabetic patients. *Diabetes Res ClinPract*. 2000 Sep;50(1):65-72.
33. Akbri MZ, Sheikh AS, Bhatti MS, Hussnain M, Chaudhry ZA. Serum sialic acid level in diabetic retinopathy. *J Ayub Med Coll Abbottabad*. 2001 Mar;13(1):29-30.
34. Crook MA, Tutt P, Pickup JC. Elevated serum sialic acid concentration in NIDDM and its relationship to blood pressure and retinopathy. *Diabetes Care*. 1993 Jan;16(1):57-60.
35. Pickup JC, Crook MA. Is type II diabetes mellitus a disease of the innate immune system? *Diabetologia*. 1998 Oct;41(10):1241-8.
36. Thamer C, Tschritter O, Haap M, Shirkavand F, Machann J, Fritsche A, *et al*. Elevated serum GGT concentrations predict reduced insulin sensitivity and increased intrahepatic lipids. *HormMetab Res*. 2005 Apr;37(4):246-51.
37. André P, Balkau B, Born C, Charles MA, Eschwège E, D.E.S.I.R. study group. Three-year increase of gamma-glutamyltransferase level and development of type 2 diabetes in middle-aged men and women: the D.E.S.I.R. cohort. *Diabetologia*. 2006 Nov; 49(11):2599-603.
38. Marchesini G, Avagnina S, Barantani EG, Ciccarone AM, Corica F, Dall'Aglio E, *et al*. Aminotransferase and gamma-glutamyltranspeptidase levels in obesity are associated with insulin resistance and the metabolic syndrome. *J Endocrinol Invest*. 2005 Apr; 28(4):333-9.
39. Lee DH, Silventoinen K, Jacobs DR, Jousilahti P, Tuomileto J. gamma-Glutamyltransferase, obesity, and the risk of type 2 diabetes: observational cohort study among 20,158 middle-aged men and women. *J ClinEndocrinolMetab.* 2004 Nov; 89(11):5410-4.
40. Malnick SDH, Beergabel M, Knobler H. Non-alcoholic fatty liver: a common manifestation of a metabolic disorder. *QJM*. 2003 Oct; 96(10):699-709.
41. Targher G. Non-alcoholic fatty liver disease, the metabolic syndrome and the risk of cardiovascular disease: the plot thickens. *Diabet Med*. 2007 Jan;24(1):1-6.
42. Lee D-H, Blomhoff R, Jacobs DR. Is serum gamma glutamyltransferase a marker of oxidative stress? *Free Radic Res*. 2004 Jun;38(6):535-9.
43. Paolicchi A, Emdin M, Ghliozeni E, Ciancia E, Passino C, Popoff G, *et al*. Images in cardiovascular medicine. Human atherosclerotic plaques contain gamma-

glutamyltranspeptidase enzyme activity. *Circulation*. 2004 Mar 23; 109(11):1440.
44. Emdin M. Gamma-glutamyltransferase as a cardiovascular risk factor. *European Heart Journal*. 2006 Aug 9; 27(18):2145-6.
45. Hanley AJG, Williams K, Festa A, Wagenknecht LE, D'Agostino RB, Haffner SM. Liver markers and development of the metabolic syndrome: the insulin resistance atherosclerosis study. Diabetes. 2005 Nov;54(11):3140-7.
46. Dandona P, Aljada A, Chaudhuri A, Mohanty P, Garg R. Metabolic syndrome: a comprehensive perspective based on interactions between obesity, diabetes, and inflammation. *Circulation*. 2005 Mar 22;111(11):1448-54.

How to cite this article:

Kishan Prasad HL *et al* (2018) 'Serum Gamma Glutamyl Transferase And Sialic Acid Levels In Type 2 Diabetic Patients And Its Association With Glycemic Control', *International Journal of Current Advanced Research,* 07(5), pp. 12361-12366.
DOI: http://dx.doi.org/10.24327/ijcar.2018.12366.2170

****