1.1 Introduction

Diabetics suffer not only from hyperglycemia, but also the hyperlipidemia and dyslipidemia which puts the patients at an increased risk of coronary artery disease CAD and brain stroke (Koenig and Seneff,2015; Sarli et al.,2013). Persistent hyperglycaemia causes glycosylation of proteins, mainly collagen cross linking and matrix proteins of arterial wall , this eventually leads to endothelial cell dysfunction and further to atherosclerosis (Krishna et al.,2005). The cardiovascular disease is a cause of morbidity and mortality, commonly presents as an abnormally high level of triglycerides (TG), a high proportion of small dense low density lipoprotein cholesterol (LDL-C), low high density lipoprotein cholesterol (HDL-C), and postprandial lipemia (Ginsberg,2006; Haffner,2002; Goldberg,2001).

This pattern of lipid profile in DM type 2 is termed diabetic dyslipidemia.(Rosalki et al.,1971)

Hepatic dysfunctioning has been closely associated with the type 2 DM, obesity and insulin resistance (Kasapoglu et al.,2016; Vijayasamundeeswari and Sudha,2014). Insulin resistance is responsible for the chronic ectopic fat deposition within the hepatocytes, resulting in liver problems (steatohepatitis), and this in turn increases the Gamma glutamyl transferase activity (Kasapoglu et al.,2016; Kasapoglu et al.,2010).

Gamma glutamyltransferase (GGT) catabolises extracellular glutathione (Whitfield, 2001). Although GGT is produced in all tissues, differences in the sugar moieties allow that only the liver GGT is detectable in serum (Huseby,1982). The studies have demonstrated that abnormal hepatocellular function is associated with obesity,insulin resistance, and type 2 diabetes (Rajarajeswari et al.,2014; Sabanayagam et al.,2009; Lim et al.,2007). The loss of a direct effect of insulin to suppress hepatic glucose production and glycogenolysis in the liver causes an increase in hepatic glucose production (Rajarajeswari et al., 2014; Duckworth et al.,1988). Raised liver enzymes reflect chronic ectopic fat deposition (Kasapoglu et al.,2010).

Serum GGT may be a simple and reliable marker of hepatic fat deposition and hepatic steatosis which can lead to hepatic insulin resistance and long term hepatic insulin resistance may lead to type 2 DM (Lippi et al.,2007). Only few studies have compared the serum GGT with lipid profile, hence, in this context, the present study was under taken to study the serum GGT levels and its possible associations with TG, TC, in type 2 DM. Many recent studies have investigated GGT levels and the risk of developing diseases including metabolic syndrome, type 2 diabetes and cardiovascular disease (Lee et al.,2007; Bo et al.,2005; Emdin et al.,2005; Emdin et al., 2002).

1.2 Rationale

Gamma glutamyl transferase evaluation helps in early intervention to prevent diabetic atherosclerosis complication and predicting an early increased risk of developing type 2 diabetes mellitus. Many recent studies have investigated GGT levels and the risk of developing diseases including metabolic syndrome, type 2 diabetes and cardiovascular disease (Lee et al., 2007; Bo et al., 2005; Emdin et al., 2005; Emdin et al., 2002).

To the best of our knowledge, no published data were found regarding association of gamma glutamyl transferase and lipid profile in Sudanese diabetics as predictive markers of cardiovascular disease, that's why we attempt to do this study.

1.3 Objectives :

1.3.1 General objective

1- To investgate the association of gamma glutamyl transferase , cholesterol and triglycerides in type 2 diabetic patients .

1.3.2 Specific objective

1- To measure and compare level of gamma glutamyl transferase , cholesterol and triglyceride in study group .

2- To correlate between level of gamma glutamyl transferase , cholesterol and triglyceride among type 2 diabetics .

3- To define the correlation of gamma glutamyl transferase with in type 2 diabetics .

4- To correlate between that disease duration gamma glutamyl transferase level , cholesterol and triglyceride in diabetic patients .

2.1 Diabetes mellitus: Diabetes mellitus (DM) is a multi-factorial disease which is characterized by hyperglycemia, (elevation of blood glucose level caused by a relative or absolute deficiency in insulin) lipoprotein abnormalities and oxidative stress (Scoppola *et al.,*2001; wild *et al.*,2004). Chronic hyperglycemia is associated with the long-term consequences of diabetes that include damage and dysfunction of the cardiovascular system, eyes, kidney and nerves (wild *et al.*,2004).

2.1.1 Classification of Diabetes Mellitus:

The World Health Organization has described diabetes under the clinical classes of DM and impaired glucose tolerance (IGT). The major classes of DM include:

• Insulin Dependent Diabetes Mellitus (IDDM), known as type l DM.

• Non-Insulin Dependent Diabetes Mellitus (NIDDM), known as type 2 DM.

• Gestational Diabetes Mellitus (GDM).

• Other types of diabetes mellitus associated with specific conditions.

2.1.1.1 Insulin Dependent Diabetes Mellitus (IDDM):

The onset of IDDM or type l diabetes is most common in children or young adults and accounts for around 10% or less o f the total number of people with diabetes (WHO, 1999). Type l indicates the processes of beta-cells destruction that may ultimately lead to diabetes mellitus in which insulin is required for survival to prevent the development of ketoacidosis (acidosis due to an excess of ketone bodies, which accumulate due to the incomplete metabolism of fatty acids), coma and death. An individual with a type l process may be metabolically normal before the disease is clinically manifest, but the process of beta-cells destruction can be detected.

Type l is usually characterized by the presence of anti-glutamic acid decarboxylase (anti-GAD) antibodies, islet cell or insulin antibodies which identify the autoimmune processes that lead to beta-cells destruction. In some subjects with this clinical form of diabetes, particularly non Caucasians, no evidence of an autoimmune disorder is demonstrable and these are classified idiopathic type l. Etiological classification may be possible in some circumstances and not in others. Thus, the category of type l diabetes can be identified if appropriate antibody determinations are performed (WHO,2003).

2.1.1.2 Non -Insulin Dependent Diabetes Mellitus (NIDDM):

The second type of diabetes mellitus is (NIDDM) or type 2 is more complex in etiology and characterized by a relative insulin deficiency reduce insulin action and insulin resistance of glucose transport in skeletal muscle and adipose tissue.
It develops gradually without obvious symptoms and the progression to full diabetes ensues when pancreatic beta-cells hypersecretion of insulin fails to compensate for insulin resistance (Polonsky *et al.*,1996). Type 2 DM usually diagnosed by tests that indicate glucose intolerance, it is linked with behavior (life style), environment and social factor such as over weigh and unhealthy dietary habits and obesity. Patients with type 2 DM have two to fourfold increase in cardiovascular disease (CVD) and dramatically higher risk o f accelerated cerebral and peripheral vascular disease (King *et al.,*1993; Brown, 2000). The metabolic alternation observed in NIDDM are milder than those described for the insulin-dependent diabetes mellitus form of the disease, and are thought to be due to a combination of two factors dysfunctional beta-cells and insulin resistance. The incidence and prevalence of type 2 diabetes mellitus are rapidly increasing worldwide in both developing and developed nations (Amos *et al.,* 1997).

2.1.1.3 Gestational Diabetes Mellitus:

Gestational diabetes is a state of carbohydrate intolerance resulting in hyperglycemia of variable severity, with onset or first recognition during pregnancy. It does not exclude the possibility that the glucose intolerance may antedate pregnancy but has previously gone unrecognized. The definition applies irrespective of whether or not insulin is used for treatment or whether the condition persists after pregnancy.
Women who are known to have diabetes mellitus and who subsequently become pregnant do not have gestational diabetes but have (diabetes mellitus and pregnancy) and should be treated accordingly before, during and after the pregnancy (WHO, 1999). In the early part of pregnancy fasting and postprandial glucose concentrations are normally lower than in normal, non-pregnant women. Elevated fasting or postprandial plasm glucose levels may well reflect the presence of diabetes that antedates pregnancy, but criteria for designating abnormally high glucose concentration at this time in pregnancy have not yet been established. The occurrence of higher than usual plasm a glucose levels at this time in pregnancy mandates careful

management and may be an indication for carrying out an OGTT. Nevertheless, normal glucose tolerance in the early part of pregnancy does not itself establish that gestational diabetes will not develop later. Individuals at high risk for gestational diabetes include older women, obese, women those with previous history of glucose intolerance, any pregnant woman who has elevated fasting or casual blood glucose levels those with a history of gestational diabetes mellitus those with a history of large for gestational age babies, women from certain high risk ethnic groups and strong family history of diabetes mellitus. It may be appropriate to screen pregnant women belonging to high risk population groups during the first trimester of pregnancy in order to detect previously undiagnosed diabetes mellitus. Women at high risk who screen negatively and average risk women should be tested between 24 and 28 weeks of gestation (WHO,1999; WHO,1994).

2.1.1.4 Other Specific Types of Diabetes Mellitus:

Other specific types are currently less common causes of diabetes mellitus, but are conditions in which the underlying defect or disease process can be identified in a relatively specific manner they include:

• Sub classed as obese or non obese DM and they are associated conditions and syndromes.

• Genetic defects in beta-cells, such as maturity onset diabetes of the young.

• Genetic defects in insulin action, such as Leprechaunism.

• Diseases o f the exocrine pancreas, such as cancer of the pancreas, cystic fibrosis and fibrocalculous pancreatopathy (a form of diabetes, which was formerly classified as one type of malnutrition related diabetes mellitus).

• Endocrinopathies, such as cushing syndrome, acromegaly and phaeochromocytoma.

• Drugs or chemicals, such as steroids and thiazides.

• Uncommon forms of immune related diabetes, such as the type associated with insulin receptor antibodies.

• Other rare genetic syndromes associated with diabetes, such as Klinefelter syndrome and Down syndrome (WHO,1999).

• Malnutrition related diabetes mellitus (MRDM) it is associated with nutritional deficiency and is 'seen in tropical developing countries (William and Pickup,1992).

2.1.2 Pathogenesis of Diabetes Mellitus:

2.1.2.1 Type I

2.1.2.1.1 Antibodies

The most practical markers of B –cell auto immunity are circulating anti bodies that can be detected in the serum years before the onset of hyperglycemia. The most important Abs islet cell cytoplasm Abs (ICA), to glutamic acid decarboxylase (anti – GAD) (Carl,Edward,2001) .

2.1.2.1.2 Genetics

Susceptibility to type I diabetes is inherited, but the mode of inheritance is complex and has not been define. The concordance rate between identical twins isapproximately 30 %.(Carl,Edward,2001) .

2.1.2.1.3 Environment

Various studies have indicated that environmental factors are involved in the initiation of diabetes. Viruses such as rubella, mumps have been implicated. Other environmental factors that have been suggested include chemicals and cow's milk (Carl,Edward,2001) .

2.1.2.2 Type II

Al least two major identifiable pathological defects exist in individuals with type II diabetes .One is a decreased ability of insulin to act on the peripheral tissues. This defect is called insulin resistance and it thought by many investigators to be the primary underlying pathological process. The other is B – cell dysfunction, which is an ability of the pancreas to produce sufficient insulin to compensate for the insulin resistance. Type II diabetes mellitus clearly is can extremely heterogeneous disease, and no single cause can adequately explain the progression from normal glucose tolerance to diabetes. The fundamental molecular defects in insulin resistance and insulin secretion result from a combination of environmental and genetic factor (Carl,Edward,2001).

2.1.2.2.1 Genetic

The concordance rate for type II diabetes in identical twins approaches 100% Type II diabetes is 10 fames more likely to occur in an obese individual with adiabatic parent than in an equally obese individual without diabetic family history (Carl,Edward, 2001) .

2.1.2.2.2 Environment

Environment factors, such as diet and exercise, are important determinants in the pathogenesis of type II diabetes. Convincing development of type II diabetes, but the association is complex. Although 60% to80 % of those people, even those with normal carbohydrates tolerance, have hyperinsulinemia and are insulin resistant. Other factors, such as family history of type II diabetes, the duration of obesity, and the distribution of fat, also are important. The mechanism of the protective effect of exercise is thought to be an increased sensitivity to insulin in skeletal muscle and adipose tissues (Carl,Edward,2001) .

2.1.2.2.3 Loss of B- cell function

The increase B- cell demand induced by insulin resistance is ultimately associated with a progressive loss of B-cell function that is necessary for the development of fasting hyperglycemia (Godkar,2003) .

2.1.2.2.4 Insulin resistance

Insulin resistance is defined as decreased biological response to normal conc. Of circulating insulin and is found in both obese, non diabetic individuals and those with Type II diabetes (Carl,Edward,2001) .

2.1.3 Signs and symptoms of diabetes mellitus:

- Fatigue (due to low entry of glucose in muscle and depletion of its glycogen stores).
- Polyuria: large urine volume due to loss of glucose in urine (osmotic dieresis)
- Polydipsia (excessive thirst due to hyperglycemia and hyperosmolarity caused by the polyuria).
- Hyperphagia (increase appetite due to decrease utilization of glucose by the cells of the ventromedial nuclei of the hypothalamus (satiety center) thus allowing the appetite area to work without inhibition.
- Weight loss due to; loss of the energy substrate (glucose) in urine and its failure to enter the cell; the cell use protein as alternative source of energy.
- Coma (occurs due to many causes: diabetic keto-acidosis, non ketotic hyperosmolar coma, lactic acidosis and rarely brain edema).
- Diabetic patient may also present with hypoglycemia coma due to wrong higher dose of insulin or increased exercise with low good intake. This is usually fatal if not treated immediately.

- Kussumaul breathing rapid deep respiration due to acidosis as in diabetic Keto acidosis (Haki,2008) .

2.1.4 Diagnosis of diabetes mellitus:

The diagnosis of type I diabetes and many cases of type II, is usually prompted by recent – onset symptoms of excessive urination (polyuria) and often accompanied by weight loss. Diabetes mellitus is characterized recurrent or persistent hyperglycemia and diagnosed by:

1. Fasting blood glucose.
2. 2hours post prandial.
3. Glucose tolerance Test GTT (Haki,2008) .

2.1.5 Monitoring of Diabetes:

Good control of blood glucose concentration is important, particularly in younger patient, as this helps to prevent the development of long term complication this is done by:

1. Urine analysis: Urinary glucose gives an induction of integrated blood glucose concentration above renal threshold since the bladder was last emptied. Self – monitoring in IDDM is more usually under taken by blood glucose monitoring.
2. Blood glucose analysis.
3. Glycated hemoglobin: hemoglobin in most normal adult consists of one major component. Several minor hemoglobin fractions including glycated hemoglobin. This is formed after reticulocytes have been released from bone marrow by the action of glucose on hemoglobin. Several different HbA are found. The major fraction being HbA1c in which glucose binds to the terminal valine of B chain. Normally, about 5% of circulation hemoglobin is glycated, the amount depending on the overage blood glucose concentration over the previous 2 month.

- The measurement of glycated hemoglobin therefore gives an indication of overall degree of blood glycaemia control in contrast to glucose measurement which gives information for a single time point (Laker,1996) .

2.2 Gamma-glutamyl transferase:

GGT is found in the kidneys, biliary system, pancreas, and intestine (Dufour,2000).

Briefly, GGT protein catalyzes an enzymatic action, which is the transfer of a glutamyl residue to an acceptor through the glutamate's gamma carboxylic acid to an

amine or other amino acid. The most abundant natural substrate is glutathione.

Glutathione is extracellular and cannot pass through the cell membrane. Glutathione can be broken down into 3 amino acids (including cysteine, which may be deficient in low-protein diets) at the cell membrane by GGT. These amino acids can be taken up in the cells by the γ-glutamyl cycle.

Glutathione is then reformed in the cells, where it protects cells against oxidants that are produced during normal metabolism. An increased need for reduced glutathione occurs with oxidative stress (Zhang et al.,2005; Lee et al.,2004; Whitfield.,2001).

Nonalcoholic fatty liver disease has been linked to a higher prevalence of diabetes (Bloomgarden, 2005, p. 1519).

Oxidative stress resulting from nonalcoholic fatty liver disease (NAFLD) has been suggested in the mechanisms of insulin resistance, β-cell dysfunction, poorly-controlled type 2 diabetes, and subsequent complications (Wright et al.,2006; Bo et al.,2005; Thamer et al.,2005;Robertson et al.,2004).

GGT 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 (Seitz,2006; Lee et al.,2005). Research by Jimenez- Alonso et al., (1983) showed that hyperglycemia itself does not increase the hepatic enzyme GGT in uncontrolled diabetics. Although most researchers report that GGT appears to be a marker for oxidative stress, there is some controversy regarding the role that GGT plays in oxidative stress.

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 (Meisinger et al.,2005; Zhang et al., 2005; Lim et al., 2004). However, others including Lee, Blomhoff et al., (2004) have noted 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.

Numerous studies have 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 (Yamada et

al., 2006; Bo et al., 2005; Emdin et al., 2005; Sakuta et al., 2005; Wannamethee et al., 2005; Lee, Jacobs et al., 2003; Emdin et al., 2002; Whitfield., 2001). Meisinger et al.,(2005) postulated that possible mechanisms by which GGT is a marker for increased risk of type 2 diabetes include the following: (a) elevated serum GGT could indicate excess fat deposits in the liver, which may cause hepatic insulin resistance and increase the risk of type 2 diabetes by contributing to systemic insulin resistance; (b) increased GGT is a marker for oxidative stress; and (c) increased GGT may be the 5 expression of inflammation.

(Ortega et al., 2006) reported that GGT was a significant predictor of insulin resistance independently of weight, BMI, or percentage of fat in Pima Indian children. Many studies have reported an increased risk of type 2 diabetes with increased levels of GGT. Lee, Ha, et al., (2003) prospectively studied a group of 4,088 healthy, male Korean workers and found a strong dose response relationship between serum GGT levels at baseline and incident type 2 diabetes after 4 years of follow up. This relationship was observed even in nondrinkers. Nakanishi et al., (2004) found that increased serum GGT increased the risk of incidence of metabolic syndrome and type 2 diabetes in 3,000 middleaged Japanese male office workers. Lee, Silventoinen et al., (2004) evaluated 20,158 Finnish subjects of both genders, aged 25-64, in a prospective cohort study and found that higher serum GGT was directly associated with an increased risk of type 2 diabetes. The CARDIA study (Lee, Jacobs, et al., 2003) recruited Black and White Americans in 1985-86 and followed them for 15 years. They reported that the risk of type 2 diabetes was strongly increased with higher normal levels of GGT. In addition, they postulated that this may be related to oxidative stress. Perry, Wannamethee, and Shaper (1998) examined the
association between GGT levels and the risk of NIDDM in about 7,500 British men (aged 40-59). Their findings suggested that a raised serum GGT level is an independent risk factor for NIDDM. Wannamethee et al., (2005) conducted a prospective study of 3,500 of the surviving men from the previous study (now aged 60-79). They reported that both ALT and GGT were independent predictors of type 2 diabetes in older men and could be useful in predicting those at high risk of diabetes.

2.3 Normal lipids Metabolism:

Lipids defined as biological substances that are generally hydrophobic in nature and in many cases soluble in organic solvents (Smith,2000). These chemical properties cover a broad range of molecules, such as fatty acids, phospholipids, sterols, sphingolipids, terpenes, and others (Christie,2003). Lipid classes are fats, oils, waxes, and complex lipids involved in various biological processes such as sterols, phospholipids, glycolipids, lipoproteins and sphingolipids (Vilhemsen et al.,2005).

Lipids are first absorbed from the small intestine and emulsified by bile salts which are synthesized from cholesterol in the liver, stored in the gallbladder and secreted following the ingestion of fat. As an emulsion dietary fats are accessible to pancreatic lipase. The products of pancreatic lipase, i.e. free fatty acids (FFA) and a mixture of monoacylglycerols (MG) and diacyl glycerols (DG) from dietary TG diffuse into the intestinal epithelial cells where the resynthesis of triacyglycerols occurs.

Lipids are insoluble in plasma, thus their transport is mediated by lipoproteins which differ in particle size, composition and density. These are chylomicrons (CYM), very low density lipoproteins (VLDL), low density lipoproteins (LDL) and high density lipoproteins (HDL). All of them have a hydrophobic core containing TG and cholesteryl ester (CE) and a polar periphery with phospholipids (PL),cholesteryl (C) and apolipoproteins (Mathews et al., 2000).

2.3.1 Cholesterol:

Cholesterol is an unsaturated steroid alcohol containing 4 rings (A, B, C and D) it has single C-H side chain tail similar to fatty acid in the physical properties. It is oriented in lipid layers, and can be exist in an esterified form called cholesterol ester (CE).

Cholesterol has three types low-density lipoprotein (LDL-C) often called “bad” cholesterol because it carries cholesterol to the tissues of the arteries, causing plaque to build up and the blood vessels to narrow, high-density lipoprotein (HDL-C) it called “good” cholesterol because it helps to keep cholesterol from building up inside your blood vessels and keeps them from getting blocked higher levels of HDL can reduce the risk of cardiovascular disease and very-low density lipoprotein (VLDL C) this form contains the highest amount of triglyceride like LDL, this is considered “bad cholesterol.” A value less than 32 mg/dl is desirable. VLDL is usually not measured

directly and it can be calculated from the other lipoprotein concentrations (Bishop et al., 2004).

In diabetes mellitus the plasma cholesterol level is usually elevated and this plays a role in the accelerated development of the atherosclerotic vascular disease that is a major long term complication of diabetes in human. The rise in plasm a cholesterol level is due to an increase in plasm a concentration of VLDL and LDL, which may be due to increase hepatic production of VLDL or decrease removal of VLDL and form the circulation (Ganong,2003).

Hypercholesterolemia in diabetic patients is characterized by high levels of triglycerides (hypertriglycerides), high levels of small LDL particles and low levels of HDL. So dietary intake appears to be one of the most important factors to control level of lipid (Van Dam et al.,2002; Feskens et al.,1995). In addition to Physical activity, it decreases both BMI and central fat accumulation (Gilliat-Wimberly et al., 2001) and can partly counterbalance the negative age related changes in lipid spectrum and increase in BMI by diminution of HDL decrease. The changes in lipids due to physical activity are largely independent of changes in body weight (Owens et al., 1992).

2.3.2 Triglycerides:

Triglycerides is the most common type of lipid formed in animals it contain three fatty acid molecules attached to one molecule of glycerol by ester bond and containing saturated fatty acid which' do not have kinks in their structure, pack together more closely and tend to be solid at room temperature. In contrast triglycerides containing cis unsaturated fatty acid with bends in their structure, typically from oils at room temperature (Bishop,2004). Triacylglycerol (TG) is stored in lipid droplets in the cytoplasm of skeletal muscle. They can be mobilized by catecholamine, exercise and electrical stimulation the exercise induced decrease of TG can be reduced by Beta-adrenergic blockade. The effect of catecholamine on intramuscular TG is compatible with a role o f hormone sensitive lipase (HSL) in muscle. A value below 150 mg/dl indicates no increased risk, 150 -200 indicates a slight risk, and over 200 mg/dl is a high risk.

Recent studies have demonstrated that in diabetic patients TG levels is a risk factor for CVD independent o f HDL-C level and despite glycemic control, the incidence of macrovascular disease is increased two to five-fold in diabetics as compared to nondiabetic patients. This is attributed mainly to diabetic dyslipidemia (Stamler et al., 1993).

2.3.3 Diabetes Mellitus and Lipids:

In DM changes in lipid levels and consequent disorders of lipid metabolism and stress have been observed (Betteridge, 1994). Such as increases in circulating levels of free fatty acids (FFA), triglycerides and dense low-density lipoprotein cholesterol particles together with reduced levels of high-density lipoprotein cholesterol levels (Haffner, 1998). It is play an important role in pancreatic cell responses (Yaney and Corkey, 2003). FFA provided exogenously or produced in the cell are essential to maintain proper nutrient induced insulin secretion. Acutely FFA generates an increase in glucose induced insulin secretion, whereas chronic exposure to elevated lipids results in cell exhaustion, impaired secretory response to glucose, and eventually, induction of cell apoptosis (Prentki et al., 2002).

The principles abnormal of lipid metabolism in diabetes are acceleration of lipid catabolism, with increased formation of ketone bodies and decreased synthesis of fatty acid and triglycerides. The manifestations of the disordered lipid metabolism are so prominent the diabetes has been called more a disease of lipid than of carbohydrate metabolism. Fifty percent of an ingested glucose load is normally burned to C02 and H20 5% is converted to glycogen and 30-40% converted to fat in fat depots.

In diabetes less than 5% converted to fat even though the amount burned to C02 and H20 is also decreased and the amount converted glycogen is not increased. Therefore, glucose accumulates in the bloodstream and spills over into the urine (Ganong, 2003).

Recent study reported that insulin increases the number of LDL receptor so chronic insulin deficiency might be associated with a diminished level of LDL receptor. These cases the increase in LDL particles and result in increase in LDL cholesterol value in diabetes mellitus (Suryawanshi et al., 2006).

Sudan University of Science & Technology

Faculty of Medical Laboratory Sciences

Questionnaire

Association of gamma glutamyl transferase and lipid profile in Sudanese type 2 diabetics patients .
For M.SC degree

- Name : _____
- Sex : _____
- Age : _____
- height : _____
- weight : _____
- BMI : _____
- Duration of disease : _____
- Type of treatment (if any) : _____
- Do you have diabetic parents :

  Yes ( ) No ( )

- If yes : define :

  Father ( ) mother ( ) both ( )