

ASSOCIATION OF AMYLASE ACTIVITY WITH SOME BIOCHEMICAL VARIABLES IN UNCONTROLLED TYPE 1 IRAQI DIABETIC PATIENTS

Khalidah S. Merzah

Department of Medical Laboratory Techniques, Asoul el Deen University College, Iraq.

e-mail : kmerzah@gmail.com

(Received 26 October 2019, Revised 8 January 2020, Accepted 19 January 2020)

ABSTRACT : This study aims to find the relationship among fasting blood glucose (FBG), glycated hemoglobin (HbA1c) and pancreatic amylase activity serum of 50 uncontrolled type 1 diabetes children and 30 non-diabetic healthy control children. From each fasting patient, five milliliters of venous blood were drawn. The diabetic patients had a similar average age to that of control (4-12 years). The results shows that FBG and HbA1c levels were significantly increased whereas serum amylase activity was not significantly increased in type 1 diabetes mellitus (DM) when compared with the corresponding activities of the control subjects. These findings may explain the role of defect of antioxidants and the increased end products advanced glycation (AGE) and the repercussions of the vascular system in people with diabetes. The oxidative stress will increase in both types of diabetes, but it is more in non-insulin dependent. It has been noted that uncontrolled type 1 diabetes is associated with endothelial dysfunction and may lead to future microvascular complications. Some studies have confirmed that type 1 diabetes is the result of an autoimmune reaction with pancreatic island cell antigens. Therefore low insulin secretion can be caused by the autoimmune destruction of the pancreas. Loss of insulin secretion that will lead to metabolic imbalances associated with IDDM. It can be seen that patients with pancreatic insufficiency are at risk for diabetes because pancreatic duct obstruction and gastrointestinal enzyme damage lead to fibrosis or scarring of the pancreas.

Key words : Amylase enzyme, diabetic children.

INTRODUCTION

Diabetes mellitus is a disorder of the metabolic homeostasis controlled by insulin, resulting in abnormalities of carbohydrate and lipid metabolism. Diabetes type 1 caused by an absolute insulin deficiency, as a result of a loss of the insulin-producing beta cells of the pancreas (David and Leslie, 2008).

Diabetes is one of the most common endocrine diseases and has a significant relationship to metabolic disorders determined by chronic hyperglycemia.

In addition to disturbances in carbohydrates, fats and protein metabolism caused by defects of insulin secretion or the effectiveness of insulin or both (Taylor, 1999).

DM is a disease of universal distribution, affecting individuals of different ages with widely prevalence rates vary in several societies and in the community. Epidemiologic studies showed an increase recurrence related to variables in way of living, civilized level, type of food, weight gain and psychological stress are among the factors assumed in this direction for glucose intolerance and diabetics in some societies (Owen and

Shuman, 1990).

The major pathway for the breakdown of polysaccharides to oligosaccharides and accomplished by a battery of carbohydrates in the brush border of the mature enterocytes in the hydrolysis by secreted enzymes (Roy, 1994).

By the pancreas, the amylase is excreted in the small intestine. Carbohydrates are broken down and converted into oligosaccharides by the amylase enzyme. Monosaccharide units are broken down by the glucosidase enzyme. Monosaccharides will only be absorbed into the body (Atkinson and Maclaren, 1994; American Diabetes Association, 2004; Goruch *et al*, 1983). Through the hepatic portal vein, glucose and other monosaccharides are transported to the liver (Bosi, 1987). The abnormally high concentration of blood sugar is the most common feature of metabolism (Firoozrai *et al*, 2007). This condition is due to abnormally high glucose production or low glucose use (Saravanan and Pari, 2008). High blood sugar concentration is due to both of these processes. Secondary complications that can be observed in diabetics