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ANALYSIS OF LEVEL OF MUSCLE GLYCOGEN CONTENT IN TYPE 2 DIABETES IN PAKISTAN

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Abstract:
Introduction: *Glycogen metabolism is controlled predominantly by the coordinated action of two enzymes, glycogen synthase and glycogen phosphorylase, both of which are regulated by phosphorylation and allosteric modulators.* ***Aims and objectives:*** *The basic aim of the study is to analyze the level of muscle glycogen content in type 2 diabetes in Pakistan.* ***Methodology of the study:*** *This cross sectional study was conducted in Holy Family Hospital Rawalpindi during May 2018 till November 2018. The data was collected from 100 diabetic patients who visited the OPD of the hospital. 5cc blood sample was collected from each patients.Blood was centrifuged and separated the serum for further analysis. Serum creatine phosphorkinase (CPK) levels were measured to exclude skeletal muscle disorder. The EDL muscle of all patients was assessed for glycogen content, using an anthrone based method. Serum free carnitine levels were measured from blood serum using L-carnitine assay kit.* ***Results:*** *The data was collected from 100 diabetic patients. The mean age of the selected patients was 55.66±4.56 years, fasting serum TG (101.38 ± 7.15mg/dl), fasting serum HDL (79.65 ± 5.11mg/dl) in all selected patients. The serum free carnitine was measured by terminal sampling and glycogen content of the EDL muscle were calculated. An increase in glycogen content was observed in diabetic group (82.55±10.30 mg/100 gm muscle). The serum free carnitine levels showed a significant rise in diabetic group.*

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INTRODUCTION:

Glycogen metabolism is controlled predominantly by the coordinated action of two enzymes, glycogen synthase and glycogen phosphorylase, both of which are regulated by phosphorylation and allosteric modulators [1]. Insulin promotes the net dephosphorylation of both glycogen synthase and glycogen phosphorylase through the inhibition of protein kinases and the activation of protein phosphatases. Among the protein kinases, glycogen synthase kinase–3 (GSK-3) is thought to be an important target for insulin in its stimulation of glycogen synthase activity. Among the protein phosphatases, protein phosphatase 1 (PP1) has been implicated in this action of insulin [2]. Glucose and lipid metabolism are inter related phenomenons in the human body. The number of people with T2DM is spiking worldwide, thus efficient management is required to improve the quality of life of diabetics. Initial management of T2DM consists of weight reduction, regular exercise and controlled diet. Exercise plays a dominant role in controlling hyperglycemia by way of increase in peripheral insulin sensitivity, strengthening insulin bonding and minimizing obesity [3].

Both glucose and free fatty acids (FFA) are consumed by skeletal muscles as sources of fuel for energy production. During the fasting state, FFAs provide major source of energy production, as skeletal muscle glucose uptake is considerably low. After uptake of glucose, insulin secretion from the beta cells of pancreas is stimulated leading which lowers the rate of lipolysis leading to a reduction in plasma FFA levels [4]. Simultaneously, there is a rise in the rate of glucose oxidation in muscle. This transition from fatty acid oxidation to glucose oxidation is called metabolic flexibility [5]. After transfer of glucose into the muscle cells through of GLUT-4 transporter (glucose transporter 4), it is phosphorylated by hexokinase, and then either oxidized by glycolytic pathway or stored as glycogen. As the insulin levels rise, glycogen synthesis rate also improves, i.e; about 70% of glucose is converted to glycogen [6].

Aims and objectives

The basic aim of the study is to analyze the level of muscle glycogen content in type 2 diabetes in Pakistan.

METHODOLOGY OF THE STUDY:

This cross sectional study was conducted in Holy Family Hospital Rawalpindi during May 2018 till November 2018. The data was collected from 100 diabetic patients who visited the OPD of the hospital. 5cc blood sample was collected from each patients. Blood was centrifuged and separated the serum for further analysis. Serum creatine phosphorkinase (CPK) levels were measured to exclude skeletal muscle disorder. The EDL muscle of all patients was assessed for glycogen content, using an anthrone based method. Serum free carnitine levels were measured from blood serum using L-carnitine assay kit.

Data Analysis

Data was analysed using SPSS 17. Mean and standard deviation were calculated for all values. Statistical significance of difference between the subgroups was determined by applying independent samples t-test.

RESULTS:

The data was collected from 100 diabetic patients. The mean age of the selected patients was 55.66±4.56 years, fasting serum TG (101.38 ± 7.15mg/dl), fasting serum HDL (79.65 ± 5.11mg/dl) in all selected patients. The serum free carnitine was measured by terminal sampling and glycogen content of the EDL muscle were calculated. An increase in glycogen content was observed in diabetic group (82.55±10.30 mg/100 gm muscle). The serum free carnitine levels showed a significant rise in diabetic group.

Table 01: Serum free carnitine levels and muscle glycogen content of groups

Variables	Diabetic patients	Control group	P-value
Carnitine levels (nmol/µl)	0.109 ± 0.014	0.312 ± 0.158	$p < 0.001$
Glycogen content (mg per 100gm muscle)	82.55 ± 10.30	124.20 ± 17.78	$p < 0.001$

All values are expressed as Mean ± SD

DISCUSSION:

The mechanisms that account for IR of Glyc synthesis include impaired glucose transport, elevated plasma fatty acids and IMCL, impaired insulin signaling, and reduced activation of glycogen synthase. Because of the severe impairment in rates of insulin-stimulated Glyc formation in skeletal muscle in T2DM and Ob subjects, it seems logical to postulate an equally severe reduction in muscle Glyc. Yet prior studies indicate a relatively modest reduction in muscle Glyc in T2DM, which in itself is intriguing [7]. Therefore, we reexamined this issue, taking into account fiber type, IMCL, and OX-Enz, factors not assessed in the cited studies [8].

Studies by Tamamogullari *et al*. had determined the levels of total, free and esterified carnitines in humans and observed that the levels of these carnitines were decreased in diabetic patients [9]. Free carnitine levels were found to be lower in diabetics while esterified carnitines were found higher in diabetics. L- carnitine supplementation in the carnitine group of our study probably enhanced the PDH activity by buffering the excess acetyl CoA [10].

CONCLUSION:

It is concluded that Levo carnitine increases the glucose uptake by the skeletal muscle and improves the skeletal muscle glycogen stores in type 2 diabetes mellitus.

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