

# Evaluation of Serum Creatine kinase Activity in Sudanese Patients with Type1 and Type2 Diabetes Mellitus in Khartoum State

Nbra Hashim Mohamed<sup>1</sup>, Rihab Akasha<sup>2\*</sup>

<sup>1</sup>Department of Clinical Chemistry, Faculty of Medical Laboratory Sciences, Al-Neelain University, 52nd St, Al Khurtum, Sudan

<sup>2</sup>Department of Clinical chemistry, faculty of Medical Laboratory Science, International University of Africa, Madani St, Al Khurtum 12223, Sudan

DOI:10.21276/sjbr.2019.4.6.2

| Received: 15.06.2019 | Accepted: 26.06.2019 | Published: 30.06.2019

\*Corresponding author: Dr. Rihab Akasha

## Abstract

**Background:** Diabetes mellitus is a standout amongst the most widely recognized metabolic disarranges that follow by many complications and influence many body systems and organs make them less effective and these incorporate skeletal muscles. **Aim:** The aim of this study is to evaluate serum creatine kinase activity in Sudanese patients with type1 and type2 diabetes mellitus in Khartoum state. **Material and methods:** This study was case-control study and conducted during the period from April 2018 to November 2018, 90 participants (30 type1DM, 30 type2 DM as case and 30 controls). The samples was collected by simple random sampling technique through a self-administering questionnaire, Concentration of glucose was measured for each patient with GOD method, and activity of total creatine kinase was determined also CKMB activity was measured for the tow study groups and control group as marker of cardiac functions. The analysis of data done by SPSS version 21 using T-test for comparing mean and simple correlation for correlation of continuous numerical data. **Results:** There was a significant increase in glucose concentration in compare to control group p-value 0.000 in both type1and type2 diabetic patient compare to control mean±SD type1 (241.9±77.5) type2 (211.3±95.1) control group (111.1±36.4). There was a significant increase in activity of total creatine kinase (CK) enzyme in type1 and type2 diabetic patients in compare to healthy individuals the p-value 0.019 and 0.010 respectively mean±SD type1 (78.0±13.2 IU/L) type2 (71.6±10.0 IU/L) and control group (58.4±23.9 IU/L). There was positive insignificant correlation between the activity of total CK and age in type1 DM patient (R-value= 0.197, P-value= 0.297), but negative insignificant correlation between the activity of total CK and age in type2 DM (R-value= -0.269, P-value= 0.151). Positive insignificant correlation between duration of diabetes and the activity of total CK level in type1 (R-value=0.164, P-value=0.386) and type 2 (R-value=0.158, P-value=0.403). Negative significant correlation between glucose and total CK in type1 diabetes mellitus (R-value= -0.410, P-value=0.024) and positive insignificant correlation between glucose and total CK in type2 diabetes mellitus (R-value=0.091, P-value=0.633). **Conclusion:** The activity of total CK among diabetic patient with type1 and type2 diabetes mellitus was higher when compare to control group, there was also significant correlation between glucose and activity of total CK in type1 diabetes mellitus.

**Keywords:** Creatine kinase, diabetes mellitus, Khartoum state.

**Copyright © 2019:** This is an open-access article distributed under the terms of the Creative Commons Attribution license which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use (NonCommercial, or CC-BY-NC) provided the original author and source are credited.

## INTRODUCTION

Diabetes mellitus is gathering of metabolic issue the principle imperfection is in carbohydrate metabolism so it describe by underutilization of glucose prompting increment in blood glucose (hyperglycemia) and this deformity happen because of complete inadequacy of insulin (type1 diabetes mellitus) or insulin resistance (type2 diabetes mellitus) [1]. WHO characterize diabetes mellitus as fasting plasma glucose focus 7.0 mmol/L or more and random plasma glucose 11.0 mmol/L or more and this definition depends on research facility findings [2]. In creating nations diabetes mellitus is fourth most regular reason for death 1– 2% of Western Populations are influenced by

diabetes. Numerous patients can't perceive that they are diabetic already which can prompt most more regrettable diabetes complication and patient could achieve the healing center in late complexity or even in coma [3]. Furthermore, it is relied upon to establish 552 million diabetics by 2030 and the diabetic patients that previously evaluated before 285 to 366 cases. The predominance is 9.3% - 30% undiscovered diabetic in united states between 1999 to 2002 and these number is consider as immense increment, in 2010 the pervasiveness is turned out to be 11.3% in populace more than 19 year and over 25% of case in found in age more than 56 year. In UK 3 million individuals are determined to have diabetes mellitus [1]. Diabetes

mellitus order already base on treatment to type I insulin dependent (also juvenile onset) and type II insulin non-dependent (also adult onset) yet every one of these names are not utilize anymore and it better to group base on cause as opposed to treatment so the last characterization by American Diabetes Association (ADA) to Type 1 diabetes, Type 2 diabetes, Other explicit kinds of diabetes and Gestational diabetes mellitus (GDM) [4]. Ordinarily Glucose uninhibitedly enter  $\beta$  cell by GLUT2 and repress ATP delicate  $K^+$  channels by increment ATP creation and after that calcium convergence will increment due depolarization of  $\beta$  cell membrane this lead to exocytosis of insulin [5] and afterward insulin begin to encourage the passageway of glucose to other body cells to deliver vitality need to work well. Diabetes mellitus can pursue by many complication result from the mechanism of the illness (either insulin resistance or complete absence of insulin) and these complications can be partitioned into two classes 1) microvascular entanglements and this incorporate retinopathy, nephropathy and neuropathy and 2) macrovascular complications this incorporate coronary illness (like atherosclerosis or myocardial infarction) and stroke [1]. These complexities can result from uncontrolled long standing diabetes. The greater part of diabetes inconveniences make it one of the principal reasons for death overall like cardiovascular malady which additionally consider as significant reason for death among diabetic [6]. Next to the deformity on carbohydrate metabolism other metabolic pathway can influence like protein metabolism and lipid metabolism and this imperfections lessen measure of vitality that explicit cells needs to capacity will like muscle cell, for instance In diabetes lipid peroxidation and oxidative pressure will increment due hyperglycemia this lead to build free radicals which can prompt numerous microvascular and microvascular intricacies [7]. Skeletal muscle is one of cells that effect by insufficiency or opposition of insulin so decline use of glucose prompting lessen measure of vitality that muscle need and prompting muscle damage.

What's more, expanding in dimension of free unsaturated fat due lipolysis increment peripheral tissue resistance to insulin likewise proteins deformity influence skeletal muscle capacity to deliver vitality required. Muscle deformity can result in arrival of specific enzymes that found ordinarily intracellular and consider as muscle harm biomarker for instance creatine kinase is Cytosolic compound aides mitochondria in phosphate exchanging through cytoplasm as wellspring of high vitality [8]. it have three isoenzyme CK-MM which found in muscles and consider as one of muscle harm biomarker that discharge to blood if there should be an occurrence of muscle cell harm so in diabetic patient the dimension of all out creatine kinase will increment due rise of this isoenzyme and the wellspring of this expansion is devastation in skeletal muscle that outcome from diminish of vitality. Other isoenzyme CK-MB

(myocardial cell) and CK-BB (cerebrum) are ordinary [9]. So the source of hoisted complete CK in diabetic is skeletal muscle harm primarily CK-MM isoenzyme without any heart problem.

## MATERIALS AND METHODS

This study was a case-control study and conducted during the period from April 2018 to November 2018, 90 participants (60 patients 30 with type 1 DM and 30 with type 2 DM as cases and 30 healthy individual as controls), gender and age was matched (case and control aged from 18 to 85 years, males and females). Blood samples were collected from Ibrahim Malik Teaching hospital (Khartoum state-Sudan), international university of Africa clinic. All patients with both type 1 and type 2 diabetes mellitus were included in this study, while patients with thyroid disease, muscles disease and myocardium disease were excluded. This study was approved by the ethical committee of Medical Laboratory Sciences, Clinical Chemistry Department – AlNeelain University. Subjects involved in this study were informed by the aims of the study and its importance, and verbal informed consent was obtained from each participant. The whole blood samples collected in comfortable condition then plasma and serum was separated. The levels of plasma glucose and serum total CK and CKMB activity were measured using creatine kinase kits from Biosystem Company Costa Brava, 30, Barcelona (Spain). Data were analyzed using SPSS version 21. The results were expressed as percentage, Mean and SD. Independent T-test was performed to compare the study parameters in case versus control groups. Correlation was done to study the relationship between study parameters and study variables. The p-value less than 0.05 were considered significant.

## RESULTS

The results after analysis showed that there was a significant increase in the level of glucose in type 1 and type 2 in compare to control group with p-value (0.000), and the activity of total creatine kinase was significantly increased in diabetic patient with both type 1 and type 2 diabetic in compare to control group with p-value (0.019, 0.010) respectively (table 1). There was positive insignificant correlation between the level of glucose and age in both study groups with p-value (0.229 for type 1 and 0.128 for type 2), positive insignificant correlation between the activity of total CK and age in type 1 DM patient p-value (0.297) but negative insignificant correlation between the activity of total CK and age in type 2 DM p-value (0.151) (table 2). Positive insignificant correlation between duration of DM and the activity of total CK level in type 1 with p-value (0.386) and type 2 with p-value (0.403) (Table-3). Negative significant correlation between glucose and total CK in type 1 diabetes mellitus with p-value (0.024) (Figure-1) and positive insignificant correlation between glucose and total CK in type 2 diabetes mellitus with p-value (0.633) (Figure 2).

**Table-1: Show the mean study parameters in case versus the control group**

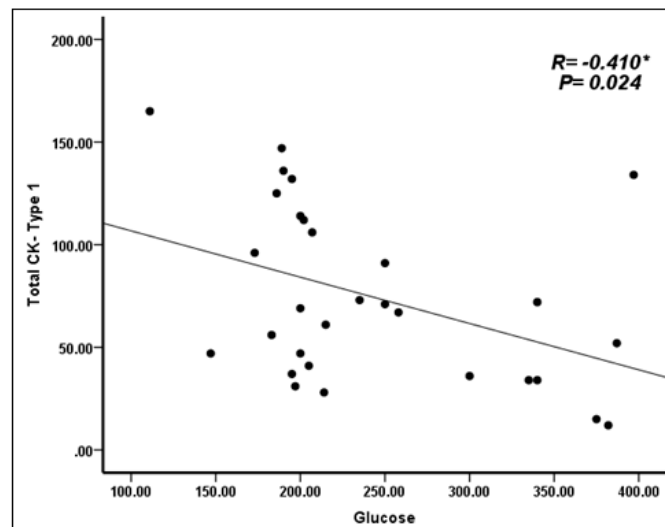
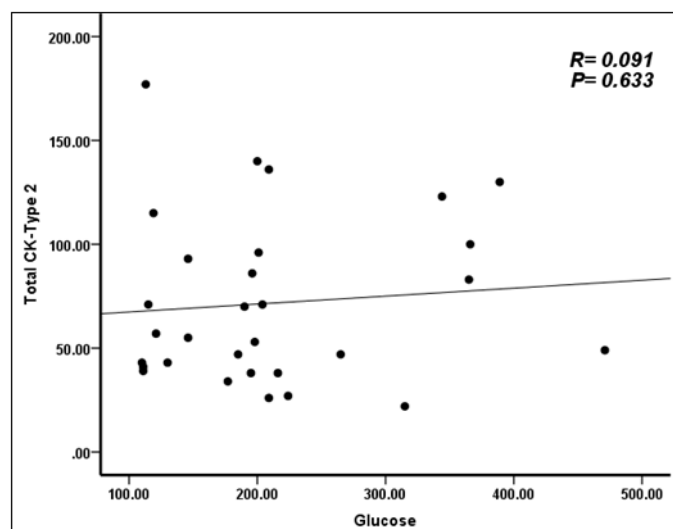
	Type1	Type2	Control
Glucose	241.9±77.5	211.3±95.1	111.1±36.4
p-value	0.000	0.000	
Total CK	78.0±13.2	71.6±10.0	58.4±23.9
p-value	0.019	0.010	

**Table-2: Show the correlation between study parameters and age**

	Type1		Type2	
	R- value	P-value	R- value	P-value
Glucose	0.226	0.229	0.284	0.128
Total CK	0.197	0.297	-0.269	0.151

**Table-3: shows Correlation between duration of DM and the activity of total CK level**

	Type1		Type2	
	R- value	P-value	R- value	P-value
Total CK	0.164	0.386	0.158	0.403

**Fig-1: Show correlation between glucose and the activity of total CK level (DM type 1)****Fig-2: Show correlation between glucose and the activity of total CK level (DM type 2)**

## DISCUSSION

In this study, total CK activity was increase in diabetic patient with either type1 or type2 diabetes mellitus in compare to control group with p-value 0.019 and 0.010. This finding was in agreement with results of previous study done by Adlija *et al.*, [10] which reported that patients with diabetes mellitus with type1 or type2 showed elevated activity of total CK. It also agreed with Akram *et al.*, [11] who conclude that total CK is elevated in patient with type2 diabetes. This may occur due to several factors including defect in Krebs cycle which result from increasing in proteolysis in diabetic. Defect in this cycle beside other metabolic pathway like respiratory chain affects the morphological appearances and decrease the function of mitochondria so that the amount of ATP production will decrease in skeletal muscle cells [10]. The skeletal muscles of diabetic patients with type 2 diabetes mellitus contain small mitochondria than the normal so the ability of producing energy is impaired [12]. Decrease in synthesis of creatine phosphate well occur as result of low ATP and this can lead to (AMP activated protein kinase) activation as late complication [10]. The diabetic patients have high risk of muscle atrophy and change in muscle is clearly associated with type1 diabetes mellitus [13]. There was positive insignificant correlation between duration of DM and the activity of total CK level in type1 (R-value= 0.164, P-value= 0.386) and type2 (R-value= 158, P-value= 0.403). Akram *et al.*, [12] reported that there was weak positive correlation between the activity of total CK and duration of diabetes in long standing diabetic with type2 diabetes.

## CONCLUSION

The activity of total CK among diabetic patient with type1 and type2 diabetes mellitus was higher when compare to control group, there was also significant correlation between glucose and activity of total CK in type1 diabetes mellitus.

## REFERENCES

1. Burtis, C. A., & Bruns, D. E. (2015). *Tietz fundamentals of clinical chemistry and molecular diagnostics-e-book*. Elsevier Health Sciences, 7, 618-644.
2. Crook, M. A. (2013). *Clinical biochemistry and metabolic medicine*. CRC Press, 8, 184.
3. Simon, W., Geoffrey, B., Peter, R., Peter, A. (2013). Whitby's clinical biochemistry lecture note, 9, 79-80.
4. Bishop, M. L., Fody, E. P., & Schoeff, L. E. (Eds.). (2013). *Clinical chemistry: principles, techniques, and correlations*. Lippincott Williams & Wilkins, 8, 298..
5. Murray, R. K., Granner, D. K., Mayes, P. A. (2003) Harper's illustrated biochemistry, 26, 160-162.
6. Ammari, F. (2004). Long-term complications of type 1 diabetes mellitus in the western area of Saudi Arabia. *Diabetologia Croatica*, 33(2), 59-63.
7. Rahimi-Madiseh, M., Malekpour-Tehrani, A., Bahmani, M., & Rafieian-Kopaei, M. (2016). The research and development on the antioxidants in prevention of diabetic complications. *Asian Pacific journal of tropical medicine*, 9(9), 825-831.
8. Hughes, J., & Jefferson, J. A. (2008). *Clinical Chemistry Made Easy E-Book*. Elsevier Health Sciences.
9. Marshall, W. J., Bangert, S. K., & Lapsley, M. (2012). *Clinical chemistry*, 7, 284-287.
10. Jevrić-Čaušević, A., Malenica, M., & Dujić, T. (2006). Creatine kinase activity in patients with diabetes mellitus type I and type II. *Bosnian journal of basic medical sciences*, 6(3), 5-9.
11. Awadalla, A. H., Elabid, B. E., & Alzubeir, M. (2010). Serum Creatine Kinase Level In Sudanese Patients With Long Standing Diabetes Mellitus Type 2 In Khartoum State, Central Sudan. *Gezira Journal of Health Sciences*, 6(1).
12. Kelley, D. E., He, J., Menshikova, E. V., & Ritov, V. B. (2002). Dysfunction of mitochondria in human skeletal muscle in type 2 diabetes. *Diabetes*, 51(10), 2944-2950.
13. Santos, A. J. C. A., Silva, E. L. A., Albuquerque, Y. M. L., Oliveira, B. D. R., & Caiaffo, V. (2016). Effects of diabetes mellitus type I on skeletal muscle: an integrative review. *Journal of Morphological Sciences*, 33(02), 118-120.