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SERUM CREATINE KINASE LEVEL IN SUDANESE PATIENTS WITH LONG STANDING DIABETES MELLITUS TYPE 2 IN KHARTOUM STATE, CENTRAL SUDAN

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ABSTRACT

Objectives: To evaluate and correlate the serum activity of creatine kinase in Sudanese patients with type 2 long standing diabetes mellitus.

Patients, Materials and Methods: This is a prospective, descriptive, cross- sectional, hospital based, case-control study was done in fifty patients (Twenty male &thirty female) above forty years with long period diabetes mellitus type 2 referred to Jabber Abu Eliz Diabetes Center and Bahri Diabetes Center in Khartoum state in central Sudan during May – October 2007. Thirty healthy volunteers with matching age and sex and socioeconomic status were included. Data was collected through, clinical evaluation form, questionnaire, and laboratory investigations. Serum concentrations of creatinine kinase were measured by spectrophotometery and t. test and correlation were used in statistical analysis for the comparison between test and control group.

Results: The result showed a significant difference between the mean of serum creatine kinase of the control group compared with that of the case group (Mean±SD): (58.02±12.3); (90.46±47.6) U/L respectively, ($P<0.05$). Also there was a weak positive correlation (r) = 0.40, P =0.01 between the serum levels of

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creatine kinase and the duration of diabetes mellitus

Conclusion: From this study it was concluded that; the increase of the serum levels of creatine kinase may be due to the damage of cardiac or skeletal muscles, so this study recommends the measurment of creatine kinase isoenzymes for differentiation.

Key words: Creatine kinase, Diabetes Mellitus

الملخص

مستوى إنزيم الكرياتين ناقل الفوسفات في مصل الدم عند السودانيين المصابين بمرض السكري طويل الأمد من النمط الثاني بولاية الخرطوم وسط السودان

هدفت هذه الدراسة لقياس مستوي انزيم الكرياتين ناقل الفوسفات فى مصل الدم للمرضى المصابين بمرض السكرى طويل الامد من النوع الثانى وبحث علاقة الارتباط بين انزيم الكرياتين ناقل الفوسفات وفترة مرض السكري.أجريت هذه الدراسة المتوقعة خلال الفترة من شهر مايو الى اكتوبر 2007 على 50 شخص كمجموعة مرضى و 30 شخص أصحاء كمجموعة ضابطة وتم اختيارهم عشوائيا من مركزي جابر ابو العز و بحري للسكرى بالخرطوم.أوضحت النتائج أن هنالك إرتفاع ذو دلالة إحصائية ($P<0.05$) فى متوسط مستويات إنزيم الكرياتين ناقل الفوسفات لدى مرضى السكرى مقارنة مع المجموعة الضابطة (المتوسط ±الانحراف المعياري): (90.46±47.6) ; (58.02±12.3) وحدة\ اللتر علي التوالي وكذلك وجدت علاقة إرتباط موجبة لكن بصورة ضعيفة بين مستوى إنزيم الكرياتين ناقل الفوسفات مع فترة مرض السكرى (r). 0.40 =. من هذه الدراسة نستخلص أن الزيادة فى إنزيم الكرياتين ناقل الفوسفات ربما يعزى الى تلف فى عضلات القلب اوالعضلات الهيكلية.لذلك فإن دراسة مماثلات إنزيم الكرياتين ناقل الفوسفات ضرورية للتمييز.

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INTRODUCTION

Diabetes mellitus is a group of metabolic diseases characterized by high blood glucose levels (Hyperglyceamia), which result from defects in insulin secretion, or action, or both, identified as a disease associated with “sweet urine," and excessive muscle loss in the ancient world. Normally, blood glucose levels are tightly controlled by insulin by lowering blood glucose level, insulin a hormone produced by the pancreas. [1]

Globally, the statistics are staggering. Diabetes is the third leading cause of death in the United States after heart disease and cancer. [2]

Approximately two thirds of people with diabetes die from heart disease or stroke.

Men with diabetes face a 2-fold increased risk for coronary heart disease, and women have a 3- to 4-fold increased risk. In 1994, one of every seven health care dollars in the United States was spent on patients with diabetes mellitus. The 2002 estimate for direct medical costs due to diabetes in the United States was $92 billion, with another $40 billion in indirect costs. Approximately 20% of Medicare funds are spent on these patients.[3]

Since the tight control of enzyme activity is essential for homeostasis, any malfunction (mutation, overproduction, underproduction or deletion) of a single critical enzyme can lead to a genetic disease . The importance of enzymes is shown by the fact that a lethal illness can be caused by the malfunction of just one enzyme out of the thousands of enzymes present in our bodies.[4]

Plasma Creatine Kinase activity is raised in all types of muscular dystrophy, but not usually in neurogenic muscle disease such as poliomyelitis.[5]

Patients with diabetes mellitus type 2, disbalance at the level of regulation of glucose metabolism as well as lipid metabolism has been noted in skeletal muscles. It was assumed that in this type of diabetes, these changes were reflected at the level of total activity of enzyme creatine kinase and changes in enzyme activity were more expressed in patients with diabetes type 2. [6]

PATIENTS, MATERIALS & METHODS

This study was carried out in Khartoum state in central Sudan. The subjects were selected in Khartoum city randomly. The data was obtained from different sites in Khartoum city: Khartoum and Khartoum north (Bahri). The study was conducted during the period between May – October 2007.

Fifty patients (Twenty male & Thirty female) above forty years with long standing diabetes mellitus (more than ten years), were selected after taking their consent. Each volunteer in this study was asked to come to Jabber Abu Eliz Diabetes Center in Khartoum 2 and Bahri Diabetes Center for medical assessment and sample collection. Thirty healthy subjects (Twelve males, eighteen females) were selected as a control group who were age, sex and socioeconomic status matched to the diabetic group (test group).

Clinical data was obtained from the patient’s history and recorded on a questionnaire sheet. Clinical assessment of the study group was done by a medical doctor and they were not suffering from myocardial infarction or even have recent injuries which leaded to increase Creatine Kinase level.

After informed consent, venous blood sample (5ml) was collected from the study subjects. After blood clotting, the samples were centrifuged within 20 minutes after collection at 3000 rpm for 5 minutes and the sera were stored -20 °C until analysis.

The serum was allowed to reach the room temperature and creatine kinase

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specific activity was measured spectrophotometrically in the direction of ATP synthesis at 340 nm wavelength using creatine kinase kits from Biosystem company Costa Brava, 30, Barcelona (Spain).

Quality control: Control serum of known Creatine Kinase value was used to verify the performance of the measurement procedure.

Statistical analysis:

The data collected in this study were analyzed by using SPSS computer program package. The mean and standard deviations of creatine kinase were used to compare between the test group and the control group. The P values were obtained using the (t) test. Correlation between the serum levels of creatine kinase and the duration of the disease were tested using Pearson correlation. P.values < 0.05 were considered to be statistically significant P.values < 0.01 were considered highly significant.

RESULTS

Serum Creatine Kinase:-

Table 1 shows a significant difference between the mean of serum Creatine Kinase of the control group compared with that of the test group (Mean ±SD): (58.02±12.3) versus (90.46±47.6) U/L, (P<0.05).

Table 1:

Comparison of the means of serum creatine kinase of the control group and the test group:

Table 1 : Comparison of the means of serum creatine kinase of the control group and the test group

Variable	Control group Non diabetic n=30	Test group Diabetic n=50	P
Serum C.K. (U/L)	58.02±12.3	90.46±47.6	‹0.05

- The table shows the mean ± SD and probability (P).
- t-test was used for comparison.

Figure 1 shows a weak positive correlation between the serum levels of creatine kinase level and the duration of diabetes mellitus. Correlation coefficient (r) = 0.40, P =0.01 Correlation is highly significant at (P<0.01)

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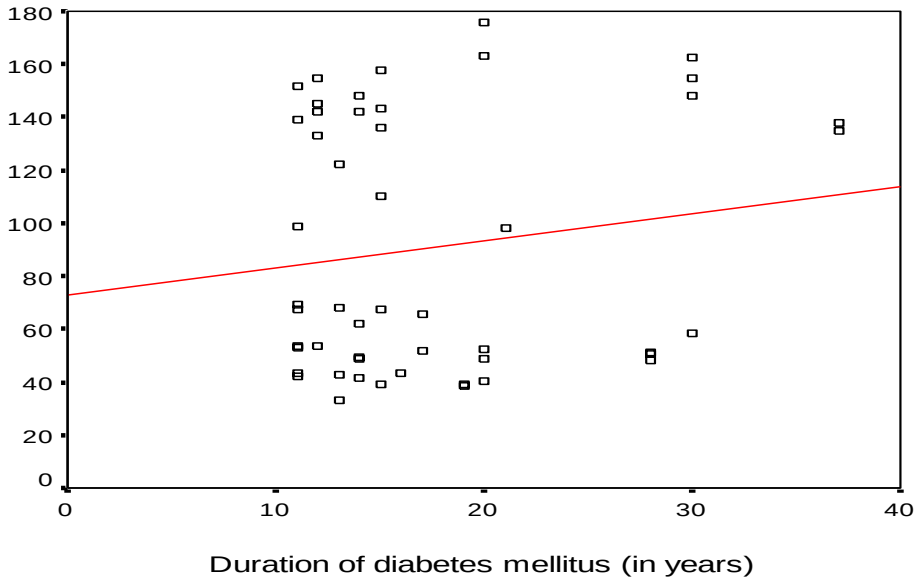

Figure 1: A scatter plot shows correlation between the serum levels of CK and the duration of diabetes mellitus (r=0.40, p =0.01).

DISCUSSION

This study showed a significant increase in the mean serum levels of creatine kinase of the test group compared with control group (P<0.05) as shown in (Table 1). This may be due to the effect of long standing type 2 diabetes mellitus on cardiac and skeletal muscles as a complication of atherosclerosis. CK isoenzymes study to determine the source of the elevation of CK may be needed. In this study there was a weak positive correlation (r =0.40) between the serum levels of CK and the duration of diabetes mellitus. (Fig.1)

The above results agree with a study done by Jevri, etal(2006), who found that in patients with diabetes mellitus type2, disbalance at the level of regulation of glucose metabolism as well as lipid metabolism has been noted in skeletal muscles. It was assumed that in this type of diabetes, these changes were reflected at the level of total activity of enzyme creatine kinase and changes in enzyme activity were more expressed in patients with diabetes type 2. In the same study positive correlation between concentration of glucose and serum activity of the enzyme was seen in both categories of diabetic patients which was

not the case for the control group.

Another study observed a moderate correlation between the degree of glycemia and CK elevation but with improvement of the carbohydrate metabolism the CK level became normal.[7]

The above observations disagree with a study done by Margiavic,etal(1986) who observed that the concentration of CK activity in the diabetic patients did not differ from the control level.[8]

CONCLUSION

This study concluded that the serum activity of CK is elevated with a weak positive correlation

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with the duration of diabetes mellitus in patients with long standing diabetes mellitus type 2.

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