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A Comparative study of Magnesium, C-reactive protein and lipids profile in Diabetic with Complication

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Abstract

The present study was undertaken in 90 subjects, 30 non diabetic as normal control (groupI), 30 diabetics without complication (group II) and 30 diabetics with complication (group III). Blood sugar levels, Magnesium, C-reactive protein (SRP), Glycosylated hemoglobin (HbA_{1c}), Cholesterol, Triglyceride, HDL and LDL were analyzed from serum of patients and controls group. Serum magnesium levels in group III were found to be significantly lowered (1.24±0.28meq/l) than in group I (2.56±0.58). There was also significant difference in magnesium levels of group II and group I (2.56±0.58, 1.98±0.32 meq/l respectively).

The results also indicate that the diabetic patients showed significant rise in serum C-reactive protein ($p<0.05$), glycosylated hemoglobin ($p<0.001$), serum cholesterol, triglyceride ($p < 0.01$), HDL-cholesterol and LDL-cholesterol ($p < 0.001$) as compared to control. The C-reactive protein values in group I, group II and group III were measured as 2.46±3.66, 8.4±4.87and 10.6±6.44, respectively. These values were found to be significantly higher in group II and group III ($p<0.05$). The HbA_{1c} (%) were measured as 4.89±0.68, 8.66±0.77and 10.88±1.23 respectively. These values were also found to be significantly higher in group II and group III ($p<0.001$) .The aim of this study was to evaluate the serum of magnesium, C-reactive protein, Glycated hemoglobin (HbA_{1c}) and lipid profile status.

Introduction

Genetic and environmental factors contribute to the pathogenesis of diabetes and acts as trigger for the disease among subjects at high risk because of inherited susceptibility (Groop and Tuomi, 1997). The composition of our diet has changed considerably during the past few decades, and these are thought to be contributing greatly to the increasing incidence of diabetes mellitus (Vessby, 2000).Direct associations of trace and macro elements with diabetes mellitus have been observed in many research studies (Nouramomammadi *et al.*, 2000). Insulin action was reported to be potentiated by some trace elements as magnesium, zinc, and selenium (Candilish, 2000). Proposed mechanism of trace elements enhancing insulin action includes activation of insulin receptor sites (Vincent, 2000), serving as cofactors or components for enzyme systems involved in glucose metabolism (Murray *et al.*, 2000), increasing insulin sensitivity and acting as antioxidants preventing tissue per-oxidation (Kruse- Jarres and Rukguaer, 2000). Magnesium is a cofactor in the glucose transporting mechanisms of the cell membrane and various enzymes in carbohydrate oxidation, it is also involved at multiple levels in insulin secretion, binding and enhances the ability of insulin to activate tyrosine kinase (Suarez, 1993).

Hypomagnesaemia has long been known to be associated with diabetes mellitus; Mather *et al*., 1979 confirmed the presence of hypomagnesemia in nearly 25% of their diabetic out patient. Several studies have suggested that hypomagnesaemia may be implicated in the aetiology for diabetic complications (Aradhana *et. al*., 2007).

The association between diabetes mellitus and hypomagnesemia is compelling for its wide ranging impact on diabetic control and complications (Wada, *et al*., 1983).As the magnesium is an effector of inositol transport, it increases the affinity of the transporter for inositol. Hypomagnesaemia causes a decrease in the affinity of the inositol-transport protein for inositol. This allows hypomagnesaemia and the polyol thereby to be rationalised in to a single mechanism for the aetiology of diabetic complications (Aradhana *et. al*., 2007).

Recent experimental findings suggest that magnesium deficiency in rats may be involved in exacerbation of immune stress response, which increases the levels of acute-phase reactants (Kao ***et. al***., 1999). C-reactive protein (CRP) is one of the most sensitive acute-phase reactants in humans (Ridker *et. al.*, 2001), its elevated serum levels are a precise index of inflammatory activity, and have been related with coronary heart disease (Yudkin *et. al.*, 2000) supporting the hypothesis that inflammation plays a role in the development of metabolic syndrome and diabetes. High CRP levels have been linked to an increased risk of thrombotic events including myocardial infarction (Kervinen *et. al.*, 2001).CRP levels are higher in people with diabetes compared with those without diabetes (Goldberg, 2000).Furthermore, elevated CRP levels have also been linked to an increased risk of later development of diabetes (Pradhan *et. al.*, 2001). Also recent research evidence supports a link

between hyperglycemia and inflammation. CRP is known to be higher in people with impaired glucose tolerance and frank diabetes (Wu *et. al.*, 2002).

Glycosylated Hemoglobin (HbA_{1c}) results from post translational changes in the haemoglobin molecule, and their levels correlate well with glycemic levels over the previous six to ten weeks. Glycosylation of haemoglobin takes place under physiological condition by a reaction between glucose and Terminal valine of Beta chain of Hb molecules (Ishrat *et. al*., 2004).Glycosylated hemoglobin has been postulated as a biochemical model for the pathogenesis of diabetic sequelae through the glycosylation reactions (Peterson. and Jones., 1977). In both type 1 and type 2 diabetes, large prospective clinical studies have shown a strong relationship between time-averaged mean levels of glycemia, measured as hemoglobin A_{1C} (HbA_{1c}), and diabetic complications.(Michael Brownlee *et. al*.,2006)

Materials and methods

This study was conducted in 60 diagnosed cases of diabetes mellitus (type1and type2) and 30 healthy subjects of same age and sex who were taken as control. The subjects were in the age group of 40 to 65 years. Diabetes in this study was defined based on laboratory findings as fasting plasma glucose levels greater than 126 mg/l (WHO, 1999). Patients were divided into three groups healthy control subjects (group I), diabetic patients without complications (group II) and diabetic patients with complications (group III).A thorough interrogation, clinical examination and relevant laboratory investigations (which include fasting blood sugar, HbA_{1c}, lipid profile, blood urea, serum creatinine and ECG) were carried out in all the patients.Malabsorption syndrome, liver disease, renal failure ,Pregnancy, alcoholism, acute infectious diseases and drugs like diuretics) were excluded from the study. 10 ml of blood was drawn by venipuncture and collected in a vial with EDTA in the morning after an overnight fast. Serum was separated and the samples stored at 4 ºC to 10 ºC till being processed.

Fasting blood sugar was estimated by Barham and Trinder, method (1972). Cholesterol byCHOD-PAP method (Tietz, 1999), HDL by CHOD-PAP method (Assmann, 1979) and triglyceride by Tietz's TG-PAP method (1995).Glycosylated Hemoglobin (HbA_{1c}) estimation was carried out by a modified calorimetric method of (Fluckiger and Winterhalter, 1976).C-reactive protein measurement was done by (Ward and Cooper, 1975) method, while serum magnesium was estimated using the flame atomic absorption spectrophotometry.

Statistical analysis

Data are expressed as mean±□SD. Statistical significance was evaluated by Student's *t* test. Differences were considered significant at ($p<0.05$) (الراوي، 1984).

Results

The average concentration of magnesium in groups I (Control), group II (diabetic without complication), and group III (diabetic with complication) were measured as 2.56±0.58, 1.98±0.32and 1.24±0.28meq/l respectively. The patients in group III has hypomagnesaemia ($p < 0.01$, table1) when compared to group II. Also group II showed hypomagnesaemia ($p < 0.01$) when compared to group I. Groups I, group II, and group III were also compared with respect to C-reactive protein, glycosylated hemoglobin, blood glucose, triglyceride, cholesterol , HDL-cholesterol and LDL-cholesterol levels.

The C-reactive protein values in group I, group II and group III were measured as 2.46±3.66, 8.4±4.87and 10.6±6.44, respectively .These values were found to be significantly higher in group II and group III ($p<0.05$, table1).

The HbA1c (%) were measured as 4.89±0.68, 8.66±0.77and 10.88±1.23 respectively. These values were found to be significantly higher (group II and group III $p<0.001$, table1) and correlated positively with blood glucose levels.

The serum cholesterol levels were estimated as 160.2±12.39, 222.3±14.53, and 266.4±12.44 mg/dl for group I, group II and group III respectively. The serum triglycerides levels were 115.82±8.21, 156.6±12.9 and 188.4±6.77for group I group II and group III respectively.The serum HDL-cholesterol levels were estimated as 36.66±3.04, 39.1±6.4, and46.6±4.35, mg/dl for group I, group II and group III, respectively. The serum LDL-cholesterol levels were 104.5±14.7, 134.5±21.8, and 148.8±14.3mg/dl for group I, group II and group III respectively.The serum cholesterol, triglyceride, HDL-cholesterol and LDL-cholesterol values were significantly higher in group II and group III ($p < 0.01$, $p < 0.001$, table2) as compared to groupI.

A significant negative correlation ($p<0.05$, table3) was observed between serum magnesium and C-reactive protein ($r = -0.473$), magnesium and fasting blood sugar ($r = -0.56$), magnesium and glycosylated hemoglobin ($r = -0.56$) and non significant correlation between serum magnesium and triglycerides ($r = -0.13$), serum magnesium and cholesterol($r=-0.23$, $p>0.05$ table3) of diabetics patients.

Discussion

There are several studies on the role of trace elements in diabetes and its complications (Aradhana *et. al*., 2007).Magnesium activates more than 300 enzymes in body and is a critical cofactor of many enzymes in carbohydrate metabolism. Cellular magnesium deficiency can alter the activity of membrane bound sodium-potassium ATPase (Grofton and Borte, 1992) which is involved in maintenance of gradients of sodium, potassium and in glucose transport. Low levels of magnesium can reduce secretion of insulin by the pancreas. In diabetes there is a direct relationship between serum magnesium level and cellular glucose disposal that is independent of insulin secretion. This change in glucose disposal has been shown to be related to increased sensitivity of the tissues to insulin in presence of adequate magnesium levels (Yajnikcs *et. al*., 1984).

The actual role of this element in the pathogenesis and progress of diabetes is still unclear (Tuvemo and Gebremedhim, 1983). The observed alteration in the status of this element seen in diabetics has been attributed to hyperglycemia and increased protein glycosylation seen in this condition (Srivastava *et al.*, 1993).

Our observations revealed a definite lowering of serum magnesium in diabetic patients without complications and more over patients who had complication were found to have lowest concentration of serum magnesium (1.98±0.32and 1.24±0.28meq/l respectively ,table 1). These results are similar to other workers (Aradhana *et. al*., 2007 and Ishrat *et. al*., 2004). All had strongly reported association of hypomagnesaemia with diabetes mellitus. The exact cause of diabetic hypomagnesaemia is still unknown but an increased urinary loss of magnesium may contribute to it. Two factors may work together in this respect namely, the osmotic action of glucosuria and the hyperglycemia per se, the latter being known to depress the net tubular reabsorption of magnesium in normal man (Garland, 1992).

Inflammatory markers such as CRP have been related to the development of insulin resistance and type 2 diabetes (Festa *et. al*., 2007). In this study it was observed that the mean serum CRP level was statistically significantly high (8.4±4.87and 10.6±6.44, $p<0.05$, table 1) in diabetic patients in comparison to control subjects (2.46±3.66, table 1). These results were in accordance with the observation of (Goldberg, 2000, Yudkin *et. al.*, 2000, Wu *et. al.*, 2002 and Pradhan *et. al.*, 2001).in this regard, several studies have related hyperglycemia to inflammation by demonstrating simultaneous inflammation, endothelial dysfunction, and insulin resistance at the physiologic level (Guerrero-Romero & Rodríguez-Morán**,** 2000). One of the several mechanisms proposed is oxidative stress on the endothelium, which promotes inflammation and is enhanced by hyperglycemia (Dandona and Aljada, 2001).

In the present study significant higher levels of HbA1c was seen in diabetic patients compared to healthy control (4.89±0.68, 8.66±0.77and 10.88±1.23 respectively). The mean HbA1c levels in diabetic with complications were higher than in diabetes without complication and it was statistically significant (table1, $p < 0.001$).This is

accordance with various other studies (Koeing *et al.* ,1976, Gonen, *et al.* ,1977 and Aradhana *et. al*., 2007). HbA1c has special affinity for oxygen thereby causes tissue anoxia and plays a role in causation of micro and macroangiopathy (Ronal *et al.*, 1975).Thus, within the same sphere recent studies mentioned that the measurement of glycosylated hemoglobin not only shows promise of being a successful approach to the monitoring of diabetic patient but also provides a conceptual frame work for the pathogenesis of secondary sequelae of diabetes (Ishrat *et. al*., 2004).In our study dyslipidemia ($p < 0.01$, $p < 0.001$,table 2) is seen in diabetes with and without complication.

Inverse correlation was found between serum magnesium and C - reactive protein levels ($r=-0.47, p<0.05$ table 3).In this regard, recently, Malpuech-Brugere *et. al*., 2000 reported that magnesium deficient rats showed a significant increase in the acute-phase reactants and reduced serum albumin, and concluded that inflammatory response is an early consequence of magnesium deficiency, suggesting that reduced extracellular magnesium might be responsible for the activated state of immune cells. Thus, it is significant that both increased acute-phase response and decreased serum magnesium levels are related to aging, a well-known risk factor for glucose metabolic disorders and cardiovascular disease (Pickup *et. al*., 1997).

Present study also observed a strong inverse relation between HbA1c and serum magnesium ($r=-0.56$, $p<0.05$ table3) Ishrat *et. al*., 2004 observed that hypomagnesaemia was an important indicator of poor diabetic control. In their study they observed close correlation of HbA1c level well with concentration of magnesium. There was no statistically significant correlation between serum magnesium and serum triglyceride and cholesterol ($p>0.05$, table 3). Our results in this regard are similar to the findings of other workers (Jiang *et. al*., 1995).

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Table (1) Showing meam±S.D of Serum magnesium, Glycated hemoglobin, C-reactive protein and Fasting Glucose in different Groups.

	(Group I) control	**(Group II) DM without complication**	**(Group III) DM with complication**	P_{value}
Number	30	30	30	-
Magnesium(meq\l)	2.56±0.58	1.98±0.32	1.24±0.28	<0.01

Glycated hemoglobin (%)	4.89±0.68	8.66±0.77	10.88±1.23	<0.001
C-reactive protein(mg\dl))	2.46±3.66	8.4±4.87	10.6±6.44	<0.05
Fasting Glucose (mg %)	94.28±6.55	200±56.44	260±26.01	<0.001

Table (2) Showing meam±S.D of Lipid profile in different Groups.

	Group I control	**Group II DM without complication**	**Group III DM with complication**	P_{value}
Number	30	30	30	-
Cholesterol (mg %)	160.2±12.39	222.3±14.53	266.4±12.44	<0.01
Triglycerides (mg %)	115.82±8.21	156.6±12.9	188.4±6.77	<0.01
HDL- cholesterol (mg %)	36.66±3.04	39.1±6.4	46.6±4.35	<0.001
LDL- cholesterol (mg %))	104.5±14.7	134.5±21.8	148.8±14.3	<0.001

Table (3) Correlation Analysis of Magnesium levels in diabetic patients

Parameter	**Mg**(r)	P_{value}
C-reactive protein	-0.47	<0.05
Fasting blood sugar	-0.56	<0.05
Glycated hemoglobin	-0.56	<0.05
Triglycerides(TG)	-0.13	>0.05
Cholesterol	-0.23	>0.05

دراسة مقارنة لمستويات المغنيسيومِ، بروتينC-الفعال ومستويات الدهون في مضاعفات مرضى السكري

مجلة القادسية للعلوم الصرفة المجلد 13 العدد 1 لسنة 2008
المؤتمر العلمي الاول لكلية العلوم المنعقد في 26-27 اذار لسنة 2008

عباس جواد كاظم

جامعة القادسية/ كلية العلوم/ قسم الكيمياء

أجريت الدراسة على 90 فرد،30 غير مصابين بالسكري اعتبروا كمجموعه سيطرة (groupI) ،30 مصابين بالسكري ولكن بدون مضاعفات (group II) و30 فرد مصابين بالسكري المصاحب بمضاعفات(III group).حللت مستويات المغنسيوم ،بروتينC - الفعال،الهيموكلوبين السكري ومستويات الدهن في مصل كل من المرضى والأصحاء . أظهرت النتائج أن هناك انخفاض معنوي لمستوى المغنسيوم(1.24±0.28meq/l) في group III بالمقارنة (2.56±0.58) مع group I وكذلك أظهرت النتائج بان هنالك فروق معنوية بين group II و groupI (p<0.01،1.98±0.32 meq/l ,2.56±0.58).
كما أظهرت نتائج الدراسة الحالية أن هنالك ارتفاع معنوي في مستويات كل من بروتينC- الفعال(p<0.05) ، الهيموكلوبين السكري(p<0.001) ومستويات الدهون (p < 0.001, p < 0.01) في مرضى السكري وذلك بالمقارنة مع مجموعه الأصحاء. حيث كانت قيم بروتينC- الفعال في المجاميع الثلاثة على التوالي (2.46±3.66, 8.4±4.87and 10.6±6). و قيم الهيموكلوبين السكري فكانت (4.89±0.68 8.66±0.77and 10.88±1.23)وبذلك فان النتائج أظهرت زيادة معنوية في كل من بروتينC- الفعال و الهيموكلوبين السكري في المرضى المصابين بداء السكري بالمقارنة مع الأصحاء.الهدف من الدراسة تقيم مستويات المغنسيوم والبروتينC- الفعال ،والهيموكلوبين السكري ومستوى الدهون في مرضى السكري المصحوب بمضاعفات.