SERUM MAGNESIUM LEVELS AMONG TYPE 2 DIABETIC PATIENTS AND PREDIABETIC SUBJECTS IN DUHOK CITY/ IRAQ

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ABSTRACT

Background: Type 2 diabetes mellitus and prediabetes are reported to be associated with abnormalities in the metabolism of magnesium. Magnesium deficiency by itself may be a contributing factor to the progression of Type 2 diabetes mellitus and its complications.

Methods: a cross-sectional study was carried out at the Diabetic unit in Azadi teaching hospital. A total of 248 subjects were included in the study constituting 99 patients with diabetes mellitus, 50 subjects with Prediabetes Mellitus and 99 as a control group that were apparently healthy subjects.

Results: The present study has found a significant decrease in serum magnesium levels in Type 2 diabetic patients and prediabetic subjects as compared to controls (1.88±0.23, 1.93±0.20, 2.00±0.17 mg/dl respectively, P=0.003). The rate of hypomagnesaemia was high among diabetes patients and prediabetes subjects (23.2%, 10.0%, and 3.0%, respectively). The highest frequency of hypomagnesaemia was found in those diabetes patients with poor glycemic control, old age, increased fasting serum glucose, and who are also overweight and obese.

Conclusion: Type 2 diabetes mellitus patients and Prediabetes subjects have a higher rate of hypomagnesaemia and lower mean Mg level compared with healthy control subjects.

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Keywords: Hypomagnesemia, Prediabetes, Type 2, Diabetes Mellitus.

Type 2 diabetes mellitus (T2DM) is a metabolic disease characterized by an increase blood glucose level, caused by either, insulin resistance or insulin deficiency and it represents 90% of diabetic cases[1],[2]. The rising rate of T2DM is a public health problem as it is a major cause of coronary heart diseases, preventable visual loss, end-stage renal disease, and non-traumatic amputations[3]. People with T2DM usually begin with insulin resistance, a condition, in which muscle, liver, and fat cells do not use insulin properly. In time, however, the beta cells fail and the pancreas loses the ability to secrete enough insulin in response to glucose load and glucose persistently builds up in the blood and the body cannot make efficient use of its main source of fuel[4].

Prediabetes on the other hand is an impaired glucose tolerance (IGT), and impaired fasting glucose (IFG)[5]. Prediabetes is regarded as a very high risk

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factor for D.M development. The individuals with prediabetes are characterized by increased blood glucose level, which is higher than the normal range and lower than that of D.M range. Since not all individuals with prediabetes progress to T2DM, the use of this term in its strictest sense has been questioned, and some prefer to use the term intermediate hyperglycemia[6,7].

Magnesium (Mg), as the second most major intracellular cation in the human body, is the basic composition of many enzymes and play a very essential role in energy metabolism, protein and nucleic acid synthesis, and cell proliferation[8,9]. Mg may has an important role in transporting glucose through the cell membranes, and an essential role in enzyme activity involved in carbohydrate oxidation and improves insulin response and action, by increasing affinity of insulin to its receptors[10].

It was worth mentioning also, that T2DM is frequently associated with both extracellular and intracellular Mg deficits. It has been documented that hypomagnesaemia affects blood sugar level; and may be a cause or a consequence of diabetic complications due to its negative effect on glucose homeostasis and insulin resistance[11,12,13]. As a result of aforementioned, With respect of prediabetes, Mg regulates glucose metabolism at cellular level and acts as a second messenger for insulin. It has been noticed that insulin enhances uptake of Mg from extracellular compartment to intracellular compartment, and this mediates diverse effects ascribed to insulin. Hypomagnesaemia may induce altered cellular glucose transport, reduced secretion of insulin from pancreas, impaired post-receptor insulin signaling and altered interaction of insulin with insulin receptor and thus aggravates insulin resistance and may lead to prediabetes[14]. The association between Mg levels and prediabetes might be due to reverse causation. Since, in subjects with prediabetes, serum glucose levels are lower than the threshold for urinary Mg losing, it is unlikely to influence serum Mg levels. Low serum Mg levels may associate with an increased risk of prediabetes and this increased risk is similar to that of diabetes[15].

The aim of this study is to estimate the serum magnesium levels in Type 2 diabetic patients and prediabetic subjects in comparison with those of apparently healthy individuals in Duhok city, Kurdistan Region of Iraq. Furthermore, evaluation of the relation between blood HbA1c, blood glucose, age, body mass index, central obesity, family history of diabetes mellitus and gender in prediabetic subjects and T2DM patients.

MATERIALS AND METHODS

Subjects and study design:

This cross sectional study was carried out in the Endocrine and Diabetes unit at Azadi Teaching Hospital, and the Department of Medical Chemistry, College of Medicine / University of Duhok - Kurdistan Region, Iraq. A total number of 99 of diabetic outpatients visiting the endocrine and diabetic unit were selected. Based on the diabetes record for each diabetic patient, the following conditions were excluded: pregnant women, patients with history of liver, renal and heart

diseases, patients taking Mg, and the patients with Type 1 diabetes mellitus. Additionally, 50 subjects with prediabetes, chosen from relatives of diabetic patients, and 99 healthy individuals (control group) were recruited by personal request from the staff of Azadi Teaching Hospital and Emergency Teaching Hospital. The study group ages were between 20 and 70 years. Informed consent was obtained from each participant commenced before the study and the recruited one were approved by the Ethics Committee of the Directorate of Health of Duhok city Governate. The questionnaire form was similar to the diabetic clinic questions at the Endocrine and Diabetic Unit with some modifications. The questionnaire form includes questions such as: name, age, sex, physical activity, duration of D.M, type of drugs taken for D.M (only for patients), family history of diabetes and history of mineral taking (Mg+2) with measurement waist circumference and calculation of body mass index.

Collection and processing of blood sample:

Ninety nine blood samples were collected from T2DM patients diagnosed according to the WHO protocol. Ninety-nine blood samples were also collected from apparently healthy individuals (control group), and another fifty blood samples were collected, from prediabetic subjects. Participants who attended at the endocrine and diabetic unit in the morning were fasting overnight for 12 to 14 hours. Venous blood samples (6ml) were collected between 8:30-11:30 AM, and withdrawn by venipuncture using vacutainer from the antecubital vein. Two ml were collected immediately into a vacuum tube containing EDTA as anticoagulant for estimation of HbA1c by DCA Vantage Analyzer (Siemens); the remainder 4 ml was collected in vacutainer system – gel separator tubes. The serum was separated from the whole blood after clotting using centrifugation process (HITACHI centrifuge, model O5P-21) at 5000 rpm for 10 min, and then the serum was processed immediately for measuring glucose and magnesium.

Measurements:

Serum glucose and magnesium concentrations were detected using the commercial available kits (Biolabo SA, France); for determination of HbA1c, the quantitative method in whole blood was done using DCA Vantage Analyzer (Siemens). The analyses were carried out according to the manufacturer's instructions. Waist circumference was measured at minimal respiration at the high point of iliac crest to the nearest 0.1 cm, and central obesity was estimated. Body Mass Index is defined as the body weight divided by the square of the body height, and is universally expressed in units of kg/m2, resulting from mass in kilograms and height in meters.

Statistical analysis:

Statistical analysis of data was done by using the computer program under Statistical Package for the Social Science (SPSS) Version (23.0). For the comparison of values between the group; t-test, one-way ANOVA, Kruskal-Wallis, and Chi-Square was used, represented by 'p-value, Statistical $p<0.05$ was considered significant.

RESULTS

With stratification of patient, and subject characteristics based on the study groups, the result in Table 1, revealed that the majority of the patients and subjects were females; (60, 60.4%) in the control group and (32, 64%) prediabetic subjects, and (61, 61.6%) diabetic patients with no statistically significant difference between the subjects genders (p=0.922). In addition, the study revealed that the majority of the controls (86.9%), prediabetic (79.6%) and diabetic patients (85.9%) were non-exercisers (p=0.836). With respect to the family history, it was shown that most of the patients had familial history from their mothers (58.0% in prediabetic and 46.5% in diabetic patients), but from both of the parents (5.05%) among the controls followed by 12.0% and 15.2% from both parents in the prediabetic and diabetic patients, respectively. The similar mean±SD of age was found in the controls (37.48±9.85 years) and prediabetic subjects (37.00±9.18), in contrast with 52.13±7.52 in diabetic patients with a statistically substantial difference (p=0.0001). The results in Table 1 showed also that the mean±SD of glucose (mg/dl), HbA1c (%), waist circumference (cm), and BMI in diabetic patients were greater than in the control and prediabetic subjects, (200.00±6.66 mg/dl for glucose (p=0.0001), 7.42±1.49 % for HbA1c (p=0.0001), 104.05±10.85 cm for waist circumference (p=0.0001), and 32.48±7.38 kg/m2 for BMI (p=0.0001). On the other hand, the study showed the mean±SD of Mg in diabetic patients and prediabetic subjects to be lower than in control subjects [(1.88±0.23, 1.93±0.20) mg/dl, and 2.00±0.17, p=0.003)]. The rate of hypomagnesemia in diabetic patients, prediabetic subjects, and control subjects was found to be (23, 23.2%; 5, 10.0%; and 3, 3.0%, respectively) with statistically significant (p=0.0001).

Table 1. Patients and subjects' characteristics according to control, prediabetic, and diabetic patients:

Subject's Characteristics (n=248)	Frequency Distribution			p-value (two side)	Test of Difference
	Controls (n=99)	Prediabetic (n=50)	Diabetic (n=99)		
Age (years)	37.48±9.85	37.00±9.18	52.13±7.52	0.0001	One way ANOVA
Gender					
Male	39(39.4%)	18 (36%)	38 (38.4%)	0.922	Chi-Square
Female	60(60.4%)	32 (64%)	61 (61.6%)		
W.C. (cm)	97.37±9.71	102.1±12.48	104.0±10.85	0.0001	One way ANOVA
BMI	27.95±5.48	30.90±5.46	32.48±7.38	0.0001	"
Family history of D.M					
Parents	5(5.05%)	6 (12.0%)	15 (15.2%)	0.0001	Fishers' exact test
Mother	33(33.3%)	29 (58.0%)	46 (46.5%)		
Father	12(12.1%)	1 (2.00%)	7 (7.1%)		
None	49(49.0%)	14 (28.0%)	31 (31.3%)		
Exercise					
Yes	13 (13.1%)	10 (20.4%)	14 (14.1%)	0.836	Chi-Square
No	86 (86.9%)	39 (79.6%)	85 (85.9%)		

Subject's Characteristics (n=248)	Frequency Distribution			p-value (two side)	Test of Difference
	Controls (n=99)	Prediabetic (n=50)	Diabetic (n=99)		
Glucose (mg/dl)	88.00±11.0	111.00±6.14	200.00±6.66	0.0001	Kruskal-Wallis
HbA1c (%)	4.62±0.41	5.58±0.91	7.42±1.49	0.0001	One way ANOVA
Mg (mg/dl)	2.00±0.17	1.93±0.20	1.88±0.23	0.003	"
Mg <1.6	3 (3.0%)	5 (10.0%)	23 (23.2%)	0.0001	Chi-squar

The association of Mg level with biochemical categories was also presented in the current study (Table 2), the mean ±SD of Mg among diabetic patient with FSG < 140 mg/dl was significantly higher in comparison with those with ≥ 140 mg/dl (1.97 vs. 1.85, p-0.029) in diabetic patients. In control subjects, serum Mg level was not different significantly with respect to biochemical indicators.

Table 2. Magnesium levels in T2DM patients, prediabetic and healthy subjects in relation with biochemical indicators:

Characteristics	Serum Mg levels in T2DM patients			Serum Mg levels in prediabetic patients			Serum Mg levels in controls		
	N	Mean±S.D	P-value	N	Mean±S.D	P-value	N	Mean±S.D	P-value
FBS									
<140 mg/dl	22	1.97±0.19	0.029	50	1.93±0.20	n.a	99	2.00±0.17	n.a
≥140 mg/dl	77	1.85±0.24		0			0		
HbA1c									
Good (< 6.5%)	32	1.87±0.22	0.726	42	1.96±0.19	0.084	99	2.00±0.17	n.a
Fair (=>6.5%)	67	1.88±0.25		8	1.79±0.23		0		

Further results manifested in Table 3, reveled the differences of serum Mg levels including < 1.6 mg/dl, 1.6-2.6 mg/dl, among subjects' characteristics and categories of biochemical indicators. The rate of hypomagnesemia with some related variables, which shown in Table 3. The diabetic patients that showed higher rate of hypomagnesemia was 100% of age or more 40 years; 21.7% males and 87.3% females; 56.5% with positive family history; 87.0% non-exercise, 100% BMI ≥25, 91.3% FBS ≥140, and 69.6% poor glycemic control ≥ 6.5. The prediabetes subject that show higher rate of hypomagnesemia was, 80% of them were less than 40 years of age and 20% were 40 years or more. Gender 40.0% were males and 60.0% females, 80% had positive family history; 80% did not exercise; 80% had BMI ≥25; 0% FBS ≥140 and 40% showed poor glycemic control ≥6.5.

Table 3. Serum magnesium levels and its association with general and biochemical characteristics in patients, prediabetic, and control subjects:

Characteristics	Serum Mg levels T2DM n (%)			Serum Mg levels Prediabetic n (%)			Serum Mg levels Control n (%)		
	<1.6 mg/dl	1.6-2.6 mg/dl	p-value	<1.6 mg/dl	1.6-2.6 mg/dl	p-value	<1.6 mg/dl	1.6-2.6 mg/dl	p-value
Age									
<40 years old	0(0.0)	3 (3.9)	1.0	4(80.)	26(57.8)	0.63	2 (66.7)	55 (57.3)	1.0
≥40 years old	23(100)	73 (96.1)		1(20.0)	19(42.2)		1 (33.3)	42 (42.4)	
Gender									
Male	5 (21.7)	33 (43.4)	0.06	2 (40.0)	16 (35.6)	1.0	1 (33.3)	38 (39.6)	1.0
Female	18 (78.3)	43 (55.6)		3 (60.0)	29 (64.4)		2 (66.7)	58 (60.4)	
Family history of D.M									
Parents	4 (17.4)	11 (14.5)		1 (20.0)	5 (11.1)	0.68	0 (0.0)	50 (52.1)	0.11
Mother	9(39.1)	37(48.7)	0.26	3 (60.0)	26 (57.8)		0 (0.0)	0 (0.0)	
Father	0 (0.0)	7(9.2)		0 (0.0)	1 (2.2)		0 (0.0)	0 (0.0)	
None	10 (43.5)	21 (27.6)		1 (20.0)	13 (28.9)		3 (100)	46 (47.9)	
Exercise									
Yes	3 (13.0)	11 (14.5)	0.86	1 (20.0)	9 (20.5)	1.0	0 (0.0)	13 (13.5)	1.0
No	20 (87.0)	65 (85.5)		4 (80.0)	35 (79.5)		3 (100)	83 (86.5)	
BMI									
<18.5	0 (0.0)	2 (2.6)		0 (0.0)	1 (2.2)		0 (0.0)	2 (2.1)	
18.5-24.9	0 (0.0)	5 (6.6)	0.42	1(20.0)	2 (4.4)	0.10	0 (0.0)	18 (18.8)	0.12
>25-29.9	10 (43.5)	22 (28.9)		0 (0.0)	19 (42.2)		0 (0.0)	47 (49.0)	
=>30	13 (56.5)	47 (61.8)		4(80.0)	23 (51.1)		3 (100)	29 (30.2)	
FBS									
<140 mg/dl	2 (8.7)	20 (26.3)	0.07	5 (100)	45 (100)	n.a	3 (100)	96 (100)	n.a
≥140 mg/dl	21 (91.3)	56 (73.3)		0 (0.0)	0 (0.0)		0 (0.0)	0 (0.0)	
HbA1C									
Good (< 6.5%)	7 (30.4)	25 (32.9)	0.82	3 (60.0)	39 (86.7)	0.17	3 (100)	96 (100)	n.a
Fair (=>6.5)	16 (69.6)	51 (67.1)		2 (40.0)	6 (13.3)		0 (0.0)	0 (0.0)	
W.C (Male)									
=>102 cm	2 (40.0)	12 (36.4)	1.0	1 (50.0)	5 (31.3)	1.0	1 (100)	6 (15.8)	0.17
<102 cm	3 (60.0)	21 (63.6)		1 (50.0)	11 (68.8)		0 (0.0)	32 (84.2)	
W.C (Female)									
=>88 cm	18 (100)	40 (93.0)	0.54	2 (66.7)	26 (89.7)	0.34	2 (100)	51 (87.9)	1.0
<88 cm	0 (0.0)	3 (7.0)		1 (33.3)	3 (10.3)		0 (0.0)	7 (12.1)	

DISCUSSION:

In the present work, the relation between D.M and Mg deficiency is well investigated. Mg deficiency may be involved in the pathogenesis of D.M itself, because Mg affects sodium potassium ATPase pump, which has a role in maintaining the gradient of glucose across membranes and affects secretion of insulin from the pancreas[16]. Two important findings were obtained from the present cross sectional study, first the rate of hypomagnesemia was high among T2DM patients and prediabetes subjects, when

compared with healthy subjects, with statistically significant differences (23.2%, 10.0%, and 3.0%, respectively, p= 0.0001). Second, the mean serum Mg level was low among T2DM patients and prediabetes subjects in comparison with healthy subjects with statistically significant differences (1.88 ± 0.23, 1.93 ± 0.20 and 2.00 ± 0.17, respectively, p= 0.003). Epidemiological studies had shown that the rate of hypomagnesemia and mean serum Mg level varied markedly throughout various diabetes and prediabetes populations. The previous study introduced by 17 showed that the frequency of hypomagnesemia in diabetes patients was 38.6%, which is higher than that of our finding; whereas, the mean serum Mg level was similar to that of the present study, 1.88 ± 0.28 mg/dl[17]. Other studies showed that the rate of hypomagnesemia is lower than that in our study, which is only 3.8% of their diabetes patients, with a lower serum Mg level of that of our study, 1.78 ± 0.29 mg/dl. While the studies 18, reported that diabetes patients have a higher rate of hypomagnesemia (37.3%), than that of our diabetes patients with very low serum Mg level (0.88 ± 0.22 mg/dl) 18. In any case, the presence of high rate of hypomagnesemia among patients with T2DM is multifactorial. It could be attributed to increased Mg loss in urine by osmotic diuresis of glycosuria, hyperglycemia, insulin resistance and metabolic acidosis, which may be the causes of decreasing expression of Mg channel transient receptor potential melastatin[19], decreased reabsorption of Mg through renal tubules, loss of Mg from extracellular compartment to intracellular compartment due to hyperglycemia[20], impaired intestinal absorption of Mg mostly due to diarrhea that could be induced by diabetic autonomic neuropathy or antihypoglycemic drugs use mainly metformin and in addition to, low intake of diet that are rich in Mg mainly decrease use of whole grains and more use of meat and meat products, the food processing by itself affect the amount of Mg in diet and finally it is important to remember the role of genetic particularly magnesiotropic genes mutation[21].

Regard to prediabetes populations, a study done by 15 shows a low frequency of hypomagnesemia among prediabetes subjects (2.9%) accompanied by low serum Mg level (0.714 ± 0.25), both of which values are lower than of our findings. The factors that may contribute to the high rate of hypomagnesemia in prediabetes subjects include hereditary factors, glomerular hyperfiltration, osmotic diuresis, hypokalemia, hypophosphatemia, autonomic dysfunction and rarely poor intake of diets that are rich in Mg. One of the most important observations in the present work is that the rate of hypomagnesemia was 100% among T2DM patients with age 40 or more years, despite the similar mean ± SD of serum Mg level among both groups. This higher rate may be related to the decreased bone mass with the aging process, increased urinary loss of Mg and gradual decline in intestinal absorption of Mg[22]. Two previous studies performed by 23,24 showed higher frequency of hypomagnesemia among T2DM patients with an age more than 40 years (33.6% and 62.6%, respectively), which are however, lower than the rate of

hypomagnesemia in T2DM patients in our diabetes populations.

Conversely, to T2DM patients, the hypomagnesemia rate among prediabetes subjects was much more common in those subjects of age, less than 40 years (80%), and had higher mean ± SD of serum Mg level when compared with those of more than 40 years. A previous study observed an inconsistent result with our results, which showed higher frequency of hypomagnesemia among those with age equal or more than 40 years (59.7%)[25].

In the present study most of T2DM patients were female, (61.6%). Interestingly, we observed that the frequency of hypomagnesemia in Type 2 diabetes females was higher than Type 2 diabetes males (78.3%, 21.7%, respectively). Although the excretion of Mg in urine is higher in males than females, we found that the mean ± SD of serum Mg level was lower in diabetes females than diabetes males with statistical significance. Controversially, a previous study showed lower rate of hypomagnesemia and higher mean ± SD of serum Mg level in females diabetes patients (44.7%, 1.388 ± 0.588 mg/dl), when compared with males diabetes patients (53.3%, 1.30 ± 0.509 mg/dl) 24. The rate of hypomagnesemia in female prediabetes subjects was higher than in male prediabetes subjects, with lower mean ± SD of serum Mg level. Other study showed a non-significant higher frequency of hypomagnesemia in male's gender (59.7%)[26].

It has been reported that increased BMI is a significant predictor of glucose intolerance 27. In our study, 92% of the diabetes patients were with BMI equal to or more than 25 kg/m² and with lower mean Mg level when compared with those with BMI less than 25 kg/m2. The frequency of hypomagnesemia among diabetes patients in the present study was distributed between obese and overweight subjects (56.5%, 43.5%), which indicates that hypomagnesemia is found in all diabetes patients with BMI equal or more than 25 kg/m². The presence of low Mg level among overweight and obese individuals may relate to the fact that those individual are commonly associated with carbohydrates metabolism disturbance and lipids metabolism disturbance, as well as high blood pressure. Data from previous studies showed different values from that of our values. A study done by 24 showed that 29.0% of overweight and 27.1% of obese diabetes patients had hypomagnesemia. Another study observed that lower Mg level was found in obese and overweight patients (0.85 ± 0.32 mg/dl, 1.167 ± 0.51 mg/dl) 28. Our findings have showed that prediabetes subjects with BMI more than 30 kg/m2 had low mean ± SD serum Mg level, with very high rate of hypomagnesemia. While our finding disagree with the results of 26, which showed higher frequency of hypomagnesemia in those prediabetes subjects with normal BMI (51.7%).

The results of present study demonstrated also a strong and statistically significant association of fasting serum glucose with both the rate of hypomagnesemia and the mean ± SD of serum Mg level. The study proved that T2DM patients with fasting serum glucose equal to or more than 140 mg/dl had a higher rate of hypomagnesemia and lower mean ± SD of serum Mg level, when compared with

those patients with less than 140 mg/dl. However, in the previous studies. They noticed that 77.33% of T2DM patients with fasting serum glucose equal to or more than 140 mg/dl, had hypomagnesemia[29], and other studies showed that the mean ± SD of serum Mg level was 1.88 ± 0.24 mg/dl 30. Moreover, in the present study, the HbA1c assessment was used mainly as an index for glycemic control. Despite that, the mean ± SD of serum Mg level was equal in both good, and poor glycemic patients, 69.6% of those patients with poor glycemic control had hypomagnesemia compared with 30.4% with good glycemic control. Similarly, high frequency of hypomagnesemia in poor glycemic patients was found in previous studies by 31,32, which showed that 72.7% and 75% of their poor glycemic patients had hypomagnesemia.

The impact of positive family history of D.M on Mg status was also studied. We found out that 68.7% of diabetes patients had positive family history of D.M, with 46.5% from the mother side. The rate of hypomagnesemia was 56.5% (13 patients) in those patients with positive family history, and 43.5% (10 patients) in those patients with negative family history of D.M with no difference in mean ± SD of serum Mg level. Inconsistent with our results, a previous study has showed that the rate of hypomagnesemia was 11.1% in patients with positive family history and 88.9% in patients with negative family history, with no difference in mean ± SD of serum Mg level. Regarding the association between prediabetes subjects with positive family history and rate of hypomagnesemia, our finding showed a higher rate of hypomagnesemia in prediabetes subjects than in diabetes patients (80%), with similar mean values for serum Mg level in both. A study performed by 26 found a reverse association of prediabetes subjects with positive family history with rate of hypomagnesemia, 85.6% of prediabetes subjects with negative family history showed hypomagnesemia.

The association between central obesity and Mg status in T2DM patients and prediabetes subjects was observed. Centrally obese females and males showed a high rate of hypomagnesemia and a lower mean ± SD of serum Mg level, the lowest level being in females. One hundred percent of Type 2 diabetes centrally obese females showed hypomagnesemia, where 40% of Type 2 diabetes centrally obese males showed hypomagnesemia. The mean ± SD of serum Mg level among T2DM centrally obese females and males were 1.83 ± 0.25 mg/dl and 1.91 ± 0.19 mg/dl, respectively. Earlier research had come out with a similar findings which showed a high rate of hypomagnesemia among central obese females and males (86.6% and 54.7%, respectively)[33],[34]. Furthermore, the role of exercise or physical activity on Mg status in diabetes patients and prediabetes subjects was clearly observed. Higher rate of hypomagnesemia and lower mean ± SD was observed among diabetes patients who did not exercise. The frequency of hypomagnesemia among physically inactive diabetes patients was 87%. Inconsistent with our results, a previous data showed that 38.5% of diabetes patients without exercise had hypomagnesemia with equal mean ± SD of

serum Mg level (1.89 ± 0.26 mg/dl) among both exercised and no exercised patients[35],[36].

CONCLUSION:

From the present work we can conclude that patients with T2DM and subjects with prediabetes have a higher rate of hypomagnesemia and lower mean Mg level compared with healthy control subjects and that the hypomagnesemia in T2DM patients was strongly associated with old age, high body mass index, high fasting serum glucose level, high blood HbA1c percent, positive family history of diabetes, female gender and increase central obesity. Moreover, the rate of hypomagnesemia in prediabetes subjects was strongly associated with young age, high body mass index, positive family history of diabetes and female gender. For this reason an intervention study is recommended to confirm better association between hypomagnesemia and T2DM patients and prediabetes subjects. Finally, Mg rich food are recommended for daily use in order to overcome the problems of hypomagnesaemia, which may be helpful in increasing insulin sensitivity and management of diabetics.

REFERENCES:

1. Barbagallo M, Dominguez LJ. Magnesium and type 2 diabetes World J Diab. 2015;6(10):1152-7.
2. Anastasia P., Andrey V., Paolo P. The Diabetes Mellitus–Atherosclerosis Connection:The Role of Lipid and Glucose Metabolism and Chronic Inflammation. International journal of molecular sciences. 2020; 21,1821-1835.
3. American Diabetes Association. Classification and Diagnosis of Diabetes: Standards of Medical. Diabetes Care. 2018; 41(1): 513-527.
4. Cantley J., Ashcroft F. insulin secretion and type 2 diabetes. BioMed Central. 2015; 1-7
5. Babey. S, Wolstein J., Diamant A., Goldstein H. Prediabetes in California: Nearly Half of California Adults on Path to Diabetes. Health Policy Brief. 2016; 1-7.
6. Panesar K. Prediabetes management. Diabetes. 2013; 38(10): 11-17.
7. Jitendra K., Wadhwani J., Manuj S., Dubey T. Prevalence of impaired glucose tolerance in different grades of obesity. International Journal of Advances in Medicine. 2019; (6)5: 1-4.
8. Bohn T. Magnesium Absorption in Humans. Dissertation. University Frankfurt, Germany. 2003.
9. Wei J., Zeng C., Li X., Gong Q., Lei G., Yang T. Association among dietary magnesium, serum magnesium, and diabetes: a cross sectional study in middle-aged and older adults. Journal of Health, Population and Nutrition. 2016; 2-6.
10. Meludu S., Adeniyi F. Effect of magnesium supplementation on plasma glucose in patients with diabetes mellitus. Afr. J. Biomed. Res. 2001; 4: 111 – 113.
11. El-said N., Sadik N., Mohammed N. Magnesium in type 2 diabetes mellitus and its correlation with glycemic control. J Res Med Sci. 2015; 3(8):1958-1963.

12. Arundhati D., Dipti S., Uma K. Hypomagnesemia in type 2 diabetes mellitus. Indian Journal of Endocrinology and Metabolism. 2016; (16)6: 1000-1003.
13. American Diabetes Association. Standards of Medical Care in Diabetes. Diabetes Care. 2017; 40(1): 1-135.
14. Sumathi K., Chandrika A. Role of magnesium in pre diabetes. Scholars Academic Journal of Pharmacy. 2015; 5(1): 12-13.
15. Kieboom B., Ligthart S., Dehghan A., Kurstjens S., Baaij J., Franco O. et al. Serum magnesium and the risk of prediabetes: a population-based cohort study. Diabetologia. 2017; 60:843–853.
16. James O. Subclinical magnesium deficiency: a principal driver of cardiovascular disease and a public health crisis. DiNicolantonio JJ. 2018; 1-16.
17. Parlapally R., Kumari K., Jyothi S. Serum magnesium levels in type 2 diabetes mellitus. International Journal of Scientific Study. 2016; 4(5): 176-179.
18. Anjum A, Yousaf M, Zuber M, Ahmad HB, Zahoor AF. Comparative study on calcium, magnesium and cobalt in diabetic and non-diabetic patients (males) in Punjab, Pakistan. AJB 11. 2012; 7258-7262.
19. Kurstjens S., Baaij J., BourasH., Bindels R, Tack C., Hoenderop V. Determinants of hypomagnesemia in patients with type 2 diabetes mellitus. European Journal of Endocrinology. 2017; 176: 11–19.
20. Kauser M., Afreen A., Prabhakar K, Kumar V., Javarappa D. Study of Serum Magnesium in Type 2 Diabetes Mellitus and its Correlation with the Modality of Treatment. Global Journal of Medical Research. 2014; 14(4): 32-37.
21. Papanas N., Maltezos E. Metformin in the treatment of type 2 diabetes. Clinical Medicine. 2009; 1367–1381.
22. Barbagallo M., Belvedere M., Dominguez L. Magnesium homeostasis and aging. International Magnesium Symposium. 2009; 22(4): 235-246.
23. Mamatha B., Shankarprasad D., Manjula R. A comparative study of serum magnesium in type 2 diabetes mellitus patients and non diabetics and its correlation with glycemic status. International Journal of Bioassays. 2014; 3 (10): 3395-3398.
24. Farooq M., Abbasi B., Ali A., Islam N. Effect of hypomagnesemia on control of diabetes and lipid abnormalities. Medical Chaneel. 2017; 23(1): 5-11.
25. Wahid A., Verma G., Meena C., Pathan A. Study of serum magnesium level in patients with type 2 diabetes mellitus and it’s correlation with glycosylated hemoglobin and diabetic complications. International Journal of Advances in Medicine. 2017; 4(2):311-316.
26. Chen S., Jin X., Liu J., Sun T., Xie M., Bao W., Yu X. Association of Plasma Magnesium with Prediabetes and Type 2 Diabetes Mellitus in Adults. scientific reports. 2017; 7: 1-8.

27. Dahl A. Body mass index, cognitive ability and dementia. dissertation. Jonkoping University. 2009.
28. Babikr W., Shalayel M., Abdelraheem A., Elhussein A. Impact of Body Mass Index on Hypomagnesemia and Hypocalcemia in Type 2 Diabetic Patients. Biomedical & Pharmacology Journal. 2016; 9(3): 933-940.
29. Muhammad R. Hypomagnesemia in Type 2 Diabetes Mellitus. Pak Armed Forces Med J. 2016; 66(6): 845-850.
30. Mane M., Chaudhari G., Reddy E. Hypomagnesaemia in Diabetic patients and Biochemical action on the cardiovascular system. Int J Biol Med Res. 2012; 3(1): 1273-1276.
31. Shaikh M., Devrajani B., Soomro A., Shah S., Devrajani T., DasT. Hypomagnesemia in Patients with Diabetes mellitus. World Applied Sciences Journal. 2011; 12 (10): 1803-1806.
32. Shardha A., Vaswani A., Faraz A., Alam M., Kumar P.. Frequency and Risk Factors Associated with Hypomagnesaemia in Hypokalemic Type-2 Diabetic Patients. Journal of the College of Physicians and Surgeons Pakistan. 2014; 24 (11): 830-835.
33. Kamath A., Shivaprakash G., Adhikari P. Body mass index and Waist circumference in Type 2 Diabetes mellitus patients attending a diabetes clinic. International Journal of Biological & Medical Research. 2011; 2(3): 636-638.
34. Lisanne M., Joost G., René J., Jeroen H. Hypomagnesemia in Type 2 Diabetes. Diabetes. 2016; 65: 3-13.
35. Peters K., Chubb S., Davis W., Davis T. The Relationship between Hypomagnesemia, Metformin Therapy and Cardiovascular Disease Complicating Type 2 Diabetes: The Fremantle Diabetes Study. PLOS ONE. 2013; 8(9): 1-8.
36. Yael P., Eilon R., Jacob L. Hypomagnesemia is associated with new-onset diabetes mellitus following heart transplantation. Cardiovasc Diabetol. 2019; 3-10.

پوختە

نەخوشیا شەکۆیێ ژ جۆرێ (2)

باگراوند وئارمانج: دهێتە دیتن نەخوشیا شەکرێ ژجۆرێ (2) ودەسپێکێن نەخوشیا شەکرێ گرێدایینە ب کێمبوونا کەرستێ مەگنسیومێ. چێدبیت کێمبوونا مەگنسیومێ بخۆ هۆکارەکێ پشکدار بیت بۆ گەشەکرنا نەخوشیا شەکرێ ژجۆرێ (2) وئاریشێن وێ.

شێواز: ڤەکولینەك هاتە ئەنجامدان دیەکەیا نەخوشیا شەکرێ لنەخوشخانا ئازادی یا فێرکرنێ. (248) کەس بەشداربوون دڤەکولینێ، (99) نەخوشێن شەکرێ، (50) کەسێن دەسپێکێن نەخوشیا شەکرێ لدەڤ هەین، و(99) کەس وەك کونترول گروپ تەندروستیا وان یا باش بوو.

ئەنجام: ڤەکولینێ کێمبوونەکا مەزن ئاشکراکر دئاستێن مەگنسیوما خوینێ لدەڤ نەخوشێن شەرکێ ژجۆرێ (2) وکەسێن توشی نەخوشیا شەکرێ بووین ب بەراورد ب پیڤەرێن (1.88±0.23, 1.93±0.20, 2.00±0.17) **دیسیلتر** لدویف ئێك (P=0.003). رێژەیا کێمبوونا مەگنسیوما خوینێ یا بلند بوو لدەڤ نەخوشێن شەکرێ وکەسێن توشی دەسپێکێن نەخوشیا شەکرێ بووین (23.2٪ ، 10.0٪ ، 3.0٪ لدویف ئێك). بلندترین ئاستێ کێمبوونا مەگنسیوما خوینێ هاتە دیتن لدەڤ نەخوشێن شەکرێ یێن کونترولا لاواز هەی لسەر رێژەیا شەکرێ دخوینێ، کەسێن پیر، زێدەبوونا گلوکوزا خوینێ لدەمێ ڕۆژیبوونێ، کەسێن قەلەو وکێشا وان مەزن.

دەر ئەنجام: نەخو شیا شەکرێ ژ جۆرێ (2) وکە سێن دە سپێکێن نەخو شیا شەکرێ لدەڤ هەین ر ێژەکا بلندترا هەی ژکێمبوو نا مەگنسیوما خوینێ وکێمبوونا نافنجیێ ئاستێ مەگنسیومێ ب بەراورد دگەل کەسێن تەندروست.

الخلاصة

مستويات المغنيسيوم المصل بين مرضى السكري من النوع 2 والأشخاص الذين يعانون من مقدمات مرض السكري في مدينة دهوك / العراق

الخلفية والأهداف: يرتبط داء السكر من النوع الثاني وحالات ما قبل السكري ارتباطا وثيقا بالخلل في استقلاب عنصر المغنيسيوم؛ حيث ان نقص المغنيسيوم بحد ذاته قد يكون عاملاً مساهماً في تطور داء السكر من النوع الثاني ومضاعفاته.

الطرق: أجريت هذة الدراسة في وحدة امراض السكري في مستشفى آزادي التعليمي. حيث تضمنت اختيار 248 شخصًا 99 مريضًا منهم يعانون من داء السكر، و50 شخصًا يعانون من حالات ما قبل السكري بالاضافة الى 99 شخصًا تم اختياهم كمجموعة سيطرة.

النتائج: شملت الدراسة الحالية دراسة تأثير نقص عنصر المغنيسيوم عند مرضى داء السكر من النوع الثاني وحالات ما قبل السكري، حيث اشارت نتائج البحث الى وجود انخفاض معنوي في مستويات مغنيسيوم مصل الدم عند مرضى داء السكر من النوع الثاني والأشخاص في مرحلة ما قبل السكر مقارنةً مع مجموعة السيطرة (الاصحاء)، (1.88 ± 0.23، 1.93 ± 0.20، 2.00 ± 0.17ملغم/ 100مل على التوالي، P = 0.003). أظهرت نتائج الدراسة ايضا ان معدل نقص مغنسيوم الدم كان مرتفعاً بين مرضى السكر ولدى الأشخاص المصابين بحالات ما قبل السكري (23.2٪، 10.0٪، 3.0٪ على التوالي). من خلال دراسة العلاقة بين مستويات المغنيسيوم وعوامل الخطورة للحالات المرضية المدروسة؛ لوحظ ان أعلى معدل لنقص مغنسيوم الدم في مرضى السكري كان لدى المرضى الذين يعانون من ضعف التحكم في نسبة السكر في الدم، الشيخوخة، والذين يعانون من زيادة الوزن والسمنة.

الاستنتاجات: مرضى داء السكر من النوع الثاني والأشخاص الذين يعانون من حالات ما قبل السكري لديهم معدل أعلى من نقص مغنيسيوم مصل الدم وكذلك انخفاض متوسط مستوى المغنيسيوم مقارنة مع الأشخاص الأصحاء.