Erythrocyte Magnesium Levels in type I and type II Iraq diabetic patients effect of antidiabetic treatment

*Dr.Faris Abdul Kareem Kazaal M.B.CH.B., CABM, FRCP **Lewai S. Abdulaziz Ph D.
** Ragad I.Naji M.Sc

ABSTRACT

Background: Direct measurement of intracellular magnesium using erythrocytes has been suggested as a sensitive indicator for the estimation of body magnesium store. Marked depletion in plasma and erythrocyte magnesium levels was particularly evident in diabetic patients with advanced retinopathy and poor diabetic control. While insulin has been shown to stimulate erythrocyte magnesium uptake, hyperglycemia per se suppressed intracellular magnesium in normal human red cells.

Aim of the study: To investigate the erythrocyte magnesium level in Iraqi type I and II diabetic patients, with specific emphasis on the effect of both, metabolic control and the type of antidiabetic treatments.

Methods: Sixty two diabetic patients (7 with type I and 55 with type II diabetes mellitus) recruited from the outpatient diabetes clinic at the Specialized Center For Endocrine Diseases-Baghdad, during the period from 1st October 2005 to 28th February 2006. Eighteen non-diabetic normomagnesemic healthy controls matched for age and sex were participated in this study. Of the diabetics, 22 were using insulin (7 with type I and 15 with type II diabetes mellitus), 40 were taking oral antidiabetic agents (All with type II diabetes mellitus) and none were using both. Serum and erythrocyte magnesium concentration were measured for both groups, and Glycated hemoglobin levels were estimated only for diabetics.

Results : Mean serum and erythrocyte magnesium levels were significantly ($p<0.001$) lower in the diabetic group as compared to controls. Serum level of magnesium was not a significant predictor of erythrocyte magnesium concentration. No significant correlation was observed between HbA1c and erythrocyte magnesium. Significantly ($p<0.001$) lower serum magnesium levels were consistently evident through the entire diabetic subgroups as compared to controls. Erythrocyte magnesium contents were significantly ($p<0.001$) reduced in patients with type I , type II and type II receiving oral antidiabetic agents, but not in patients with type II receiving insulin ($p= 0.120$), as compared to controls. Significant difference in erythrocyte magnesium levels was observed between patients with type II receiving oral antidiabetic agents and those receiving insulin ($p<0.001$). The frequency of magnesium deficiency in diabetic patients, as judged by a lower serum magnesium reference limit was constantly 100% in all subgroups. While, judgments based upon a lower erythrocyte magnesium reference limit, discloses variable frequencies in diabetic subgroups.

conclusion: The near normal erythrocyte magnesium levels in type II insulin-receiving patients, could be credited to the stimulatory action of exogenous insulin on cellular magnesium uptake and may indicate a possible role of insulin treatment as a potential implications on health policy, by ameliorating cellular magnesium depletion in the continuously expanding diabetic population.

key words: Diabetes, Iraqi, Erythrocyte, Magnesium, Insulin

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Introduction

Among the endocrine and metabolic disorders associated with magnesium deficiency, diabetes mellitus is the most common [(1)]. Recently, the prevalence of hypomagnesemia among Iraqi diabetics was shown to be extremely higher than any worldwide reported study [(2)]. Although, hypomagnesemia reliably indicates magnesium deficiency; normal plasma magnesium concentration does not necessarily exclude magnesium depletion. Moreover, during acute or chronic experimental magnesium deficiency a change in erythrocyte magnesium was observed, which occurs more slowly and discreetly than in plasma magnesium [(3)]. When serum magnesium decreased, hormones that secured magnesium homeostasis in blood changes, and more magnesium is transported from the cell to the blood sera. This indicates that intracellular magnesium concentration is a more sensitive parameter to detect hypomagnesemia in organism[(4)]. Hence, direct measurement of intracellular magnesium using erythrocytes has been suggested as a more sensitive indicator for the estimation of body magnesium store [(5)].

However, major differences in erythrocyte magnesium content have been shown to exist between different ethnic groups, as well as among subjects within a given population. These interethnic and interindividual variations appeared to be relatively stable and partly independent of the climate in which the subjects were residing. Leadingly the hypothesis of genetic control of the erythrocyte magnesium level in humans has been evolved [(6)]. The

existence of a genetic regulation of erythrocyte magnesium content has been fully confirmed by Darin et al., who were able to discriminate the genetic component from the familial non-genetic component and stating that environmental factors had some influence on the plasma magnesium and none or very little on the erythrocyte magnesium values[7].

Erythrocyte magnesium levels are thought to reflect extracellular magnesium at the time of erythropoesis with a small, gradual loss of intracellular magnesium during their life span [8]. It has been suggested in human studies that any magnesium entering the cells did so during early phases of erythropoesis, with subsequent decrease during cell ageing. Increased reticulocytes count might result in an increase in the population of younger erythrocytes with higher magnesium concentration. Thus, concentration of erythrocyte magnesium is related to their age and that their quick renewal goes with an increase of erythrocyte magnesium unrelated to a magnesium overload[4,8]. Genetically low erythrocyte magnesium values could, therefore, be ascribed to the older age of erythrocytes [3].

The existence of a close relationship between metabolic control and impaired magnesium balance was confirmed by the groundwork of Fujii et al., who observed that a marked depletion in plasma and erythrocyte magnesium levels was particularly evident in diabetic patients with advanced retinopathy and poor diabetic control [9]. Later, low erythrocyte magnesium has been described in type I and II diabetic subjects [10].Moreover, reduction in plasma, erythrocyte and platelet magnesium levels in patients with type II diabetes mellitus were reported by Corica et al. [11], who argued that this condition may underlie platelet function alterations, which in turn can exacerbate vascular complications from diabetes.

In a cross-sectional study, electromyographical (EMG) signs of polyneuropathy were shown to be significantly more frequent in type-I diabetic patients with low erythrocyte magnesium [12]. In two subsequent intervention studies, logistic regression analysis shows that longer duration of diabetes and low erythrocyte magnesium are the major determinants of polyneuropathy evolution. In the short-term study, one year supplements result in an improve nerve conduction in patients presented with early signs of the neurological complication[12]. While supplements for a period of 5 years was able to restore a normal magnesium status and influence favorably the natural evolution of polyneuropathy as compared to non supplemented type-I diabetic patients, however, normalization of EMG signs was only seen in incipient polyneuropathy[13].

In healthy subjects, insulin has been shown to stimulate erythrocyte magnesium uptake [5]. More recently, a positive effect of insulin administration over intracellular magnesium concentrations was evident, stimulation with 100 mU insulin induces an improvement in intracellular magnesium concentration in obese children and patients with type I and II diabetes mellitus [14]. Furthermore, hyperglycemia, independent of insulin, due to oral glucose ingestion has a potential role in cellular ionic changes of magnesium. The hyperglycemia per se suppressed intracellular magnesium in normal human red cells [15].

The study presented herein, was aimed to investigate the erythrocyte magnesium level in Iraqi type I and II diabetic patients, with specific emphasis on the effect of both, metabolic control and the type of antidiabetic treatments.

Methods

Patients and Controls: Sixty two diabetic patients (7 with type I and 55 with type II diabetes mellitus) recruited from the outpatient diabetes clinic at the Specialized Center For Endocrine Diseases-Baghdad, during the period from 1st October 2005 to 28th February 2006. Eighteen non-diabetic, normomagnesemic (Serum magnesium >0.7mmol/l)[16], healthy controls matched for age and sex were participated in this study. Exclusion criteria for both groups included high reticulocytes count (>2.5%), diarrhea and loop diuretics, known to be associated with higher fecal and urinary magnesium losses, respectively. None were taking magnesium supplements.

Age, body mass index and sex distribution were comparable between both study groups. Of the diabetics, 22 were using insulin (7 with type I and 15 with type II diabetes mellitus), 40 were taking oral antidiabetic agents (All with type II diabetes mellitus) and none were using both. Characteristics of the study groups are included in Table 1.

Sampling and Preparation of Erythrocyte haemolysate: Whether the subjects were in the fed or fasting state was not specified. Venous blood specimens from both groups were drawn,

four milliliters were transferred into heparinised 5 ml tubes. The residual blood was allowed to coagulate and serum was separated from blood cells by centrifugation at 3000 rpm for 15 minutes.

Heparinised blood samples were centrifuged at 800g for 15 minutes, and platelets rich plasma was carefully aspirated. The last 0.25cm layer of platelets rich plasma was left behind to avoid drawing up reticulocytes and young red blood cells. Packed erythrocytes were then washed three times with 3 ml of ice-cold 0.15M saline. After each cell washing, the cell suspension was centrifuged at 1200g for 5 min. The final packed cells were then lysed by three consecutive freezing-thawing cycles, diluted 1:1 with ice-cold deionized water and membranes were removed by centrifugation at 2000g for 20 min [(5)].

Biochemical Assay: Serum level of magnesium was estimated spectrophotometerically, using the Blue Xilidil method (Giesse Diagnostics, Roma-Italy). Glycated hemoglobin (HbA1c), in heparinised blood samples, was determined colorimetrically using weakly binding cation-exchange resin and a resin separator (Stanbio Glycohemoglobin Pre-Fil®, Texas-US). For the determination of erythrocyte magnesium, haemolysates were diluted with 0.3M HCl [(17)] and the analysis was performed in an acetylene-air flame atomic absorption spectrophotometer as described by Martin and Shapiro [(18)]. Erythrocyte magnesium concentrations are expressed as mmol per liter of packed cells (mmol/l) and as micromoles per gram of haemoglobin (µmol/g.Hb) [(5)].

Statistical Analysis: Data processing and statistical analysis were done using Excel 2003 (Microsoft, Seattle WA, USA). Normal distribution of data was verified by calculating the quotient of the skewness; normal distribution was assumed if the quotient was between –2.5 and +2.5. Normally distributed data were expressed as arithmetic means ± SD. Reference ranges for all parameters were defined as mean±2SD and magnesium deficiency was defined as serum or erythrocyte magnesium concentration below the lower reference limit.. Differences between groups were evaluated using unpaired Student's t-test and considered statistically significant at $p < 0.05$. ANOVA was aimed to test for association between serum and erythrocyte magnesium concentration, in addition to HbA1c levels .

Results

As presented in Table 2, mean serum and erythrocyte magnesium (expressed either as mmol/l or as µmol/g.Hb) levels were significantly ($p<0.001$) lower in the diabetic group (0.486 ± 0.08 mmol/l, 1.287 ± 0.56 mmol/l and 4.229 ± 1.80 µmol/g.Hb), as compared to controls (0.768 ± 0.02 mmol/l, 1.899 ± 0.10 mmol/l and 6.118 ± 0.65 µmol/g.Hb). The metabolic control of the diabetic patients varied broadly as indicated by the wide-ranging HbA1c values (7.31-12.46% ; 10.84%± 1.96).

In both study groups, serum level of magnesium was not a significant predictor of erythrocyte magnesium concentration. Similarly, in the diabetic group, no significant correlation was observed involving HbA1c and erythrocyte magnesium (expressed either as mmol/l or as µmol/g.Hb). However, the correlation between the two expressions of erythrocyte magnesium content was highly significant (Table 3).

Mean levels of serum and erythrocyte magnesium in diabetic patients, subgrouped according to disease type and the type of antidiabetic treatments are summarized in Table 4. Significantly ($p<0.001$) lower serum magnesium levels were consistently evident through the entire diabetic subgroups as compared to controls (0.481±0.05, 0.487±0.09, 0.484±0.09, 0.494±0.08 and 0.768±0.02 mmol/l for type I , type II, type II receiving oral antidiabetic agents, type II receiving insulin and controls, respectively). Erythrocyte magnesium contents were significantly ($p<0.001$) reduced in patients with type I , type II and type II receiving oral antidiabetic agents (1.323±0.48, 1.282±0.57, 1.138±0.44 mmol/l, respectively), but not in patients with type II receiving insulin (1.673±0.73 mmol/l; $p= 0.120$), as compared to controls (1.899±0.10 mmol/l). Moreover, within diabetics, significant differences in erythrocyte magnesium levels were observed only between patients with type II receiving oral antidiabetic agents and those receiving insulin ($p<0.001$).

The frequency of magnesium deficiency in diabetic patients, as judged by a lower serum magnesium reference limit of 0.728 mmol/l, was constantly 100% in all subgroups. However, judgments based upon a lower erythrocyte magnesium reference limit of 1.699 mmol/l, discloses variable frequencies in diabetic subgroups (85.7, 80.0, 90.0 and 53.3 % for

type I , type II, type II receiving oral antidiabetic agents and type II receiving insulin, respectively

Discussion:

Serum magnesium levels, and the frequencies of hypomagnesemia, in the diabetic patients of the current study were highly concurred with those reported in our previous one[2]. Contrarily , all the controls were normomagnesemic, as compelled by the enrollment criteria, which, in our opinion, was necessary to establish a reliable reference measure of erythrocyte magnesium contents, unbiased by the inclusion of low body magnesium subjects. However, it should be stressed here that normal plasma magnesium concentration does not necessarily exclude magnesium depletion, and an elimination of the marginal chronic magnesium deficit is mandatory before proclaiming any reference levels [3].

Furthermore, bias may be provoked by the notion that concentrations of erythrocyte magnesium, distinct from body magnesium status, are related to their age and quick renewal [4,8]. The exclusion of any patient or control with abnormal reticulocytes, in the present study, was intended to diminish such effect. Accordingly, the highly significant correlation observed here, between the two erythrocyte magnesium expressions in diabetics and controls, may reflect the normal reticulocytes and haemoglobin levels in both study groups.

The mean erythrocyte magnesium concentration of our controls, was slightly higher than that established in healthy Netherlanders (1.72±0.16mmol/l)[19], nearly analogous to that estimated in nondiabetic Germans (1.84±0.10mmol/l)[20], but much lower than that reported from Poland (2.42±0.34mmol/l)[4]. Moreover, erythrocyte magnesium contents of healthy adult male blood donors examined in Paris-France revealed a wide range (1.55-2.89mmol/l), with an uncorrected mean normal levels of 2.08±0.14mmol/l , and of 2.3±0.24mmol/l after eliminating the marginal chronic magnesium deficit[3]. Alternatively, normal erythrocyte magnesium concentrations expressed as µmol magnesium per gram haemoglobin were described to be in the range of 3.63-6.42[21], which is less than that estimated in the present study. These variations might reflect the large and fairly stable interethnic and interindividual variations, as well as the effects of environmental factors[6,7].

In the present study, using either expression, magnesium contents of diabetics' erythrocytes were significantly lower as compared to nondiabetic controls. This has been repeatedly observed by formerly published studies[9-13,20,22-23], and ascribed mainly to an increased fractional urinary magnesium excretion, due to glucose-induced osmotic diuresis, especially in patients with unsatisfactory metabolic control[20]. Epidemiological studies, on the other hand, had shown low magnesium intake in a world-wide level [24], with an average ingestion of magnesium being frequently below the Recommended Dietary Allowance, which may further promotes the development of magnesium deficiency[25].

The pathophysiological mechanism responsible for magnesium depletion in diabetes mellitus has remained controversial, particularly the relative roles of hyperglycemia and insulin resistance[26], and with the presence of additional influential factors, affecting magnesium metabolism, the relation between glycemic status and body magnesium indices might not be easily confirmable. Thus, the probable increased sweat losses of magnesium induced by our tremendous hot climate[2], along with the possible reduction in magnesium intake due to the drastic shift from the consumption of tap drinking water to a lower magnesium containing bottled water [27], may explain the absence of any significant correlation between glycated haemoglobin levels and the concentrations of magnesium in serum and erythrocytes of the diabetic patients participated in the present study.

Insulin, by regulating ATPase pump activity, stimulates the transport of magnesium through the cellular membrane[28], and an impaired insulin-induced entry of magnesium, due to relative or absolute insulin deficiency, may complicate intracellular magnesium depletion[5]. Hence, the current significantly lower erythrocyte magnesium levels observed in type II patients receiving oral antidiabetic agents, as compared to the near normal erythrocyte magnesium levels in type II insulin-receiving patients, could be credited to the stimulatory action of exogenous insulin on cellular magnesium uptake. However, in the present study, such outcome was not significantly evident in type I diabetics, nonetheless, more than half of our patients, in type II receiving insulin subgroup, were still having erythrocyte magnesium levels below the specified lower

reference limit. This could be attributed to the small number of patients investigated in type I subgroup, and also to the heterogeneous nature of the investigated patients in terms of the type of insulin therapy, adherence to treatment, as well as, the variations in dietary magnesium intake.

Consequently, results of the present study, consistent with the previous findings of Takaya et al.[14], may indicate a possible role of insulin treatment as a potential implications on health policy, by ameliorating cellular magnesium depletion in the continuously expanding diabetic population. This notion is based mainly on the cumulative reported evidences acc using intracellular magnesium depletion as a pathogenic factor in the progression of macro and microvascular diabetic complications[11-13,24]. Further evaluation of this role, in adjunct to magnesium supplementation, as a useful innovative approach to the problem of preventing these complications, should be the focus of future studies.

Conclusion:

The near normal erythrocyte magnesium levels in type II insulin-receiving patients, could be credited to the stimulatory action of exogenous insulin on cellular magnesium uptake and may indicate a possible role of insulin treatment as a potential implications on health policy, by ameliorating cellular magnesium depletion in the continuously expanding diabetic population.

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Table 1: Characteristics of study groups.

Characteristics	Controls	Diabetics	P
N	18	62	
Age (years)	49.8±17.4	53.4±15.2	NS
BMI (Kg/m2)	25.9±4.7	27.1±6.3	NS
Gender (M/F)	7/11	24/38	NS
Type of DM : NIDDM	_	55	
IDDM	_	7	
Duration (years)	_	12.3±9.2	
Treatment : Oral anti-diabetic agents	_	40	
Insulin	_	22	

Table 2: Mean Serum and Erythrocyte Magnesium levels in Diabetics and Controls.

Characteristics	Controls Mean±SD [Range]	Diabetics Mean±SD [Range]	P
Serum Mg (mmol/l)	0.768±0.02 [0.732 - 0.802]	0.486±0.08 [0.370 - 0.679]	<0.001
Erythrocyte Mg (mmol/l)	1.899±0.10 [1.728 - 2.057]	1.287±0.56 [0.745 - 2.674]	<0.001
Erythrocyte Mg (µmol/g.Hb)	6.118±0.65 [5.239 - 7.660]	4.229±1.80 [2.096 - 10.000]	<0.001
HbA_1c (%)	Not Estimated	10.84± 1.96 [7.31 – 12.46]	--

Table 3: Correlation between Serum and Erythrocyte Magnesium levels in Diabetics and Controls.

Group		R	Erythrocyte Mg (mmol/l)	Erythrocyte Mg (µmol/g.Hb)
Controls	n = 18	Serum Mg (mmol/l)	- 0.055 (NS)	0.157 (NS)
		Erythrocyte Mg (mmol/l)	—	0.774 (P<0.001)
Diabetics	n = 62	Serum Mg (mmol/l)	0.010 (NS)	- 0.054 (NS)
		Erythrocyte Mg (mmol/l)	—	0.959 (P<0.001)
		HbA_1c (%)	- 0.052 (NS)	- 0.035 (NS)

Table 4: Mean Serum and Erythrocyte Magnesium levels and frequencies of magnesium deficiency in Diabetic with different treatments.

Group	n	Serum Mg (mmol/l)	Frequency of Mg deficiency	Erythrocyte Mg (mmol/l)	Frequency of Mg deficiency
Control	18	0.768±0.02	0 (0.0%)	1.899±0.10	0 (0.0%)
Diabetics:	62	0.486±0.08*	62 (100%)	1.287±0.56*	50 (80.7%)
Type I	7	0.481±0.05*	7 (100%)	1.323±0.48*	6 (85.7%)
Type II:	55	0.487±0.09*	55 (100%)	1.282±0.57*	44 (80.0%)
Oral anti-diabetic agents	40	0.484±0.09*	40 (100%)	1.138±0.44*	36 (90.0%)
Insulin	15	0.494±0.08*	15 (100%)	1.673±0.73†	8 (53.3%)

*P<0.001 versus controls.
†P<0.01 versus Type II (oral anti-diabetic).

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*From the Department of Medicine , Al-Kindy Collage of Medicine
**From the department of Chemistry ,Al-Kindy Collage of Medicine
Correspondence Address
Dr. **Faris Abdul Kareem Khazaal**

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