Status of serum magnesium in Egyptian children with type 1 diabetes and its correlation to glycemic control and lipid profile

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Abstract
Diabetes mellitus has been suggested to be the most common metabolic disorder associated with magnesium deficiency, having 25% to 39% prevalence. This deficit could be associated with the development of late diabetic complications, especially macroangiopathy.

We aimed to evaluate the status of serum Mg in children with type 1 diabetes and assess its relation to glycemic control and lipid profile.

We included 71 Egyptian children with type 1 diabetes having their follow-up at Pediatric Endocrinology outpatient clinic, Zagazig University Hospital and 71 age- and sex-matched control. We measured Serum magnesium, HbA1c, and lipid profile in all study subjects.

Diabetic children had significantly lower serum magnesium level compared to control children (1.83 ± 0.27 mg/dL in diabetic children versus 2.00 ± 1.6 mg/dL in control children). Taking cut-off level of serum magnesium < 1.7 mg/dL for definition of hypomagnesemia, hypomagnesemia was detected in 28.2% of diabetic children compared to 9.9% of control children. In diabetic patients, there was statistically significant difference in HbA1c between hypomagnesemic and normomagnesemic group being higher in the low magnesium group, as it is mean ± SD was 11.93 ± 3.17 mg/dL in group I versus 8.92 ± 0.93 mg/dL in the normomagnesemic group. Serum magnesium was found to be positively correlated with HDL (P < 0.001), and negatively correlated with age, HbA1c, triglycerides, total cholesterol, LDL, and duration of diabetes (P < 0.001).

We concluded that total serum magnesium was frequently low in Egyptian children with type 1 diabetes and it is correlated with HbA1c and with lipid profile. Hypomagnesemia was more evident in patients with poor diabetic control and those with higher other atherogenic lipid parameters. We suggest that low serum magnesium may be included in pathogenesis of poor glycemic control and abnormal lipid profile in children with type 1 diabetes. We need to perform further studies on giving magnesium supplements in diabetic children with hypomagnesemia to observe the effect of correction of serum magnesium on glycemic control, lipid profile, and the risk of diabetic complications.

Abbreviations: HDL = high-density lipoprotein, LDL = low-density lipoprotein, Mg = magnesium, MW = Mann–Whitney U test, Q = quartile, T1DM = type 1 diabetes mellitus, T2DM = type 2 diabetes mellitus.

Keywords: children, diabetes, lipid, magnesium

1. Introduction
Type 1 diabetes is a disorder that arises following the autoimmune destruction of insulin-producing pancreatic β cells. Type 1 diabetes is a complex metabolic disorder of heterogeneous etiology with social, behavioral, and environmental risk factors unmasking the effects of genetic susceptibility. There is a strong hereditary (likely multigenic) component to the disease, with the role of genetic determinants illustrated when differences in the prevalence of type 2 diabetes mellitus in various racial groups are considered. Furthermore, the recent increases observed in diabetes mellitus prevalence are too quickly to be the result of increased gene frequency and altered gene pool, emphasizing the importance of environmental factors. Treatment of choice is lifestyle intervention followed by pharmacological treatment (e.g., metformin). New drugs such as dipeptidyl peptidase
inhibitors or glucagon like peptide 1 mimetics are in the pipeline for treatment of youth with type 2 diabetes mellitus.[6]

Increasing attention has been given to the role of certain elements in the pathogenesis of diabetes mellitus and in the progression of its complications.[7]

DM is associated with alteration of the metabolism of micronutrients with magnesium (Mg) being the most studied in this regard. Mg plays an important role in carbohydrate metabolism; it may influence the release and activation of insulin, the hormone that helps to control blood glucose levels.[8]

Magnesium is the fourth most abundant cation in the body and its vast majority is stored intracellularly. The major organs involved in magnesium homeostasis are the gut, bone, and kidney, but the regulators affecting these organs at the cellular level are not yet fully understood.[9]

The role of magnesium in the body is widespread. It is an essential cofactor of more than 300 enzymes including those important in glycolysis, transcellular ion transport, neuromuscular transmission, synthesis of carbohydrates, proteins, lipid and nucleic acids, and release of end response to certain hormones.[10]

Diabetes mellitus has been suggested to be the most common metabolic disorder associated with magnesium deficiency, having 25 to 39% prevalence.[11]

Numerous causes for low magnesium levels in diabetics can be listed including diets low in magnesium, osmotic diuresis that leads to high renal excretion of magnesium, insensitivity to insulin that affects intracellular magnesium transport and causes increased loss of extracellular magnesium, usage of loop and thiazide diuretics that promote magnesium wasting, diabetic autonomic neuropathies, and reduced tubular reabsorption due to insulin resistance. Additionally, continuous magnesium deficiency correlates to higher levels of TNFα, which may also contribute to post-receptor insulin resistance.[12]

Such Mg deficits have been linked to the development of atherosclerosis,[13,14] and in patients with coronary atherosclerosis, a Mg deficit has been related to an atherogenic lipid profile.[15] Therefore, Mg deficit in patients with type 1 diabetes could be associated with the development of late diabetic complications, especially macroangiopathy.[16]

Several studies were focused on evaluating Mg status in patients with type 2 diabetes and on role of Mg supplementation in prevention of diabetic complications and optimization of diabetic control. However, few studies have been concerned with this issue in children with type 1 diabetes with opposing results. We aimed to evaluate the status of serum Mg in Egyptian children with type 1 diabetes and assess its relation to glycemic control and lipid profile. We found in our study a higher percentage of hypomagnesaemia in diabetic children relative to controls. Also we observed a negative correlation between serum magnesium and each of HbA1c and serum triglycerides, total cholesterol, LDL as well as duration of diabetes. However, there was a positive correlation between serum magnesium and HDL level. These results draw our attention to importance of monitoring serum magnesium level in children with type 1 diabetes, monitoring patients for possible complications associated with hypomagnesaemia. Whether hypomagnesaemia detected is a cause or consequence of bad diabetic control is not known.

2. Methods

A gender and age matched case control study was conducted at Pediatric Endocrinology Outpatient Clinic, Zagazig University, Egypt, during the period from February 2014 to December 2014.

2.1. Inclusion criteria

Subjects who met the following criteria were consecutively enrolled during their follow up at Pediatric Endocrinology Outpatient Clinic, Zagazig University

1. Type 1 diabetes
2. Age between 1 and 18 years.
3. Both sexes

2.2. Exclusion criteria

1. Children who have renal disease detected by serum urea and creatinine test
2. Diuretics usage in the last 2 weeks.
3. Children with persistent diarrhea and vomiting.

2.3. Study groups

1. Patient group: 71 children with type 1 diabetes.
2. Control group: 71 age- and sex-matched healthy subjects recruited from community.

2.4. Methods

All subjects underwent the following:
1. Thorough history taking and complete physical examination.
2. Routine investigations according to our local standards every 3 months, for example, complete blood count, renal function tests, random blood glucose, glycosylated hemoglobin, and so on.
3. Special investigations:
   a. Serum magnesium level by Integra 400 Plus (Roche, Germany).
   b. Lipid profile by Integra 400 Plus (Roche, Germany).

2.5. Ethics

The protocol developed is according to Declaration of Helsinki 1964, as revised in 2000, and was approved by the institutional review board at our faculty. Informed consent was obtained from all patients’ guardians prior to participation.

2.6. Statistical analysis

All data were collected, tabulated and statistically analyzed using SPSS 15.0 for windows (SPSS Inc., Chicago, IL). Continuous Quantitative variables, for example, age were expressed as the mean ± SD & median (range), and Categorical Qualitative variables were expressed as absolute frequencies “number” & relative frequencies (percentage).

All values are expressed as median and range or mean ± SD. Differences between groups were assessed by paired Student’s t test or the Mann–Whitney U (MW) test. Correlation between variables was assessed using Spearman rank correlation coefficient. A P value of < 0.05 was considered significant.

3. Results

The study included 71 patients with type 1 diabetes (32 males and 39 females); their mean age was 9.68 ± 3.99 years and that of 71
control children (43 males and 28 females) was 9.48 ± 3.23 years. Diabetic children had significantly lower serum magnesium level compared to control children (1.83 ± 0.27 mg/dL in diabetic children versus 2.00 ± 0.16 mg/dL in control children). Taking cut off level of serum magnesium <1.7 mg/dL for definition of hypomagnesemia,17 hypomagnesemia was detected in 28.2% of diabetic children compared to 9.9% of control children (Table 1).

There was statistically significant difference in duration of diabetes between the hypomagnesemic and normomagnesemic diabetic groups, as mean ± SD of diabetes duration was 4.8 ± 2.39 years in the hypomagnesemic group versus 1.94 ± 1.45 years in normomagnesemic group with P < 0.001. There was statistically significant difference in duration of diabetes in diabetic patients and controls as regard serum magnesium level. The most contributing factor was serum magnesium which has the lowest value of serum Mg (<1.67 mg/dL), has the longest duration of diabetes (mean = 4.68) and the highest HbA1c (mean = 10.15) (Table 4). A negative correlation was found between serum magnesium level and HbA1c (r = −0.625, P < 0.001) (Fig. 1). Q1 also was associated with lowest serum HDL level and highest value of other serum lipid parameters (P < 0.001) (Table 5).

There are many factors affecting serum magnesium level in diabetic cases, including age, sex, and duration of diabetes (Table 6). The most contributing factor to hypomagnesemia was duration of diabetes which exhibited statistically high significant difference (P < 0.001) (Fig. 2). There are many factors affecting HbA1c level in diabetic cases, including age, sex, duration of diabetes, and serum magnesium level. The most contributing factor was serum magnesium which exhibited statistically high significant difference (P < 0.001) (Table 7).

### Table 1

| Magnesium level, mg/dL | Diabetic patients (n=71) | Controls (n=71) | Test | P |
|------------------------|-------------------------|----------------|------|---|
| Mean±SD                | 1.83±.27                | 2.00±.16       | MW   | <0.001|
| Median, range          | 1.93 (1.2–2.3)          | 2.03 (1.6–2.2) | 1377 | <0.001|
| Low level, <1.7 mg/dL  | 20                      | 7              | χ²   | 0.005 |
| Normal level, ≥1.7 mg/dL | 51               | 64             |      |      |

χ² = chi-square test, MW = Mann-Whitney U test, P < 0.05: significant, SD = standard deviation.

1 Normal range of serum magnesium 1.7–2.2 mg/dL.

### Table 2

| Lipid profile of diabetic patients and controls. | Diabetic patients (n=71) | Controls (n=71) | Test | P |
|-----------------------------------------------|-------------------------|----------------|------|---|
| Triglycerides, mg/dl                          | MW                     |                |      |   |
| Mean±SD                                      | 116.87±34.98           | 90.34±13.56    | <0.01|   |
| Median, range                                | 108 (70-137)           | 83 (60-140)    |      |   |
| Total cholesterol, mg/dl                     | MW                     |                |      |   |
| Mean±SD                                      | 192.72±50.34           | 159.24±12.1    | <0.001|   |
| Median, range                                | 168 (135-299)          | 151.41 (125-180) | 71   |   |
| LDL, mg/dl                                   |                         |                |      |   |
| Mean±SD                                      | 89.62±22.9             | 70.32±12.42    | <0.05|   |
| Median, range                                | 83 (65–150)            | 60 (45–99)     |      |   |
| HDL, mg/dl                                   | MW                     |                |      |   |
| Mean±SD                                      | 40.42±15.57            | 60.8±8.36      | <0.01|   |
| Median, range                                | 51 (16–61)             | 50 (41–85)     |      |   |

HDL = high-density lipoprotein, LDL = low-density lipoprotein, MW = Mann-Whitney, SD = standard deviation.

P < 0.05 was significant.
Table 4
Comparison of serum magnesium concentration quartiles as regard clinical and laboratory findings.

| Serum Mg, mg/dL | Q1 N=18 | Q2 N=19 | Q3 N=20 | Q4 N=14 | Test and P value |
|----------------|--------|--------|--------|--------|-----------------|
| Mean           | 1.43   | 1.82   | 2.01   | 2.11   | KW 65.541       |
| SD             | 0.13   | 0.08   | 0.01   | 0.08   |                 |
| Median         | 1.42   | 1.83   | 2.01   | 2.12   | P < 0.001       |
| Range          | 1.2–1.7| 1.7–1.9| 1.9–2.02| 2.02–2.3|                |

Duration of diabetes, y

| Mean  | 4.68  | 2.28   | 1.79   | 2.29   | F 8.878         |
| SD    | 2.35  | 1.8    | 1.52   | 1.74   |                 |
| Median| 4.5   | 2      | 1.5    | 1.5    | P < 0.001       |
| Range | 0.4–8 | 0.28–5.8| 0.25–5 | 0.5–5  |                 |

HbA1c, %

| Mean  | 10.15 | 9.57   | 8.61   | 8.61   | F 13.934        |
| SD    | 0.9   | 0.79   | 0.74   | 0.98   |                 |
| Median| 10.15 | 9.5    | 8.6    | 8.45   | P < 0.001       |
| Range | 8.2–12| 7.5–11 | 7.3–11 | 6.5–10 |                |

Glycemic control[40]

| Good, HbA1c < 7.5% | 0 (0%) | 0 (0%) | 1 (5%) | 1 (7.1%) | χ² 21.246 |
| Poor, HbA1c 7.5–8.5%| 8 (44%)| 15 (78.9%)| 18 (90%)| 13 (92.9%)| P = 0.002 |
| Very poor, HbA1c > 8.5%| 10 (55.6%)| 4 (21.1%)| 1 (5%) | 0 (0%) |          |

χ² = chi-square test, F = ANOVA test, KW = Kruskal–Wallis test, Mg = magnesium, P < 0.05 = significant, Q1 = serum magnesium <1.67 mg/dL, Q2 = serum magnesium 1.67–1.93 mg/dL, Q3 = serum magnesium 1.93–2.02 mg/dL, Q4 = serum magnesium >2.02 mg/dL, SD = standard deviation.

4. Discussion

Type I diabetes mellitus is the most life-threatening endocrine disorder of children and its incidence appears to be increasing.[18]

Diabetes mellitus is the most chronic disease studied with respect to serum Mg level. Magnesium plays a significant role in glucose and insulin metabolism, mainly through its impact on tyrosine kinase enzyme, magnesium may directly affect Glucose Transporter protein activity 4 and regulate glucose translocation into the cell.[19]

The serum Mg level has been assessed in type 2 DM in different studies. However, there are few studies, concerned with serum magnesium and its relation to diabetic control in type I diabetes. Among these studies, there was a great controversy in results regarding frequency of hypomagnesemia and correlation between magnesium level and HbA1c.

4.1. Percentage of hypomagnesaemia in study groups

In our study, we found statistically significant difference in percentage of hypomagnesemia between diabetic patients and control group being higher in diabetic group, as percentage was 28.2% in the diabetic group versus 9.9% in the control group with lower level of serum magnesium in patients (1.83 mg/dL) versus (2.00 mg/dL) in control children. These results are in concordance with Jiancheng et al[20] on 25 children with type I diabetes from Northeast area of China that revealed a lower serum magnesium level in type 1 diabetic children compared to control subjects. Lin et al,[21] Salmonowicz et al,[22] and Bjelakovic et al[23] have found a lower serum total magnesium in type 1 diabetic children especially compared to their healthy control. Indeed, Fort and Lifshitz[24] and Tuvemo et al[25] have found a lower serum total magnesium in type 1 diabetic children especially those with poor glycemic control when compared to their healthy age- and sex-matched control Also, our results are concordant with Seyoum et al[26] that found a higher percentage of hypomagnesemia (65%) among adults with type I and type II diabetes compared to control group and that is considered higher than that the percentage in our study. This may be due to the difference in the age of study population between it and ours. However, Hussmann et al[27] have found a reduction in ionized serum magnesium with preserved total serum magnesium in children with type 1 diabetes relative to their healthy control.

Inconsistent with our results, Matthiesen et al[28] failed to show any statistically significant difference in serum ionized Mg between Danish children with type I diabetes mellitus and control group, and also opposite to our result, Mikhail and Ehsani-poor[29] showed that serum ionized Mg was significantly higher in type 2 diabetic patients than controls. This difference may be related to different studies populations between us and them. Moreover, poorer glycemic control in Egyptian children when compared to Danish children as represented by HBA1c. Also, in our study, we measured the total serum magnesium and not the ionized one unlike the previous 2 studies. Roffi et al[30] showed...
similar concentration of serum magnesium in type 1 diabetic children and their control but their study was focused mostly on newly diagnosed type 1 diabetes patients only. This is considered supportive to our results as we found that hypomagnesemia is seen mainly in T1DM children with longer duration of diabetes.

4.2. Correlation of serum magnesium to glycemic control

By dividing study subjects to 4 quartiles based on the serum magnesium level, we found that Q 1 with the lowest serum magnesium level was associated with longer duration of diabetes, higher HbA1c, and poor glycemic control versus other quartiles with higher serum magnesium. We observed also a negative correlation between serum magnesium level and HbA1c ($r = -0.625$, $P < 0.001$). This agreed with Galli and Maggana [17] on Athens type I diabetic children that showed a lower Mg level in patients with poor glycemic control with high HbA1c. Ramadass et al[31], Sinha and Sen[32] showed similar results in adult patients with type II diabetes. Also Mikhail and Ehsanipoor[29] observed an inverse correlation between total Mg and HbA1c.

Inconsistent with our result, Lin et al[21] and Salmonowicz et al[33] did not show any correlation between serum magnesium level and HbA1c in type I diabetic children and adolescents. Also, Mathiesen et al[28] did not observe any relationship between HbA1c and ionized magnesium and Wegner et al[33] failed to show any relation between serum magnesium and diabetic controls in the form of Fasting Blood Glucose. Again this difference between these studies and ours could be attributed to the difference in study populations, degree of diabetic control

Table 5
Comparison of serum magnesium concentration quartiles as regard the lipid profile.

| Quartiles | Q1 N=18 | Q2 N=19 | Q3 N=20 | Q4 N=14 |
|-----------|---------|---------|---------|---------|
| Triglyceride, mg/dL | | | |
| Mean | 165 | 107.58 | 94.25 | 99.93 |
| SD | 17.08 | 23.57 | 15.37 | 25.78 |
| Median | 165 | 103 | 98.5 | 87 |
| Range | 113–197 | 78–162 | 70.133 | 72–159 |
| Total cholesterol, mg/dL | | | |
| Mean | 2.685 | 180.21 | 160.1 | 158.86 |
| SD | 29.19 | 28.81 | 11.72 | 15.46 |
| Median | 274 | 176 | 157.5 | 161 |
| Range | 169–299 | 153–279 | 147–201 | 135–186 |
| LDL, mg/dL | | | |
| Mean | 118.67 | 88 | 74.55 | 76 |
| SD | 17.8 | 15.42 | 12.31 | 11.85 |
| Median | 118.5 | 89 | 71 | 78 |
| Range | 70–15 | 62–120 | 60–105 | 50–103 |
| HDL, mg/dL | | | |
| Mean | 29.56 | 50.11 | 59.6 | 59.57 |
| SD | 9.69 | 10.63 | 10.19 | 7.75 |
| Median | 28.5 | 49 | 57 | 59 |
| Range | 16–62 | 26–71 | 48–81 | 50–73 |

$\chi^2$ = chi-square test, $F$ = ANOVA test, HDL = high-density lipoprotein, KW = Karskall–Wallis test, LDL = low-density lipoprotein, Mg = Magnesium, $P < 0.05 = $ significant, Q1 = serum magnesium <1.67 mg/dL, Q2 = serum magnesium 1.67–1.93 mg/dL, Q3 = serum magnesium 1.93–2.02 mg/dL, Q4 = serum magnesium >2.02 mg/dL, SD = standard deviation.

4.2. Correlation of serum magnesium to glycemic control

By dividing study subjects to 4 quartiles based on the serum magnesium level, we found that Q 1 with the lowest serum magnesium level was associated with longer duration of diabetes, higher HbA1c, and poor glycemic control versus other quartiles with higher serum magnesium. We observed also a negative correlation between serum magnesium level and HbA1c ($r = -0.625$, $P < 0.001$). This agreed with Galli and Maggana [17] on Athens type I diabetic children that showed a lower Mg level in patients with poor glycemic control with high HbA1c. Ramadass et al[31], Sinha and Sen[32] showed similar results in adult patients with type II diabetes. Also Mikhail and Ehsanipoor[29] observed an inverse correlation between total Mg and HbA1c.

Inconsistent with our result, Lin et al[21] and Salmonowicz et al[33] did not show any correlation between serum magnesium level and HbA1c in type I diabetic children and adolescents. Also, Mathiesen et al[28] did not observe any relationship between HbA1c and ionized magnesium and Wegner et al[33] failed to show any relation between serum magnesium and diabetic controls in the form of Fasting Blood Glucose. Again this difference between these studies and ours could be attributed to the difference in study populations, degree of diabetic control

Table 6
Multivariate linear regression of potential predictors of serum magnesium level in diabetic patients.

| Variables | Regression coefficient | SE | P |
|-----------|-----------------------|----|---|
| Age, y    | +0.066                | 0.007 | 0.544 |
| Sex, male/female | −0.021 | 0.055 | 0.837 |
| Duration of diabetes | −0.596 | 0.014 | <0.001 |
| Constant  | 2.012                 | – | – |

$\text{SE} = \text{standard error.} \quad \text{P}<0.05 = \text{significant.}$

Figure 2. This figure shows that there is significantly negative correlation between serum magnesium and duration of diabetes.

Table 7
Multivariate linear regression of potential predictors of HbA1c level in diabetic patients.

| Variables | Regression coefficient | SE | P |
|-----------|-----------------------|----|---|
| Age, y    | +0.163                | 0.027 | 0.119 |
| Sex, male/female | +0.009 | 0.205 | 0.926 |
| Magnesium level, mg/dL | −0.438 | 0.466 | <0.001 |
| Duration of diabetes | +0.212 | 0.061 | 0.093 |
| Constant  | 11.582                | 1.078 | – |

$\text{SE} = \text{standard error.} \quad \text{P}<0.05 = \text{significant.}$
among them, also to the different methods of evaluating serum magnesium and glycemic control.

4.3. Correlation of serum magnesium with lipid profile

Our study showed that there was a significant difference between diabetic and control subjects as regard to lipid parameters with higher values of triglycerides (\( P < 0.01 \)), total cholesterol (\( P < 0.001 \)), and LDL (\( P < 0.05 \)), in the diabetic group than control group. However, high-density lipoprotein was lower in the diabetic group than in the control group (\( P < 0.01 \)).

This is in agreement with Wang et al\[20\] and Mishra et al\[35\] on adult population type II diabetes that showed a significant difference in lipid parameters between control and diabetic group with lower HDL and higher other lipid parameters in the diabetic group. Rasheed et al\[8\] study on type II diabetic patients also detected a higher level of TG and lower value of HDL in type 2 diabetic group relative to control group (\( P = 0.03 \)).

This lipid disturbance (higher triglycerides, total cholesterol and LDL with lower HDL) is most pronounced in diabetic children with hypomagnesemia compared to those with normal serum magnesium. A negative correlation is noted between serum Mg and triglycerides (\( r = -0.636, P < 0.001 \)), total cholesterol (\( r = -0.743, P < 0.001 \)), and LDL (\( r = -0.634, P < 0.001 \)). However, there was a positive correlation between serum Mg and HDL (\( r = 0.639, P < 0.001 \)).

These results are in concordance with Mishra et al\[35\] study on adult type II diabetic patients that revealed a negative correlation between serum Mg and triglycerides (\( r = -0.519, P < 0.01 \)) and a positive correlation with HDL (\( r = 0.741 \)), but did not show any significant correlation between total cholesterol or LDL with serum Mg. Moreover, Srinivasasu et al\[36\] revealed a negative correlation between serum Mg and triglycerides (\( P < 0.05 \)) especially in poorly controlled diabetics (\( r = -0.632 \)) and Rasheed et al\[8\] revealed a positive correlation of serum Mg with HDL (\( r = 0.34, P < 0.01 \)), but there was a non-significant correlation with other lipid parameters.

Guerrero-Romero and Rodríguez-Morán\[37\] showed that in patients with T2DM, hypomagnesemia is linked with low levels of HDL, irrespective of serum glucose level. Our results are inconsistent with Wagner et al\[33\] on type I diabetic children that found that there was no significant difference in lipid parameters between patients with normal and those with low serum magnesium level. Also, Jiancheng et al\[20\] failed to show any association between serum magnesium and lipid profile in diabetic patients with or without complications.

In our study, we detected a negative correlation between serum magnesium level in diabetic children with duration of diabetes (\( r = -0.443, P < 0.001 \)). This is consistent with a study on adults with T2DM by Mishra et al\[35\] that showed a negative correlation between the serum magnesium level and duration of diabetes (\( r = -0.789 \)). However, Lin et al\[21\], Salmonowicz et al\[23\] and Sjogren et al\[38\] failed to show any relation between duration of diabetes and serum magnesium level in type I diabetic children. This difference may be due to different study populations and variable degree of diabetic control among these populations.

In our study, there was a significant difference in age between hypomagnesemic and normomagnesemic diabetic children, as mean age of diabetics with low magnesium was (4.82 ± 2.35 years) versus (1.94 ± 1.45 years) in those with normal magnesium level (\( P < 0.001 \)). This is in agreement with Mishra et al\[35\] where they found a positive correlation between age of T2DM adults and serum magnesium level (\( r = 0.721, P < 0.001 \)) but disagreed with Lin et al\[21\] and Salmonowicz et al\[23\] who showed no correlation between age of T1DM children and serum magnesium level. This disagreement could be explained by different study populations between their studies and ours. Wang et al\[34\] showed similar results to Lin et al\[21\] and Salmonowicz et al\[23\] but in type 2 diabetes patients. Jiancheng et al\[20\] stated that in adults with T2DM; older age is seen more frequent among the normomagnesemic group.

5. Conclusion

We conclude that total serum magnesium is frequently low in Egyptian children with type 1 diabetes and it is correlated with glyceric control and with lipid profile. Hypomagnesemia was more evident in patients with poor diabetic control and those with higher atherogenic lipid parameters. We suggest regular monitoring of serum magnesium in children with type 1 diabetes and correcting hypomagnesemia if present. We need to perform further studies on giving magnesium supplements in diabetic children with hypomagnesemia to observe the effect of correction of serum magnesium on glycemic control, lipid profile, and the risk of diabetic complications.

5.1. Study limitations

Several limitations of this study deserve mentioning. First, we did not measure erythrocyte magnesium content. As magnesium is a predominantly intracellular ion, its serum measurements are not representative of magnesium status or intracellular pool. In this regard, significant intracellular magnesium depletion could be seen with normal serum concentrations\[35\]; however, because we already having a higher percentage of hypomagnesaemia in serum of our diabetic patients compared to our control subjects, this potential limitation did not influence our objective and conclusions. Second we did not randomize our patients, instead, we took all patients coming consecutively to our outpatient Endocrinology unit. However, because we have a good number of patients with age and sex matched control, this potential limitation did not influence our objective and conclusions. We recommend performing further multicenter studies on different populations with different levels of glycemic control in order to get more accurate results regarding the relationship between serum and intracellular magnesium in children with type 1 diabetes.

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