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Original Article

Hypomagnesaemia and Relationship Lipid Profile in Type 2 Diabetes Patients at Janzur Hospital in Libya.

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ABSTRACT

Type 2 diabetes mellitus (T2DM) is associated with increased morbidity and mortality due to the development of complications, especially due to poor glycemic control. Magnesium (Mg) is a crucial element in human health, and its deficiency plays a critical role in the development of lipid abnormalities and the initiation of diabetic complications. The study aimed to evaluate the correlation of hypomagnesemia (Mg) with the alteration of lipid profile patterns and investigate the adverse effects of glycemic control and body mass index (BMI) on magnesium levels among patients with T2DM. A total of 163 subjects whose ages ranged between 35-60 years for both sexes (males and females) were included in this study: 90 type II diabetic patients (40 males and 50 females) and 73 healthy individuals (34 males and 39 females) in Tripoli region, Western Libya. Blood glucose, HbA1c, Magnesium, and lipid profile parameters were biochemically estimated in the study along with the measurement of BMI. All the results were statistically analyzed using SPSS version 25 for applying one-way ANOVA and Person's correlation coefficient tests. The study revealed a significant decrease in serum magnesium levels (1.59 ± 0.47 mg/dL) in T2DM patients compared to the control group (2.21 ± 0.70 mg/dL) ($p < 0.01$). Significantly elevated levels of blood glucose (140.6 ± 30.95 mg/dL vs. 89.80 ± 12.62 mg/dL, $p < 0.01$), HbA1c (7.27 ± 2.62% vs. 5.32 ± 0.65%, $p < 0.01$), cholesterol, triglycerides and LDL cholesterol also observed in T2DM patients compared to healthy individuals. Conversely, HDL cholesterol levels were significantly lower in the T2DM group than in controls (24.44 ± 8.88 mg/dL vs. 47.06 ± 8.75 mg/dL, $p < 0.01$). There is a relationship between hypomagnesemia, glycemic control, and changed lipid profile in T2DM patients. Further, our results revealed a strong relationship between increased BMI and unfavorable alterations in all biochemical parameters examined.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a global health concern, affecting over 422 million people worldwide. It is associated with increased morbidity and mortality due to the serious long-term development of complications through mechanisms involving dysregulated glucose and lipid metabolism (1). Magnesium (Mg) plays a vital intracellular role

as a cation, partitioning into three key locations within the body. Approximately 65% of magnesium is stored in the mineral component of bones, with 34% resident in the intracellular space. The remaining 1% is in extracellular fluid (2–4). Mg is required as a cofactor for over 300 biochemical reactions throughout the human body. Specifically, magnesium aids in regulating blood glucose concentrations and maintaining normal blood pressure. Many studies have shown Mg involvement in critical metabolic processes such as energy metabolism and protein synthesis (3,4). Mg plays a crucial role in various physiological processes. It acts as a cofactor for activities such as hormone receptor binding, regulation of calcium channel function, transmembrane ion transport, control of muscle contraction, neuronal signaling, vascular tone, cardiac excitability, and neurotransmitter release (5–7).

Additionally, Mg promotes the body's utilization of other essential minerals and vitamins, including sodium, potassium, calcium, phosphorus, B-complex vitamins, and vitamins C and E (7,8). Intracellular Mg localizes primarily within mitochondria due to its strong binding affinity for ATP (9). As ATP levels are higher in metabolically active cells, intracellular Mg content also tends to be elevated in such cells (9). Together, these functions underline Mg broad importance as a cofactor supporting numerous biochemical pathways critical for homeostasis, energy metabolism, signal transduction, and other vital processes. Hypomagnesaemia, or low serum magnesium levels, is highly prevalent among diabetic patients (10). Mg plays a crucial role in glucose homeostasis and insulin signaling pathways (4,5,10). It also facilitates the breakdown of triglycerides and the modulation of lipid profiles (11,12). Based on previous studies, patients with T2DM are at increased risk of many other diseases, especially macrovascular and microvascular complications (13,14). The prevalence of T2DM is increasing in Libya, yet no studies have assessed the relationship between lipid profiles in T2DM patients and magnesium levels. Therefore, the current study was conducted to evaluate the correlation between hypomagnesemia (low magnesium levels) and alterations in the lipid profile pattern among Libyan patients with T2DM and to investigate the adverse effects of glycemic control and body mass index (BMI) on magnesium levels in these patients.

METHODS

Study population

This case-control study was conducted between September and December 2023 at Janzur Hospital in Tripoli, Libya. A total of 163 subjects aged 35-60 years were recruited, consisting of 73 males and 98 females. The patient group included 90 individuals (40 males and 50 females) with T2DM. The control group comprised 73 healthy individuals (34 males and 39 females), and both groups were matched for age and sex. Written informed consent was obtained from all participants after the study's purpose and procedures were clearly explained (15).

Laboratory analyses

Several laboratory analyses were performed in this study. Glucose levels were measured to assess short-term glycemic control. Glycosylated hemoglobin (HbA1c) was measured to indicate long-term glycemic control over the previous 2-3 months. Serum magnesium (Mg) concentration was quantified to evaluate magnesium status. A lipid profile analysis was conducted to assess cardiovascular disease risk factors, including measurements of total cholesterol, triglycerides, low-density lipoprotein (LDL), and high-density lipoprotein (HDL).

Following an 8-12 hour fast, 5 mL of venous blood was collected from each participant for laboratory analyses. Blood was collected in sodium fluoride tubes to estimate blood glucose (16). Blood was drawn into EDTA tubes (Ethylene Diamine Tetra Acetic acid) to estimate HbA1c (17). Serum was isolated by centrifugation at 4000 rpm for 15 minutes. Body mass index (BMI) was calculated as weight in kilograms divided by height in square meters (18).

A structured questionnaire recorded demographic characteristics, including age, sex, family history, medical history, current medications, occupation, physical activity patterns, and lifestyle habits. Blood glucose was measured using the glucose oxidase-peroxidase (GOD-PAP) enzymatic assay on a Fulfils 4040 photometer. HbA1c was quantified via a sandwich immunoassay method using an Ichroma analyzer. Serum magnesium (Mg) concentrations were determined through a calmagite complexo metric method. Total cholesterol and HDL were measured enzymatically using the cholesterol oxidase assay. On an automated analyzer, triglycerides were assessed via the glycerol phosphate oxidase enzymatic method. The LDL was calculated using the Friedewald formula (LDL cholesterol (mg/dL) = Total cholesterol - HDL cholesterol - (Triglycerides/5).

Inclusion criteria

This study included 90 patients diagnosed with T2DM, aged 35 to 60 years, for both sexes. A control group comprised 73 healthy individuals with no reported history of disease.

Exclusion criteria

The patients who fulfilled any of the following criteria were excluded from the study: diagnosis of type I or gestational T2DM; existence of any chronic illness, such as renal, cardiovascular, or endocrine disorders, that may impact electrolyte balance; history of complications from T2DM; sudden onset of vomiting or diarrhea; current use of drugs or supplements that may affect electrolyte levels, such as thiazide diuretics; or condition of lactation or pregnancy. This stringent selection process aimed to ensure a homogenous study population of type 2 diabetic patients while minimizing the potential influence of confounding factors on electrolyte levels.

Statistical analysis

All data were statistically analysis using SPSS version 25 (Statistical Package for Social Sciences). Descriptive statistics for continuous variables were reported as means and standard deviations. One-way analysis of variance (ANOVA) was employed to compare mean differences between groups. A p-value of less than 0.01 ($p < 0.01$) was considered statistically significant. Pearson's correlation coefficient was calculated to assess the relationship between magnesium levels and the following variables: glucose, HbA1c, cholesterol, triglycerides, LDL, and HDL.

RESULTS

This study included 163 participants aged 35 to 60, comprising males and females. The case group consisted of 90 individuals (40 males, 50 females) diagnosed with T2DM. The control group comprised 73 healthy individuals (34 males, 39 females). Demographic and clinical characteristics of the case and control groups are presented in Table 1. The mean age of participants was comparable between the case (45.54 ± 7.67 years) and control (44.54 ± 7.80 years) groups ($p = 0.4$). On the other hand, the case group's mean BMI was considerably higher (27.49 ± 5.76 kg/m2) than that of the control group (21.35 ± 1.88 kg/m2) ($p < 0.01$). In addition, the case group demonstrated significantly elevated levels of fasting blood glucose (140.6 ± 30.95 mg/dL vs. 89.80 ± 12.62 mg/dL, $p < 0.01$) and HbA1c (7.27 ± 2.62% vs. 5.32 ± 0.65%, $p < 0.01$) compared to the control group. Interestingly, the case group had significantly lower mean serum magnesium levels (1.59 ± 0.47 mg/dL) compared to the control group (2.21 ± 0.70 mg/dL) ($p < 0.01$).

Analysis of lipid profiles revealed significantly higher levels of serum cholesterol (171.9 ± 25.42 mg/dL vs. 141.85 ± 6.09 mg/dL, $p < 0.01$), triglycerides (175.48 ± 25.22 mg/dL vs. 112.73 ± 20.24 mg/dL, $p < 0.01$), and LDL cholesterol (124.38 ± 26.84 mg/dL vs. 82.10 ± 8.85 mg/dL, $p < 0.01$) in the case group compared to the control group. Conversely, HDL cholesterol levels were significantly lower in the case group (24.44 ± 8.88 mg/dL) than in the control group (47.06 ± 8.75 mg/dL) ($p < 0.01$).

Table 1. Demographic, clinical, and biochemical parameters of T2DM patients and healthy controls.

Parameters	Case group Mean±SD	Control group Mean±SD	P. value
Age in years	45.54± 7.67	44.54± 7.80	0.4
BMI kg/m^2	27.49± 5.76	21.35± 1.88	p < 0.01
Fasting blood glucose (mg/dl)	140.6 ±30.95	89.80± 12.62	p < 0.01
HbA1c %	7.27± 2.62	5.32± 0.65	p < 0.01
Mg mg/dl	1.59± 0.47	2.21± 0.70	p < 0.01
Serum cholesterol (mg/dl)	171.9± 25.42	141.85± 6.09	p < 0.01
Triglyceride (mg/dl)	175.48± 25.22	112.73± 20.24	p < 0.01
HDL-Cholesterol(mg/dl)	24.44± 8.88	47.06± 8.75	p < 0.01
LDL-Cholesterol (mg/dl)	124.38± 26.84	82.10± 8.85	p < 0.01

The results were presented as mean ± standard deviation (SD). A one-way analysis of variance (ANOVA) was performed using SPSS version 25 to compare the differences in the means of the biochemical parameters of the patients and control groups. A p-value < 0.01 was considered statistically significant.

The means and standard deviations of different biochemical variables regarding BMI levels are given in Table 2. Based on their BMI, participants were stratified as either healthy weight (BMI 19–24.9 kg/m²), (BMI 25–29.9 kg/m²), and obese (BMI ≥ 30 kg/m²). The mean age was not statistically different between the BMI groups ($p = 0.41$). As shown in Table 2, our rigorous one-way ANOVA analysis has revealed statistically significant differences among the three BMI groups for several biochemical variables.

The increasing trends of blood glucose ($p = 0.07$) and HbA1c ($p < 0.01$) with higher BMI categories, along with the significant rise in serum cholesterol ($p < 0.01$), triglycerides ($p < 0.01$), and LDL cholesterol ($p < 0.01$) as BMI increased.

Equally significant is the revelation that mean magnesium levels ($p < 0.01$) and HDL cholesterol ($p < 0.01$) were notably lower in the obese group compared to the healthy weight and overweight groups.

Table 2. Distribution of biochemical variables in the study group according to BMI.

Parameters	BMI Kg			P .value
	Healthy weight n= 30 19- 24.9 kg/m2 22.19±0.98	Overweight n= 28 25-30 kg/m2 27.07±0.98	Obese n= 32 > 30 kg/m2 34.73±3.22	
Age years	48.53± 7.56	42.77± 7.03	43.82± 7.22	P= 4.41
Blood glucose (mg/dl)	119.28±17.89	138.49± 19.54	170.05±28.29	p = 0.07
HbA1c %	5.22± 1.12	7.56± 1.92	9.73± 2.29	p < 0.01
Mg mg/dl	1.97± 0.43	1.42± 0.28	1.23± 0.24	
Serum cholesterol (mg/dl)	150.04± 3.75	166.11± 9.15	204.93± 12.40	p < 0.01
Triglyceride (mg/dl)	153.76± 2.64	169.35± 10.78	208.59± 10.23	p < 0.01
HDL- cholesterol (mg/dl)	32.31± 5.16	24± 3.67	14.54± 4.31	p < 0.01
LDL- cholesterol (mg/dl)	101.43± 4.19	117.38± 9.22	159.81± 11.51	p < 0.01

One-way analysis of variance (ANOVA) was conducted to assess differences in biochemical parameters among the three BMI groups. A p-value of less than 0.01 ($p < 0.01$) was considered statistically significant

In this study, the Pearson correlation analysis revealed significant associations between serum magnesium levels, glycemic control parameters, and lipid profiles. As illustrated in Table 3, serum magnesium levels were negatively correlated with both blood glucose (r = -0.5) and HbA1c (r = -0.4). As mentioned above, the correlation results demonstrate moderate negative correlations between serum magnesium and several key lipid parameters. Specifically, lower magnesium levels were associated with higher levels of serum cholesterol (r = -0.6), triglycerides (r = -0.6), and LDL-cholesterol (r = -0.6). Conversely, a moderate positive correlation (r = 0.6) was observed between serum magnesium and HDL -cholesterol, as shown in table 4.

Table 3. Correlation Between Serum magnesium status with serum glucose and HbA1c levels.

Parameters	Blood glucose mg/dl	HbA1c%
Serum magnesium mg/dl	-0.5	-0.4

Pearson correlation coefficients (r) were presented between serum magnesium and glucose HbA1c.

Table 4. Correlation Between Serum magnesium status with lipid profile parameters.

Parameters	Serum cholesterol (mg/dl)	Triglyceride (mg/dl)	HDL- cholesterol (mg/dl)	LDL- cholesterol (mg/dl)
Serum magnesium mg/dl	-0.6	-0.6	0.6	-0.6

Data represent Pearson correlation coefficients (r) assessing the relationships between serum magnesium and lipid profile parameters.

DISCUSSION

The study aims to investigate the relationship between serum magnesium levels, glycemic control, and lipid profiles in individuals with type II diabetes compared to healthy controls in Tripoli, Libya. This study investigated 163 individuals aged 35-60, categorized into two groups. The case group comprised 90 individuals diagnosed with Type II diabetes. Conversely, the control group included 73 healthy individuals.

Magnesium is crucial for human health because it involves many cell processes, such as energy homeostasis, protein synthesis, and DNA integrity (7,9). Our results reveal significantly lower serum magnesium levels in the diabetic group compared to the healthy group; this finding is consistent with previous research highlighting magnesium deficiency as a frequent comorbidity in individuals with type II diabetes (10,19,20). This deficiency is often attributed to increased urinary magnesium excretion due to osmotic diuresis, which causes high renal excretion of magnesium (7,9–11), a phenomenon linked to hyperglycemia-induced insensitivity to insulin affecting intracellular magnesium transport and thereby causing increased loss of the extracellular magnesium (19,21). Additionally, our study's lower incidence of hypomagnesemia could be attributed to the stricter exclusion criteria followed in the research and to the region's dietary

habits. Based on the literature review, there is a relationship between hypomagnesemia and cardiovascular disease. Low magnesium levels may be an independent risk factor for various cardiovascular problems, such as heart failure (HF), atrial fibrillation (AF), and microvascular disease in T2D (22).

Our results showed significantly elevated fasting blood glucose and HbA1c levels in the case group compared to the control group. These results are in good agreement with other studies, which have shown significantly higher levels of these glycemic control parameters in individuals with T2DM compared to healthy controls (23,24). The elevated fasting blood glucose and HbA1c levels in the diabetic group can be interpreted as consequences of a complex interplay of insulin resistance, declining insulin secretion, and increased hepatic glucose production (9,10,21). Dyslipidemia is an abnormal increase in lipids in the bloodstream and is a common issue in people with type 2 diabetes (13,14). In our study, we observed dyslipidemia in the diabetic group, characterized by high levels of serum cholesterol, triglycerides, and LDL-cholesterol, along with reduced levels of HDL-cholesterol. This finding aligns with numerous studies showing that dyslipidemia is associated with an elevated risk of cardiovascular disease, particularly in individuals with type 2 diabetes (7,13,14,25). Our research revealed a clear connection between higher BMI and unfavorable metabolic health-related biochemical parameter changes.

Furthermore, the findings indicate that this correlation is not limited to weight gain but shows a consistent pattern where higher BMI categories correspond to more pronounced changes in biochemical markers. Our correlation analyses revealed significant associations between serum magnesium levels and lipid profiles. Lower magnesium levels were associated with less favorable lipid profiles, characterized by higher serum cholesterol levels, triglycerides, and LDL cholesterol and lower HDL cholesterol levels. Other studies support these findings and show a similar relationship between magnesium levels and cholesterol (25,26). On the other hand, the association between obesity and metabolic dysfunction is the notable escalation of serum lipid parameters across BMI groups. Our analysis reveals statistically significant elevations in total cholesterol ($p<0.01$), triglycerides ($p<0.01$), and LDL cholesterol ($p<0.01$) in tandem with increasing BMI. This pattern aligns with previous research demonstrating that adipose tissue, particularly visceral fat, significantly promotes dyslipidemia by influencing lipid metabolism and increasing circulating free fatty acids (12,27,28). Several mechanisms are proposed to explain magnesium's potential protective effects on lipid metabolism. Magnesium modulates key cholesterol synthesis and clearance enzymes, influencing overall cholesterol homeostasis (29,30). Magnesium deficiency, often associated with insulin resistance and chronic inflammation, is increasingly recognized as a contributing factor in developing metabolic dysfunction (9,31).

Similarly, the decline in HDL cholesterol, responsible for reverse cholesterol transport, further exacerbates the cardiovascular risk associated with obesity (32). Additionally, magnesium may enhance insulin sensitivity, indirectly impacting lipid metabolism by improving glucose uptake and utilization, thereby reducing the need for de novo lipogenesis (33). Our correlation study showed significant inverse relationships between serum magnesium, blood glucose ($r = -0.5$), and HbA1c ($r = -0.4$). These findings suggest that magnesium may play a role in regulating blood sugar levels. These results are consistent with several studies that have demonstrated the positive effects of magnesium supplementation on insulin sensitivity and blood sugar control in individuals with type II diabetes (34–37).

CONCLUSION

This study found a relationship between hypomagnesemia, glycaemic control, and changed lipid profile in T2DM patients in Janzur, Libya. Furthermore, our findings revealed a strong relationship between increased BMI and unfavorable alterations in all biochemical parameters examined. These findings underscore the necessity of monitoring serum magnesium levels in individuals with T2DM and the potential benefits of magnesium supplementation as adjuvant therapy for glycaemic management and lipid profile improvement.

Conflicts of Interest

There are no financial, personal, or professional conflicts of interest to declare.

REFERENCES

1. WHO. Discussion group for people living with diabetes. 2023.
2. Masood N, Baloch GH, Ghori RA, Memon IA, Memon MA, Memon MS. Serum zinc and magnesium in type-2 diabetic patients. J Coll Physicians Surg Pak. 2009;19(8):483–6.
3. Meludu SC, Adeniyi FAA. Effect of magnesium supplementation on plasma glucose in patients with diabetes mellitus. African J Biomed Res. 2001;4(3).
4. Hans CP, Sialy R, Bansal DD. Magnesium deficiency and diabetes mellitus. Curr Sci. 2002;1456–63.
5. Barbagallo M, Dominguez LJ. Magnesium and type 2 diabetes. World J Diabetes. 2015;6(10):1152.
6. Eby G, Eby K. Rapid recovery from major depression using magnesium treatment. Med Hypotheses. 2006;67(2):362–70.

7. De Baaij JHF, Hoenderop JGJ, Bindels RJM. Magnesium in man: implications for health and disease. Physiol Rev. 2015;
8. Giapros VI, Schiza V, Challa AS, Cholevas VK, Theocharis PD, Kolios G, et al. Vitamin D and parathormone levels of late-preterm formula fed infants during the first year of life. Eur J Clin Nutr. 2012;66(2):224–30.
9. Barbagallo M, Dominguez LJ. Magnesium metabolism in type 2 diabetes mellitus, metabolic syndrome and insulin resistance. Arch Biochem Biophys. 2007;458(1):40–7.
10. Shaikh MK, Devrajani BR, Soomro AA, Ali Shah SZ, Devrajani T, Das T. Hypomagnesemia in Patients with Diabetes mellitus. World Appl Sci J. 2011;12(10):1803–6.
11. Chaudhary DP, Sharma R, Bansal DD. Implications of magnesium deficiency in type 2 diabetes: A review. Biol Trace Elem Res. 2010;134(2):119–29.
12. Guerrero-Romero F, Rodríguez-Morán M. Hypomagnesemia, oxidative stress, inflammation, and metabolic syndrome. Diabetes Metab Res Rev. 2006;22(6):471–6.
13. Hasona N, Elasbali A. Evaluation of Electrolytes Imbalance and Dyslipidemia in Diabetic Patients. Med Sci. 2016;4(2):7.
14. Mishra S, Padmanaban P, Deepti GN, Sarkar G, Sumathi S, Toora BD. Serum magnesium and dyslipidemia in type-2 diabetes mellitus. Biomed Res. 2012;23(2):295–300.
15. Shrestha B, Dunn L. The declaration of Helsinki on medical research involving human subjects: a review of seventh revision. J Nepal Health Res Counc. 2019;17(4):548–52.
16. Wilson ML. General principles of specimen collection and transport. Clin Infect Dis. 1996;22(5):766–77.
17. Sacks DB, Arnold M, Bakris GL, Bruns DE, Horvath AR, Kirkman MS, et al. Guidelines and recommendations for laboratory analysis in the diagnosis and management of diabetes mellitus. Clin Chem. 2011;57(6):e1–47.
18. Organization WH. Obesity: preventing and managing the global epidemic. 2000. WHO Tech Rep Ser. 2017;894.
19. Pham P-CT, Pham P-MT, Pham S V, Miller JM, Pham P-TT. Hypomagnesemia in patients with type 2 diabetes. Clin J Am Soc Nephrol. 2007;2(2):366–73.
20. Kurstjens S, de Baaij JHF, Bouras H, Bindels RJM, Tack CJJ, Hoenderop JGJ. Determinants of hypomagnesemia in patients with type 2 diabetes mellitus. Eur J Endocrinol. 2017;176(1):11–9.
21. Dasgupta A, Sarma D, Saikia U. Hypomagnesemia in type 2 diabetes mellitus. Indian J Endocrinol Metab. 2012;16(6):1000.
22. Oost LJ, Tack CJ, de Baaij JHF. Hypomagnesemia and Cardiovascular Risk in Type 2 Diabetes. Endocr Rev [Internet]. 2023;44(3):357–78. Available from: https://doi.org/10.1210/endrev/bnac028
23. Manonmani M, Manimekalai K. A Study of Serum Magnesium Level in Type 2 Diabetes Mellitus Patients. J Diabetes Mellit. 2018;08(02):20–6.
24. Palomino-Schätzlein M, Lamas-Domingo R, Ciudin A, Gutiérrez-Carcedo P, Marés R, Aparicio-Gómez C, et al. A translational in vivo and in vitro metabolomic study reveals altered metabolic pathways in red blood cells of type 2 diabetes. J Clin Med. 2020;9(6):1–18.
25. Sarwar N, Gao P, Kondapally Seshasai SR, Gobin R, Kaptoge S, Di Angelantonio E, et al. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: A collaborative meta-analysis of 102 prospective studies. Lancet [Internet]. 2010;375(9733):2215–22. Available from: http://dx.doi.org/10.1016/S0140-6736(10)60484-9
26. Sadeghian M, Azadbakht L, Khalili N, Mortazavi M, Esmaillzadeh A. Oral magnesium supplementation improved lipid profile but increased insulin resistance in patients with diabetic nephropathy: a double-blind randomized controlled clinical trial. Biol Trace Elem Res. 2020;193:23–35.
27. Bergman RN, Ader M. Free fatty acids and pathogenesis of type 2 diabetes mellitus. Trends Endocrinol Metab. 2000;11(9):351–6.
28. Balducci, Stefano, Sacchetti, Massimo, Haxhi, Jonida, Orlando, Giorgio, D'Errico, Valeria, Fallucca, Sara, Menini, Stefano, Pugliese G. Physical Exercise as therapy for type II diabetes. Diabetes Metab Res Rev [Internet]. 2014;32(30):13–23. Available from: http://libweb.anglia.ac.uk/
29. Tangvoraphonkchai K, Davenport A. Magnesium and cardiovascular disease. Adv Chronic Kidney Dis. 2018;25(3):251–60.
30. Kolte D, Vijayaraghavan K, Khera S, Sica DA, Frishman WH. Role of magnesium in cardiovascular diseases. Cardiol Rev. 2014;22(4):182–92.
31. Guerrero-Romero F, Rodríguez-Morán M. Magnesium and the metabolic syndrome. In: Magnesium. CRC Press; 2019. p. 57–66.
32. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease: the Framingham Study. Am J Med. 1977;62(5):707–14.
33. Barbagallo M, Dominguez LJ, Galioto A, Ferlisi A, Cani C, Malfa L, et al. Role of magnesium in insulin action, diabetes and cardio-metabolic syndrome X. Mol Aspects Med. 2003;24(1–3):39–52.
34. Veronese N, Dominguez LJ, Pizzol D, Demurtas J, Smith L, Barbagallo M. Oral magnesium supplementation for treating glucose metabolism parameters in people with or at risk of diabetes: a systematic review and meta-analysis of double-blind randomized controlled trials. Nutrients. 2021;13(11):4074.
35. Asbaghi O, Moradi S, Kashkooli S, Zobeiri M, Nezamoleslami S, Kermani MAH, et al. The effects of oral magnesium supplementation on glycaemic control in patients with type 2 diabetes: a systematic review and dose–response meta-analysis of controlled clinical trials. Br J Nutr. 2022;128(12):2363–72.

36. Veronese N, Watutantrige-Fernando S, Luchini C, Solmi M, Sartore G, Sergi G, et al. Effect of magnesium supplementation on glucose metabolism in people with or at risk of diabetes: a systematic review and meta-analysis of double-blind randomized controlled trials. Eur J Clin Nutr. 2016;70(12):1354–9.
37. Guerrero-Romero F, Tamez-Perez HE, Gonzalez-Gonzalez G eal, Salinas-Martinez AM, Montes-Villarreal J, Trevino-Ortiz JH, et al. Oral magnesium supplementation improves insulin sensitivity in non-diabetic subjects with insulin resistance. A double-blind placebo-controlled randomized trial. Diabetes Metab. 2004;30(3):253–8.

نقص الماغنيسيوم وعلاقته بأنماط الدهون لدى مرضى السكري من النوع الثاني في مستشفى جنزور في ليبيا

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المستخلص

يرتبط مرض السكري من النوع الثاني بزيادة معدلات الإصابة والوفيات بسبب تطور المضاعفات، وخاصة بسبب ضعف التحكم في نسبة السكر في الدم. يعد المغنيسيوم عنصرًا حاسمًا في صحة الإنسان، ويلعب نقصه دورًا حاسمًا في تطور اضطرابات الدهون وبدء المضاعفات السكرية. هدفت الدراسة إلى تقييم ارتباط نقص المغنيسيوم في الدم بتغيير أنماط الدهون والتحقيق في الآثار الضارة للتحكم في نسبة السكر في الدم ومؤشر كتلة الجسم على مستويات المغنيسيوم بين مرضى السكري من النوع الثاني. تم تضمين ما مجموعه 163 شخصًا تتراوح أعمارهم بين 35-60 عامًا لكلا الجنسين (ذكور وإناث) في هذه الدراسة: 90 مريضًا مصابًا بمرض السكري من النوع الثاني (40 ذكرًا و50 أنثى) و73 فردًا سليمًا (34 ذكرًا و39 أنثى) في منطقة طرابلس، غرب ليبيا. تم تقدير مستويات الجلوكوز في الدم، والهيموجلوبين السكري، والمغنيسيوم، ومستويات الدهون كيميائيًا في الدراسة جنبًا إلى جنب مع قياس مؤشر كتلة الجسم. تم تحليل جميع النتائج إحصائيًا باستخدام برنامج SPSS الإصدار 25 لتطبيق اختبارات تحليل التباين أحادي الاتجاه ومعامل ارتباط بيرسون. كشفت الدراسة عن انخفاض كبير في مستويات المغنيسيوم في المصل (1.59 ± 0.47 مجم / ديسيلتر) في مرضى السكري من النوع 2 مقارنة بمجموعة التحكم (2.21 ± 0.70 مجم / ديسيلتر). كما لوحظت مستويات مرتفعة بشكل ملحوظ من جلوكوز الدم (140.6 ± 30.95 مجم / ديسيلتر مقابل 89.80 ± 12.62 مجم / ديسيلتر، ص < 0.01)، والهيموجلوبين السكري (7.27 ± 2.62٪ مقابل 5.32 ± 0.65٪، ص < 0.01)، والكوليسترول، والدهون الثلاثية وكوليسترول البروتين الدهني منخفض الكثافة في مرضى السكري من النوع 2 مقارنة بالأفراد الأصحاء. وعلى العكس من ذلك، كانت مستويات الكوليسترول الحميد أقل بشكل ملحوظ في مجموعة السكري من النوع 2 مقارنة بمجموعة التحكم (24.44 ± 8.88 مجم / ديسيلتر مقابل 47.06 ± 8.75 مجم / ديسيلتر، ص < 0.01). هناك علاقة بين نقص المغنيسيوم، والسيطرة على نسبة السكر في الدم، وتغير انماط الدهون في مرضى السكري من النوع 2. علاوة على ذلك، كشفت نتائجنا عن وجود علاقة قوية بين زيادة مؤشر كتلة الجسم والتغيرات السلبية في جميع المعايير الكيميائية الحيوية التي تم فحصها.

الكلمات المفتاحية. نقص الماغنيسيوم في الدم، داء السكري من النوع الثاني، مؤشر كتلة الجسم، انماط الدهون، نسبة السكر في الدم.