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Bioscience Research

Print ISSN: 1811-9506 Online ISSN: 2218-3973

Journal by Innovative Scientific Information & Services Network



RESEARCH ARTICLE

BIOSCIENCE RESEARCH, 2020 17(1): 510-514.

OPEN ACCESS

Association among anthropometric measurements, Magnesium status, lipids profile and insulin resistance in offspring of diabetic patients

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The risk for type 2 diabetes among offspring with a single diabetic parent was 3.5-fold higher and for those with two diabetic parents was 6-fold higher compared with offspring without parental diabetes. The study designed to evaluate the changes in serum level of magnesium, triglyceride, and cholesterol as well as, fat mass, fat percentage and body mass index in healthy offspring of diabetic parents as early predictors for development of diabetes. Twenty-eight children with their age range 5-12 years, eighteen of them were offspring of diabetic parents and ten sex and age matched offspring of normal parents as controls. Serum level of magnesium, cholesterol and triglyceride were measured by colorimetric enzymatic method. Calculation of body mass index, fat mass and fat percent were done using body composition analyzer BC-418. There was non-significant difference in serum levels of magnesium, cholesterol and triglyceride of offspring of diabetic parents when compared with control group. In addition, there was non-significant difference regarding BMI, fat mass and fat percentage between the studied groups. Negative non-significant correlation between magnesium and each of T.G, BMI and fat mass in offspring of diabetic parents was found. The negative non-significant correlation between magnesium and each of T.G, BMI and fat mass could highlight their possible significant role in pathogenesis of any future metabolic abnormality in offspring of diabetic parents.

Keywords: Magnesium, Lipids and Diabetes

INTRODUCTION

The World Health Organization (WHO) defines obesity as a condition of excessive fat accumulation to the extent that health and well-being are affected (Fernandez-Real et al., 2001).

WHO classified the weight into underweight (Thin) where BMI (body mass index= $\text{weight (Kg) / height (m}^2\text{)}$) <18.5 (but risk of other clinical problems increased); normal weight diagnosed when BMI is between 19-24.9 kg/m^2 ; while in overweight (pre-obese) BMI is 25-29.9 kg/m^2 ; if BMI lies between 30-35 kg/m^2 it indicates obesity class I (moderate); obesity class II (severe)

is defined when BMI from 35.0 kg/m^2 to 39.9 kg/m^2 ; lastly obesity class III (very severe) if BMI exceed or equal 40 kg/m^2 (Kershaw and Fleur, 2004).

Demographic projections for the next 30 y coincide in placing the burden of population growth primarily on the developing world, and most of this growth will be in urban areas. One can speculate that this may result from a combination of increased energy intake, decreased energy expenditure and, perhaps, reduced gastrointestinal nutrient losses. Also lack of adequate longitudinal data on patterns of physical activity in the developing world precludes an estimation of

trends. There is some evidence that a sedentary lifestyle is common among low-income urban dwellers. (Torun, 2000).

Adipose tissue is a complex and, highly active metabolic and endocrine organ. Adipose tissue is now known to express and secrete a variety of bioactive peptides, known as adipokines, which act at both the local (autocrine/paracrine) and systemic (endocrine) level (Kershaw and Fleir, 2004).

Leptin's primary role is to serve as a metabolic signal of energy sufficiency rather than excess. Common forms of obesity are characterized by elevated circulating leptin. Neither endogenously high leptin levels nor treatment with exogenous leptin is effective in ameliorating this obesity, consistent with a state of leptin resistance (Bjorbeak and Khan, 2004).

The mechanism for leptin resistance is unknown but may result from defects in leptin signaling or transport across the blood-brain barrier. In addition to its effects on energy homeostasis, leptin regulates neuroendocrine function and traditional endocrine systems (Bjorbeak and Khan, 2004).

In the liver, adiponectin enhances insulin sensitivity, decreases influx of NEFAs, increases fatty acid oxidation, and reduces hepatic glucose output (Chandran, 2003).

In muscle, adiponectin stimulates glucose use and fatty acid oxidation. Within the vascular wall, adiponectin inhibits monocyte adhesion by decreasing expression of adhesion molecules, inhibits macrophage transformation to foam cells by inhibiting expression of scavenger receptors, and decreases proliferation of migrating smooth muscle cells in response to growth factors (Yamauchi, 2003).

In addition, adiponectin increases nitric oxide production in endothelial cells and stimulate angiogenesis. These effects are mediated via increased phosphorylation of the insulin receptor, activation of AMP-activated protein kinase, and modulation of the nuclear factor κ B pathway. Taken together, these studies suggest that adiponectin is a unique adipocyte-derived hormone with antidiabetic, anti-inflammatory, and antiatherogenic effects (Diez and Iglesias, 2003).

Magnesium is an important element for plant and animal life. Chlorophylls are porphyrins based upon magnesium. The adult human daily requirement of magnesium is about 0.3 g day⁻¹. It is the fourth most abundant mineral in the body and is essential to good health. Approximately 50% of total body magnesium is found in bone. The other half is found predominantly inside cells of body

tissues and organs. Only 1% of magnesium is found in blood, but the body works very hard to keep blood levels of magnesium constant (Steppan, 2001).

Furthermore, large epidemiologic studies in adults indicate that lower dietary magnesium and lower serum magnesium are associated with increased risk for type 2 diabetes (Lopez-Ridaura et al. 2004).

Several clinical studies have examined the potential benefit of supplemental magnesium on metabolic control of type 2 diabetes. In one such study, 63 subjects with below normal serum magnesium levels received either 2.5 grams of oral magnesium chloride daily "in liquid form" (providing 300 mg elemental magnesium per day) or a placebo. At the end of the 16-week study period, those who received the magnesium supplement had higher blood levels of magnesium and improved metabolic control of diabetes, as suggested by lower glycosylated hemoglobin levels, than those who received a placebo (Moran and Guerrero-Romero, 2003).

MATERIALS AND METHODS

This study was carried out in period from 1439 to 1440 at the Medical Biochemistry Department, Faculty of Applied Medical Sciences, Umm al-Qura University and Pediatric Department Maternity and children hospital (Maternity and children hospital).

Twenty-eight children with their ages between 5 –12 years and of the same socioeconomic level were included in the study and divided into two groups.

Group I: Included ten children offspring of normal parent, nine of them were of normal average weight, the last was overweight, they represent control group.

Group II: Included eighteen children offspring of diabetic parent, their weight were within normal range.

Method:

All children of the study were subjected to:

I- Full history taking and clinical examination.

II- Calculation of:

1 Body mass index

2 Fat percentage

3 Fat mass

By using BODY COMPOSITION ANALYZER BC-418

III-Measurement of serum magnesium, cholesterol, and triglycerides by colorimetric enzymatic method using Liqui-Stat reagent and analyzer.

Blood sampling:

5 ml of peripheral venous fasting blood samples were withdrawn from each child under complete aseptic condition, collected in dematerialized plane tube, left for 30-60 minutes for clotting and centrifuged at 3000 rpm for 10 minutes. Serum samples were separated into another set of dematerialized tube and kept frozen at -20C until the time for assay for each sample.

RESULTS

Statistical analysis :Result are expressed in . All analysis were performed with SPSS statistical package (version 16.0). comparison between children of diabetic parents data and children of non-diabetic parents was assessed by using t – test . Statistical correlation were assessed using a partial correlation test (spearman correlation)

Table (1): Serum magnesium, cholesterol, T.G, BMI, fat percent and fat mass percent in group I and group II:

| Parameters | Group I | Group II | P |
|---------------------|--------------|---------------|-------|
| Magnesium (mg/dl) | 1.45 ± 0.36 | 1.29 ± 0.42 | 0.701 |
| Cholesterol (mg/dl) | 180.2 ± 22.0 | 190.6 ± 36.61 | 0.450 |
| T.G (mg/dl) | 72.5 ± 49.0 | 66.53 ± 3.04 | 0.526 |

Significant: p < 0.0 5%

Table (2): BMI, fat percent and fat mass percent in group I and group II:

| Parameters | Group I | Group II | P |
|-------------|--------------|--------------|-------|
| BMI | 19.16 ± 3.46 | 18.34 ± 3.84 | 0.904 |
| Fat percent | 17.24 ± 3.75 | 20.05 ± 5.82 | 0.258 |
| Fat mass | 3.32 ± 1.53 | 3.66 ± 2.04 | 0.253 |

Significant: p < 0.0 5%

Table (3): Pearson correlation between magnesium and all measured parameter in all children:

| Parameters | Pearson Correlation (r) | Sig. (2-tailed) |
|-------------------------|-------------------------|-----------------|
| Magnesium &T.G | -.126 | 0.506 |
| Magnesium & cholesterol | 0.033 | 0.863 |
| Magnesium & BMI | 0.436 | 0.016 |
| Magnesium & Fat percent | 0.300 | 0.108 |
| Magnesium & fat mass | 0.454 | 0.012 |

Significant: p < 0.0 5%

DISCUSSION

The rapid global rise in the prevalence of type 2 diabetes constitutes a health threat to the individual and is a major burden for health economy (Gillman, 2005).

Table (4): Pearson correlation between magnesium and studied parameter in children of diabetic parents:

| Parameters | Pearson correlation | Sig. (2-tailed) |
|-------------------------|---------------------|-----------------|
| Magnesium &T.G | -.223 | .373 |
| Magnesium &cholesterol | .280 | .260 |
| Magnesium & BMI | -.404 | .097 |
| Magnesium & Fat percent | .140 | .578 |
| Magnesium & fat mass | -.276 | .268 |

Significant: p < 0.0 5%

Meigs et al. (2000) have been found that risk for type 2 diabetes among offspring with a single diabetic parent was 3.5-fold higher and for those with two diabetic parents was 6-fold higher compared with offspring without parental diabetes.

Moreover, obesity beginning in childhood often precedes and plays central role in the insulin resistance syndrome, which includes hyperinsulinemia, hypertension, hyperlipidemia, type 2 diabetes mellitus, and an increased risk of atherosclerotic cardiovascular disease (Huerta et al.2005).

Therefore, it is crucial to identify offspring of diabetic parents as specific risk group targeting for preventive measures.

So, we aimed in this study to determine the changes in serum level of magnesium, triglyceride, and cholesterol as well as, fat mass percent, fat percentage and body mass index in healthy offspring of diabetic parents as early predictors for development of diabetes in those children.

We studied twenty eight children, eighteen of them were offspring of diabetic parents and the rest of children were offspring of normal parents.

There was non-significant difference between serum magnesium level in offspring of diabetic parents and those of healthy parents, which is in accordance with that reported by (Maltezos et al. 2004).

Reduced magnesium absorption and decreased renal tubular reabsorption could also lead to magnesium deficiency (Huerta et al. 2005).

Also, Maltezos et al. (2004) have found no relationship between total serum magnesium and the main indicators of glucose homeostasis in offspring of type 2 diabetic mother.

In addition, there was non-significant difference of serum triglyceride and cholesterol levels between offspring of diabetic parents and offspring of healthy parents. This is in agreement with (Jaskolska-Ladosz et al.2000).

On the other hand significant changes in LDL-

cholesterol, HDL- cholesterol and Apo-A1 between the same studied groups have been reported by Jaskolska-Ladosz et al. (2000) and explained by reduced influence of insulin on the enzymes which control their metabolism.

There was non-significant difference in BMI, fat mass and fat percentage between the studied groups as reported by (Kostalova et al. 2001).

On the other hand Shahid et al. (2008) have reported higher BMI and body weight in offspring of diabetic parents older than nineteen years.

Therefore, an increased BMI and body weight might be a long term consequence of maternal diabetes as reported by (Kostalova et al.2001).

A common variant in the FTO (fat, mass and obesity) gene has been identified that predisposes to diabetes through an effect on the BMI (Frayling et al. 2007).

Our results similar to Sinha S & Sen S, Lin CC and Ramadass et al as type I diabetic children that showed a lower Mg level in patients with high HbA1c.

CONCLUSION

Our study conclude that there was non-significant difference regarding BMI, fat mass and fat percentage between the studied groups. Negative non-significant correlation between magnesium and each of T.G, BMI and fat mass in offspring of diabetic parents was found. The negative non-significant correlation between magnesium and each of T.G, BMI and fat mass could highlight their possible significant role in pathogenesis of any future metabolic abnormality in offspring of diabetic parents.

CONFLICT OF INTEREST

The authors declared that present study was performed in absence of any conflict of interest.

AUTHOR CONTRIBUTIONS

All authors (Naser A. ElSawy, Reham A. Mostafa and Doaa R. Negm) designed, collect samples, performed the experiments, data analysis, wrote and reviewed the manuscript, work equally. All authors read and approved the final version.

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