Serum magnesium levels in relation to metabolic syndrome: A national population-based cross-sectional study from Jordan

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Abstract

Background/Aims:

Individuals with metabolic syndrome are at higher risk to develop cardiovascular disease, diabetes type 2, and certain types of cancers such as pancreatic, liver, and colorectal cancers. Studies suggest a potential association between hypomagnesemia and metabolic syndrome with conflicting results. The present study aims to estimate the prevalence of metabolic syndrome and its components in Jordan and to further explore the association between low magnesium levels and metabolic syndrome and its components.

Methods

Data were derived from the national population-based household survey conducted in Jordan in 2009. The study was multipurpose and collected a wide array of data including interview data, anthropometric measurements, and laboratory data. The present report deals, exclusively, with subjects aged ≥ 20 years. There was a total of 4,520 subjects (1113 men and 3407 women).

Results

We found alarmingly high prevalence rates of metabolic syndrome and its components defined by IDF (international diabetic federation) criteria (39.8%) with the highest burden being among women (40.7% in women and 37.4% in men). Among metabolic syndrome components, central obesity and low HDL were the most commonly occurring components of metabolic syndrome (62.6% and 65.5%, respectively). We observed a significant inverse association between serum magnesium levels and metabolic syndrome after adjusting for age (OR = 1.57, p value = 0.048). Among metabolic syndrome components, low serum magnesium levels were significantly associated with low serum HDL levels (OR = 2.15, p value = 0.001).

Conclusion

Metabolic syndrome prevalence in Jordan is considerably high. Our findings suggest that serum magnesium levels are inversely associated with metabolic syndrome.

1. Introduction

The epidemic growth of non-communicable diseases (NCDs) leading to an increased risk of morbidity and mortality constitutes a major global health challenge. The Global Burden of Disease 2016 study (GBD2016) showed that death from non-communicable diseases represented 72.3% of deaths in 2016 (1). Adoption of an unhealthy lifestyle in the form of physical inactivity, unhealthy diet, and tobacco use has led to the increasing burden of NCDs (2). Metabolic risk factors, on the other hand, manifested by

raised blood pressure, central obesity, hyperglycemia, and hyperlipidemia comprising what is known as metabolic syndrome are incriminated in the rising incidence of NCDs (3).

The worldwide prevalence of metabolic syndrome is on the rise and it has become an essential public health problem among numerous developed and developing counties. The prevalence of metabolic syndrome varies between 10 to 85% according to the definition used, study design, sample selection, ethnicity, and the age and gender structure of the population. The IDF, on the other hand, estimates that one-quarter of the world's population has metabolic syndrome (4). Metabolic syndrome is a multifactorial condition that stems from the interactions between metabolic, environmental, and genetic factors (5). Reducing the burden of metabolic syndrome is of great importance for the prevention of major non-communicable diseases, such as cardiovascular disease and diabetes type 2. Evidence has shown that individual metabolic syndrome components were independent risk factors for cardiovascular disease (6), and their combination augments the rates and severity of its development producing a spectrum of vascular and cardiac diseases.

Magnesium is an essential mineral for the human body being the forth abundant cation after calcium, potassium, and sodium and the second most abundant intracellular cation after potassium (7). It has several vital functions, namely muscle and cardiac contraction, neuromuscular conduction, and glycemic control. Moreover, magnesium is regarded as a cofactor for more than 300 enzymes. In addition, it plays a fundamental role in energy production, active transmembrane transport of ions, synthesis of nuclear material, and bone development (8). Magnesium hemostasis is maintained by the balance between intake, absorption, and excretion which is regulated by the intestines, the bones, and the kidneys (9). Approximately 60% of the total body magnesium is found in bone and 20% is localized to muscles, while 20% is found in other tissues of the body. Only 1% is present in the extracellular fluid compartments, of which 0.3% is in the serum (10).

Several non-communicable diseases have been associated with alterations in the levels of some minerals, among them low magnesium levels. Evidence has suggested that magnesium depletion may play an essential role in the pathophysiology of diabetes mellitus, hypertension, obesity, and dyslipidemia (6, 11–13). In this context, there is a biological plausibility to associate hypomagnesemia with metabolic syndrome. Studies suggest potential benefits of magnesium in preventing the development of metabolic syndrome and its components, although the literature is not consistent in this regard. Some studies (14–16) demonstrated an inverse association between magnesium levels and metabolic syndrome, whereas others could not establish a significant association (17–19). Surprisingly, a case-control study has proposed that higher levels of magnesium were associated with an increased risk of developing metabolic syndrome (20).

Given the worldwide increase in the prevalence of metabolic syndrome and its major medical and social burden, efforts have been made to shed light on the possible mechanisms involved in its pathogenesis so as to develop strategies focused on its prevention and management. Therefore, the aims of this study were to estimate the prevalence of metabolic syndrome among Jordanians and to investigate the

association between low serum magnesium levels and metabolic syndrome and its components in a national population-based cross-sectional study.

2. Methods

2.1. Sample selection

This study derived its data from the national population-based household survey from the 12 governorates of Jordan 2009 (21). These 12 governorates belong to the three regions of the country, namely, the North, Middle, and South. The Sample was selected using a complex multistage sampling technique taking into consideration the geographic distribution of the population as well as the urban-rural residence. Because the study procedures (anthropometric measures, drawing blood samples, etc.) required a clinical setting and the population is covered by extensive networks of health centers, the selection of households was heath center-oriented. The health director in each governorate was contacted and asked to select at least 2 health centers in the governorate which he believes to be representative and at the same time large enough to provide the space for the study team (25 members), participants, and equipment. A total of 31 health centers distributed throughout Jordan were selected and the people served by these centers were targeted. A systematic sample of households was selected from the catchment area of each selected health center.

A team of two (one man and one woman) went door-to-door selecting every third to sixth household, depending on the population density. The team invited all those aged ≥ 7 years in the selected households to report to the health center the next day after explaining the purpose of the study to them. Participants were asked to fast and not to take their medication on that day, but to bring their medications to the health center. To encourage participation, the team worked on weekends and holidays, and provided free transportation for those who requested it. Of the 9,000 subjects invited to participate 5,640 responded. The overall response rate was 63% being 36% in men and 90% in women. The main reason behind the lower response rate in men was employment, often far from their place of residence. The unemployment rate in women is much higher than in men, which makes women more available for participation.

2.2. Data collection

The study was multipurpose covering several aspects of health care for non-communicable diseases through the collection of a wide array of data, including interview data, anthropometric measurements, and laboratory data.

- **Interview data**

Data collection took place between 1st of July and 30th of November, 2009. Participants attended the health centers in the morning (8–11 am) with a minimum 10 hours fasting time. The study included a total of 5640 subjects aged between 7 and 90 years. A pilot tested structured questionnaire was prepared

and administered by well-trained interviewers to collect relevant data on sociodemographic factors, risk factors for cardiovascular disease, style of dress and sun exposure, morbidity, quality of life and health services use, among others. The present study deals, exclusively, with participants ≥ 20 years of age. There was a total of 4,520 subjects (1113 men and 3407 women).

- **Anthropometric measurements**

Anthropometric measurements including weight, height, waist, and hip circumferences were measured with the subjects wearing light clothing and no shoes. Height and weight were determined to the nearest 0.1 cm. Waist circumference was measured to the nearest centimeter using non-stretchable tailors measuring tape at the narrowest point between the iliac crest and rib cage during minimal respiration. Readings of systolic (SBP) and diastolic blood pressure (DBP) were taken in duplicates with the subject seated and the arm at heart level, after at least 5 minutes of rest, using standardized mercury sphygmomanometer with appropriate arm cuff length. The mean of these two determinations was used to express the individual's systolic and diastolic blood pressures.

- **Laboratory tests**

For all biochemical measurements, two sets of fasting blood samples were drawn from a cannula inserted into the antecubital vein into sodium fluoride potassium oxalate tubes for glucose and lithium heparin vacuum tubes for lipids. Samples were centrifuged at 3000 rpm for 10 min within 1 hour at the survey site, and plasma was transferred to separate labeled tubes and transferred immediately in cold boxes filled with ice to the central laboratory of the National Center for diabetes and endocrinology. All biochemical measurements were carried out by the same team of laboratory technicians and the same method throughout the study period. Triglycerides values were obtained on COBAS INTEGRA 700 with the cassette COBAS INTEGRA Triglycerides using enzymatic, colorimetric method (GPO/PAP) with glycerol phosphate oxidase and 4-aminophenazone. Total cholesterol was analyzed using enzymatic, colorimetric method with COBAS INTEGRA Cholesterol Gen.2. HDL cholesterol and LDL cholesterol values were obtained on COBAS INTEGRA 700 using homogeneous enzymatic colorimetric assay. Magnesium was measured by “Colorimetric Endpoint Method” using Roche/Cobas Integra 800 automated system. Other laboratory analysis was also performed for several blood constituents, but it is not described here because they are irrelevant to the current report.

2.3. Definition of variables

Metabolic syndrome was defined according to IDF definition (22) which entails the presence of central obesity (waist circumference > 94 cm in men and > 80 cm in women) along with two or more of the following: Raised triglycerides > 150 mg/dl or specific treatment for the lipid abnormality, blood glucose greater than 100 mg/dl or diagnosed diabetes, low HDL-Cholesterol: HDL-C < 40 mg/dl in men and < 50 mg/dl in women, or blood pressure ≥ 130/85 mmHg or drug treatment for hypertension. As for magnesium, our operational definition of hypomagnesemia was the occurrence of serum magnesium level below 1.82 mg/dl (23).

2.4. Ethical considerations

Ethical approval was obtained from the Ethical Committee for Research on Humans of the National Center for Diabetes, Endocrinology and Genetics, and it was supported by the Ministry of Higher Education. An informed consent was obtained from the responded individuals and their guardians in case the participants were children. Physical examinations and interviews were carried out by well-trained personnel. All study procedures were performed free of charge for participants. The privacy of participants was respected. Identifying information was kept strictly confidential and the data were used only for scientific purposes. The examination results were disseminated to each participant privately and in case there were abnormal findings, they would be referred to their doctors for necessary intervention.

2.5. Data management and statistical analysis

Data were analyzed using the Statistical Package for Social Science (SPSS version 21). The continuous data were expressed as mean and standard deviation (SD), and the category data were expressed in percentage. Prevalence rates of metabolic syndrome and its components were obtained and reported separately for each gender. The overall prevalence of hypomagnesemia was calculated and also in the subgroups defined by relevant variables. The bivariate association between low magnesium levels and a number of variables was assessed for statistical significance using the chi-square test. Multivariate logistic regression models were fit to demonstrate the association of hypomagnesemia with metabolic syndrome and its components while simultaneously controlling for age as a potential confounder. For this purpose, magnesium status was dichotomized, using 1.82 mg as the cutoff point, and entered the model as the dependent variable. All other relevant variables were treated as independent variables. Gender was removed from the logistic regression models as it was insignificant in the bivariate analysis and remained insignificant in the models. Magnesium was also analyzed as a continuous variable using linear regression models while controlling for age as a potential confounder. A P-value of < 0.05 was considered statistically significant.

3. Results

3.1. Socio-demographic Characteristics

Table 1 shows the sociodemographic characteristics of the study population. This study included 4520 participants [1113 (24.6%) men and 3407 (75.4%) women] aged 20 years or more. About 27.8% of them were between 40–49 years old. The majority of participants (79.9%) were married while 12.9% were single. Current smokers constituted 15.1% of the study participants while 79.9% were non-smokers.

3.2. Anthropometric and Clinical Characteristics

Table 2 shows the mean anthropometric and clinical characteristics for Jordanians in 2009. The mean (SD) of BMI was 29.9 (5.9) kg/m². The mean (SD) of waist circumference was 87.6 (14.5) cm. The mean (SD) for systolic blood pressure was 122.4 (17.3) mmHg while for diastolic blood pressure was 79.0

(10.3) mmHg. The mean (SD) of fasting blood sugar, HDL, triglycerides, and Magnesium were 102.6 (50.6) mg/dl, 43.9 (13.6) mg/dl, 175.9 (123.5) mg/dl, and 2.4 (0.4) mg/dl, respectively.

Table 2
Anthropometric and clinical characteristics of the study participants

Variable NO.(%)
Age (year)
20–29 827 (18.3)
30–39 1202 (26.6)
40–49 1237 (27.8)
50–59 702 (15.5)
≥ 60 502 (12.2)
Gender
Men 1113 (24.6)
Women 3407 (75.4)
Marital status
Single 585 (12.9)
Married 3613 (79.9)
Divorced 67 (1.5)
Widow 255 (5.6)
Smoking status
Current 684 (15.1)
Past 215 (4.8)
Never 4609 (79.9)
(N = 4520), Jordan 2009

3.3. Prevalence rates of metabolic syndrome and its components

Table 3 shows the gender-specific rates of metabolic syndrome and its individual components of the study participants, using IDF definition. The prevalence of metabolic syndrome was 39.8% being higher in women (40.7%) than in men (37.4%); p-value (0.041). The prevalence of central obesity was 62.6% (51.3% among men and 66.5% among women, p = 0.000). The prevalence of triglycerides was 49.2% being

higher in men (63.0%) than in women (44.7%); p value (0.000). Similarly, the prevalence of low HDL was higher in men (69.6%) than in women (64.0%) with an overall prevalence of 65.5%; p value (0.000). The prevalence of hypertension was 41.0% (53.1% in men and 37.0% in women, p = 0.000). The prevalence of hyperglycemia was 29.6% (38.4% in men and 26.8% in women, p = 0.000).

Table 3
The gender-specific rates of metabolic syndrome and its individualized components in Jordan, using the IDF definition, Jordan 2009

Variable Mean (SD)
Body mass index (kg/m²) 29.9 (5.9)
Waist circumference (cm) 87.6 (14.5)
Systolic blood pressure (mmHg) 122.4 (17.3)
Diastolic blood pressure (mmHg) 79.0 (10.3)
Fasting blood sugar (mg/dl) 102.6 (50.6)
HDL (mg/dl) 43.9 (13.6)
Triglyceride (mg/dl) 175.9 (123.5)
Serum magnesium (mg/dl) 2.4 (0.4)

3.4. Prevalence of hypomagnesemia in the study population

The overall prevalence of hypomagnesemia in the population was 2.7% (n = 120). The prevalence rates of hypomagnesemia by sociodemographic characteristics are shown in Table 4. Participants older than 60 years old were significantly more likely to have low magnesium levels (5.4%) compared to younger age groups (p = 0.000).

Table 4
Prevalence of hypomagnesemia, overall and by Sociodemographic characteristics of the study participants, Jordan 2009

Variable Men Women Total P value			
No. (%) No. (%) No. (%)			
Metabolic syndrome (IDF)			
Yes 354 (37.4) 1090 (40.7) 1444 (39.8)			
No 593 (62.6) 1590 (59.3) 2183 (60.2) 0.041			
Central obesity (Waist circumference ≥ 80cm for women & ≥ 94cm for men)			
Yes 569 (51.3) 2151 (66.5) 2720 (62.6)			
No 541 (48.7) 1083 (33.5) 1624 (37.4) 0.000			
Elevated Triglycerides (Triglycerides ≥ 150mg/dl or specific treatment for lipid abnormality)			
Yes 701 (63.0) 1523 (44.7) 2224 (49.2)			
No 412 (37.0) 1884 (55.3) 2296 (50.8) 0.000			
Low HDL (HDL-C < 40 mg/dl in men and < 50 mg/dl in women)			
Yes 778 (69.6) 2182 (64.0) 2960 (65.5)			
No 335 (30.1) 1225 (36.0) 1560 (34.5) 0.000			
Hypertension (Blood pressure ≥ 130/85 mmHg or drug treatment for hypertension)			
Yes 589 (53.1) 1258 (37.0) 1847 (41.0)			
No 521 (46.9) 2138 (63.0) 2659 (59.0) 0.000			
Hyperglycemia (Blood glucose greater than 100 mg/dl or treatment for diabetes)			
Yes 313 (38.4) 673 (26.8) 986 (29.6)			
No 502 (61.6) 1838 (73.2) 2340 (70.4) 0.000			

Table 5 shows the prevalence of hypomagnesemia by metabolic syndrome and its components. Subjects with metabolic syndrome were significantly more likely to have low magnesium levels (4.3%) than those without the condition (p = 0.000). Central obesity was significantly associated with low magnesium levels (2.8%) in comparison to non-obese subjects (p = 0.041). Subjects with low HDL were significantly more likely to have a high prevalence of hypomagnesemia (3.3%) compared to those with normal HDL levels (p = 0.000). Hypertensive subjects were significantly more likely to have low magnesium levels (3.6%) in comparison with non-hypertensive subjects (p = 0.001). The prevalence of hypomagnesemia is significantly higher among hyperglycemic subjects (5.1%) compared to subjects with normal blood glucose (p = 0.002).

Table 5
Prevalence of hypomagnesemia by metabolic syndrome and its individualized components, using IDF definition, Jordan 2009

Variable Serum magnesium levels, mg/dl		
	>1.82 No. (%)	<=1.82 No. (%) P value
Total	4400 (97.3)	120 (2.7)
Age (years)		
20–29	809 (97.8)	18 (2.2)
30–39	1174 (97.9)	28 (2.3)
40–49	1220 (98.6)	17 (1.4) 0 .000
50–59	675 (96.6)	27 (3.8)
≥ 60	522 (94.6)	30 (5.4)
Gender		
Men	1091 (98.0)	22 (2.0)
Women	3309 (97.1)	98 (2.9) 0.062
Marital status		
Single	574 (98.1)	11 (1.9)
Married	3518 (97.4)	95 (2.6)
Divorced	66 (95.5)	1 (1.5) 0.055
Widow	242 (94.9)	13 (5.1)
Smoking status		
Current	674 (98.5)	10 (1.5)
Past	211 (98.1)	4 (1.9) 0.068
Never	3503 (97.1)	106 (2.9)

3.5. Multivariate analysis of factors associated with hypomagnesemia

We performed multivariate logistic regression to identify the factors related to hypomagnesaemia after controlling for age. Table 6 shows the association of low magnesium status with metabolic syndrome after adjusting for age. We found that metabolic syndrome patients were 1.57 times more likely to have

hypomagnesemia as compared to those without the condition (p = 0.048). participants who were 60 years or more were 2.73 times more likely to have hypomagnesemia compared to younger age groups (p = 0.007).

Table 6
Factors related to hypomagnesemia among the study participants using multivariate logistic regression, Jordan 2009

Variable Serum magnesium levels, mg/dl		
>1.82	**<=1.82**	**P value**
No. (%)	**No. (%)**	
Total 4400 (97.3)	120 (2.7)	
Metabolic syndrome (IDF)		
Yes 1382 (95.7)	62 (4.3)	
No 2139 (98.0)	44 (2.0)	0.000
Central obesity (Waist circumference ≥ 80cm for women & ≥ 94cm for men)		
Yes 2644 (97.2)	76 (2.8)	
No 1593 (98.1)	31 (1.9)	0.041
Elevated triglycerides (Triglycerides ≥ 150mg/dl or specific treatment for lipid abnormality)		
Yes 2174 (97.8)	50 (2.2)	
No 2226 (97.0)	70 (3.0)	0.057
Low HDL (HDL-C < 40 mg/dl in men and < 50 mg/dl in women)		
Yes 2863 (96.7)	97 (3.3)	
No 1537 (98.5)	23 (1.5)	0.000
Hypertension (Blood pressure ≥ 130/85 mmHg or drug treatment for hypertension)		
Yes 1781 (96.4)	66 (3.6)	
No 2605 (98.0)	54 (2.0)	0.001
Hyperglycemia (Blood glucose greater than 100 mg/dl or treatment for diabetes)		
Yes 936 (94.9)	50 (5.1)	
No 2271 (97.1)	69 (2.9)	0.002

Table 7 shows the association of low magnesium status with metabolic syndrome components after adjusting for age only. In the bivariate analysis, hypomagnesemia was significantly associated with central obesity, low HDL, hypertension, and hyperglycemia. After using multivariate logistic regression, the only variable that remains statistically significant was low HDL. We found that participants with low HDL

were 2.15 more likely to have hypomagnesemia in comparison to those with normal HDL levels (p = 0.001). Interestingly, hypomagnesemia was found to be statistically significant with high triglycerides levels. People with high levels of triglycerides were 60% less likely to develop hypomagnesemia compared to those with normal triglycerides levels (p = 0.010).

Table 7

Metabolic syndrome components in relation to hypomagnesemia among the study participants using multivariate logistic regression, Jordan 2009

Variable Odds Ratio P value
Metabolic syndrome (IDF)
No 1
Yes 1.57 0.048
Age (years)
20–29 1
30–39 1.06 0.862
40–49 0.77 0.512
50–59 1.90 0.088
≥ 60 2.73 0.007
Variable Odds Ratio* P value
Central obesity (Waist circumference ≥ 80cm for women & ≥ 94cm for men)
No 1
Yes 1.12 0.621
Elevated triglycerides (≥ 150mg/dl or specific treatment for lipid abnormality)
No 1
Yes 0.60 0.010
Low HDL (HDL-C < 40 mg/dl in men and < 50 mg/dl in women)
No 1
Yes 2.15 0.001
Hypertension ((Blood pressure ≥ 130/85 mmHg or drug treatment for hypertension)
No 1
Yes 1.37 0.140
Hyperglycemia ((Blood glucose greater than 100 mg/dl or treatment for diabetes)
No 1
Yes 1.43 0.091
***Each of these variables in this table was adjusted for age only

In linear regression models, we also found an inverse association between magnesium and metabolic syndrome and low HDL when magnesium was analyzed as a continuous variable.

4. Discussion

In this national population-based study conducted in Jordan in 2009, we demonstrated the prevalence of metabolic syndrome and its components among adults and their relation to low magnesium levels. We found alarmingly high prevalence rates of metabolic syndrome and its components as defined by IDF criteria. Indeed, more than one-third of all Jordanian adults met the criteria of metabolic syndrome (39.8%), with the highest burden being among women (40.7% in women and 37.4% in men). Among metabolic syndrome components, central obesity and low HDL were the most commonly occurring components of metabolic syndrome (62.6% and 65.5%, respectively). In fact, central obesity was more prevalent among women (66.5%), while low HDL was more prevalent among men (69.6%). These findings are consistent with the study conducted in Northern Jordan, using ATP definition, which reported a metabolic syndrome prevalence estimate of 36.3%, being higher in women than in men (40.9% and 28.7%, respectively). Furthermore, this study found that central obesity was the most common abnormality in women (69.1%), while low HDL was the most common abnormality in men (62.7%) (24). Interestingly, using IDF criteria, the prevalence of metabolic syndrome in Jordan was markedly higher in 2017 being 48.2% (52.9% in men and 46.2% in women) (25).

Our metabolic syndrome prevalence estimate was slightly higher than those reported in the US population from 2003–2012 (33.0%) (26) and Australian population (35.8%) (27). On the other hand, higher estimates were reported in Turkey (44.0%), Pakistan (63.7%), Tunisia (45.5%), and Emirates (48.7%) (28). The considerable variations in the prevalence rates of metabolic syndrome may be attributable to many factors, including the criteria of metabolic syndrome used, study design and sampling, the variations in age and gender structure of the population, and a combination of genetics and environmental factors.

Our results showed high rates of abdominal obesity (62.6%). This is in line with the studies conducted among the US and Greek population with abdominal obesity rates of 53% and 72%, respectively (29, 30). Interestingly, counties with high rates of metabolic syndrome demonstrated high rates of central obesity; Portugal (51.0%)(31), Turkey (56.8%)(32), Tunisia (69.5%)(33). consequently, the high prevalence of metabolic syndrome could be explained by the high prevalence of abdominal obesity. Obesity is a worldwide health problem being attributable to the increased risk of several diseases, including diabetes and cardiovascular diseases (34). It has been proposed that by 2030, obesity will reach levels of 89% and 85% in males and females, respectively (35). Therefore, the implementation of health care programs is strongly recommended to increase people's awareness towards an adoption of a healthy lifestyle in the form of appropriate dietary habits and physical activity.

Magnesium deficiency may play an essential role in the development of metabolic syndrome. In line with this, our results revealed an independent relationship between metabolic syndrome and low serum magnesium levels. The inverse association found in the present study is supported by findings of various

other studies (14–16, 36–40). Furthermore, studies have demonstrated that metabolic syndrome is less prevalent in subjects with higher levels of dietary magnesium intake (41–46). In this context, results from these studies support the hypothesis that low body magnesium status could be a potential risk factor for the development of metabolic syndrome. The present finding is not consistent, however, with the results from cross-sectional studies which revealed no association between serum magnesium and metabolic syndrome (17, 43). No association was also found between dietary magnesium intake and supplementation with metabolic syndrome in prospective and clinical randomized studies, respectively (47, 48). Additionally, hair magnesium concentration was shown to have no significant association with metabolic syndrome (19). The discrepancy between the results of the studies could be explained by differences in the sample size, variability in the characteristics of the study populations, and the geographical locations where there are differences in the lifestyle, dietary habits, and genetic phenotype among individuals. In fact, populations may have different responses to the same level of magnesium depending on the variations in genetic background (16). Interestingly, the use of different criteria for metabolic syndrome could affect the association. Indeed, a meta-analysis study found that the association was stronger in studies that used the NCEP-ATP III definition rather than the modified NCEP-ATP III, IDF, AHA/NHLBI, or WHO definitions (16). Furthermore, hair analysis is considered to be an unreliable tool for assessing trace elements and nutritional balance in individuals (49).

The relationship between serum magnesium levels and components of the metabolic syndrome is conflicting. Our study suggests an independent association between hypomagnesemia and decreased levels of HDL-cholesterol. Our finding is in agreement with the study conducted by Guerrero-Romero concluding that hypomagnesemia contributed to lower HDL (13). A case-control study of low serum magnesium level and lipid profile among patients with osteoarthritis, found that hypomagnesemia was related to decreased serum levels of HDL ($p < 0.001$) (50). Findings suggest that low serum magnesium levels could be incriminated in the pathogenesis of cardiovascular disease through alteration in the blood lipid composition in a way that predisposes to atherosclerosis (51). One possible explanation is that magnesium acts as a cofactor for a number of enzymes involved in lipid metabolism. Indeed, it has been postulated that magnesium intake may increase the activity of lipoprotein lipase, which is involved in the conversion of triglycerides to HDL-Cholesterol (52).

Our results revealed no significant association between low magnesium levels and central obesity. This finding is in accordance with the previous reports (53–55). In fact, it has been shown that middle-aged obese individuals can maintain normal circulating levels of magnesium, compared to type 2 diabetes and older subjects (56). We have also failed to demonstrate a cross-sectional relationship between low serum magnesium levels and hyperglycemia. Our results are supported by a 12-week clinical randomized study concluding that magnesium replacement in recommended dosage didn’t reduce insulin resistance (48). Finally, our study showed that individuals with hypertension had lower levels of serum magnesium with respect to those with normal blood pressure, but this association was no longer significant in multivariate analysis.

Although our study has the strength of being a national population-based study with a relatively large sample size, there are several limitations that deserve to be mentioned. First, since the study was cross-sectional in nature, a temporal relationship between hypomagnesemia and metabolic syndrome can't be established, and thus we don't know whether low magnesium concentration was a consequence of metabolic syndrome or a precipitating factor leading to its development. Second, the dietary intake of magnesium in relation to the prevalence of metabolic syndrome and its components was not assessed in the present study. Last but not least, we have used serum magnesium to indicate low magnesium status. It is important to highlight the fact that magnesium is chiefly an intracellular ion; therefore, serum magnesium is considered to be a poor indicator of body magnesium. However, serum magnesium is the most widely used measure of magnesium status in many studies to illustrate the relationship between magnesium and metabolic syndrome.

In conclusion, the prevalence of metabolic syndrome is considerably high in Jordan accounting for more than one-third of all Jordanian adults. Central obesity and low HDL were the most occurring components of metabolic syndrome. The increase in the prevalence of metabolic syndrome and its components is probably due to the adoption of westernized behaviors and sedentary life-style. Therefore, future policies and health education programs, aiming at encouraging people to adhere to the guidelines and recommendations of a healthy diet and physical activity, should be taken into consideration. Furthermore, the findings from the present study provide evidence that serum magnesium is inversely associated with the prevalence of metabolic syndrome. However, the data cannot support a causal role for hypomagnesemia in the development of metabolic syndrome because of the cross-sectional nature of the study.

Declarations

Conflicts of Interest: The authors declare no conflict of interest.

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Tables

Table 1: Sociodemographic characteristics of the study participants (N=4520), Jordan 2009

Variable	NO.(%)
Age (year)	
20-29	827 (18.3)
30-39	1202 (26.6)
40-49	1237 (27.8)
50-59	702 (15.5)
≥ 60	502 (12.2)
Gender	
Men	1113 (24.6)
Women	3407 (75.4)
Marital status	
Single	585 (12.9)
Married	3613 (79.9)
Divorced	67 (1.5)
Widow	255 (5.6)
Smoking status	
Current	684 (15.1)
Past	215 (4.8)
Never	4609 (79.9)

Table 2: Anthropometric and clinical characteristics of the study participants (N=4520), Jordan 2009

Variable	Mean (SD)
Body mass index (kg/m²)	29.9 (5.9)
Waist circumference (cm)	87.6 (14.5)
Systolic blood pressure (mmHg)	122.4 (17.3)
Diastolic blood pressure (mmHg)	79.0 (10.3)
Fasting blood sugar (mg/dl)	102.6 (50.6)
HDL (mg/dl)	43.9 (13.6)
Triglyceride (mg/dl)	175.9 (123.5)
Serum magnesium (mg/dl)	2.4 (0.4)

Table 3: The gender-specific rates of metabolic syndrome and its individualized components in Jordan, using the IDF definition, Jordan 2009

Variable	Men No. (%)	Women No. (%)	Total No. (%)	P value
Metabolic syndrome (IDF)				
Yes	354 (37.4)	1090 (40.7)	1444 (39.8)	
No	593 (62.6)	1590 (59.3)	2183 (60.2)	0.041
Central obesity (Waist circumference ≥ 80cm for women & ≥ 94cm for men)				
Yes	569 (51.3)	2151 (66.5)	2720 (62.6)	
No	541 (48.7)	1083 (33.5)	1624 (37.4)	0.000
Elevated Triglycerides (Triglycerides ≥ 150mg/dl or specific treatment for lipid abnormality)				
Yes	701 (63.0)	1523 (44.7)	2224 (49.2)	
No	412 (37.0)	1884 (55.3)	2296 (50.8)	0.000
Low HDL (HDL-C < 40 mg/dl in men and < 50 mg/dl in women)				
Yes	778 (69.6)	2182 (64.0)	2960 (65.5)	
No	335 (30.1)	1225 (36.0)	1560 (34.5)	0.000
Hypertension (Blood pressure ≥ 130/85 mmHg or drug treatment for hypertension)				
Yes	589 (53.1)	1258 (37.0)	1847 (41.0)	
No	521 (46.9)	2138 (63.0)	2659 (59.0)	0.000
Hyperglycemia (Blood glucose greater than 100 mg/dl or treatment for diabetes)				
Yes	313 (38.4)	673 (26.8)	986 (29.6)	
No	502 (61.6)	1838 (73.2)	2340 (70.4)	0.000

Table 4: Prevalence of hypomagnesemia, overall and by Sociodemographic characteristics of the study participants, Jordan 2009

Variable	Serum magnesium levels, mg/dl		P value
	>1.82 No. (%)	<=1.82 No. (%)	
Total	4400 (97.3)	120 (2.7)	
Age (years)			
20-29	809 (97.8)	18 (2.2)	
30-39	1174 (97.9)	28 (2.3)	
40-49	1220 (98.6)	17 (1.4)	0 .000
50-59	675 (96.6)	27 (3.8)	
≥ 60	522 (94.6)	30 (5.4)	
Gender			
Men	1091 (98.0)	22 (2.0)	
Women	3309 (97.1)	98 (2.9)	0.062
Marital status			
Single	574 (98.1)	11 (1.9)	
Married	3518 (97.4)	95 (2.6)	
Divorced	66 (95.5)	1 (1.5)	0.055
Widow	242 (94.9)	13 (5.1)	
Smoking status			
Current	674 (98.5)	10 (1.5)	
Past	211 (98.1)	4 (1.9)	0.068
Never	3503 (97.1)	106 (2.9)	

Table 5: Prevalence of hypomagnesemia by metabolic syndrome and its individualized components, using IDF definition, Jordan 2009

Variable	Serum magnesium levels, mg/dl		P value
	>1.82 No. (%)	<=1.82 No. (%)	
Total	4400 (97.3)	120 (2.7)	
Metabolic syndrome (IDF)			
Yes	1382 (95.7)	62 (4.3)	
No	2139 (98.0)	44 (2.0)	0.000
Central obesity (Waist circumference ≥ 80cm for women & ≥ 94cm for men)			
Yes	2644 (97.2)	76 (2.8)	
No	1593 (98.1)	31 (1.9)	0.041
Elevated triglycerides (Triglycerides ≥ 150mg/dl or specific treatment for lipid abnormality)			
Yes	2174 (97.8)	50 (2.2)	
No	2226 (97.0)	70 (3.0)	0.057
Low HDL (HDL-C < 40 mg/dl in men and < 50 mg/dl in women)			
Yes	2863 (96.7)	97 (3.3)	
No	1537 (98.5)	23 (1.5)	0.000
Hypertension (Blood pressure ≥ 130/85 mmHg or drug treatment for hypertension)			
Yes	1781 (96.4)	66 (3.6)	
No	2605 (98.0)	54 (2.0)	0.001
Hyperglycemia (Blood glucose greater than 100 mg/dl or treatment for diabetes)			
Yes	936 (94.9)	50 (5.1)	
No	2271 (97.1)	69 (2.9)	0.002

Table 6: Factors related to hypomagnesemia among the study participants using multivariate logistic regression, Jordan 2009

Variable	Odds Ratio	P value
Metabolic syndrome (IDF)		
No	1	
Yes	1.57	0.048
Age (years)		
20-29	1	
30-39	1.06	0.862
40-49	0.77	0.512
50-59	1.90	0.088
≥60	2.73	0.007

Table 7: Metabolic syndrome components in relation to hypomagnesemia among the study participants using multivariate logistic regression, Jordan 2009

Variable	Odds Ratio*	P value
Central obesity (Waist circumference ≥ 80cm for women & ≥ 94cm for men)		
No	1	
Yes	1.12	0.621
Elevated triglycerides (≥ 150mg/dl or specific treatment for lipid abnormality)		
No	1	
Yes	0.60	0.010
Low HDL (HDL-C < 40 mg/dl in men and < 50 mg/dl in women)		
No	1	
Yes	2.15	0.001
Hypertension ((Blood pressure ≥ 130/85 mmHg or drug treatment for hypertension)		
No	1	
Yes	1.37	0.140
Hyperglycemia ((Blood glucose greater than 100 mg/dl or treatment for diabetes)		
No	1	
Yes	1.43	0.091

*Each of these variables in this table was adjusted for age only

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