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Dietary magnesium intake and rheumatoid arthritis patients' all-cause mortality: evidence from the NHANES database

Hantian Liu[1], Kui Zhang[2] and Long Xiong[3*]

Abstract

Background Rheumatoid arthritis (RA) is a chronic inflammatory joint disease with all-cause mortality increasing globally. Dietary magnesium (Mg), an anti-inflammatory nutrient, has been proven to be associated with the all-cause mortality. The association of dietary Mg intake and all-cause mortality in RA patients remains unknown. The aim of this study was to assess the association between dietary Mg intake and all-cause mortality in RA patients.

Methods RA patients were extracted from the NHANES 1999–2018, and followed for survival through December 31, 2019. Dietary Mg intake data were obtained from 24-h dietary recall interview. The association between dietary Mg intake and RA patients' all-cause mortality was explored based on weighted univariate and multivariate Cox proportional hazard models and described as absolute risk difference (ARD), hazard ratios (HRs) and 95% confidence intervals (CIs). This association was further explored in subgroup analyses based on different age, gender and body mass index (BMI).

Results Totally 2,952 patients were included. Until 31 December 2019, a total of 825 deaths were documented. RA patients with higher dietary Mg intake had a 11.12% reduction of all-cause mortality (ARD=-11.12%; HR = 0.74, 95%CI: 0.56–0.99) in the fully adjusted model, especially in female (HR = 0.68, 95%CI: 0.47–0.98), aged < 65 years (HR = 0.59, 95%CI: 0.37–0.94) and BMI ≤ 30 kg/m^2 (HR = 0.62, 95%CI: 0.42–0.91).

Conclusion RA patients who consumed adequate dietary Mg from diet as well as supplements may had a lower risk of all-cause mortality.

Keywords Magnesium, Rheumatoid arthritis, All-cause mortality, NHANES database

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Introduction

Rheumatoid arthritis (RA), an autoimmune disease featured by systemic inflammation and invasive destruction of multiple joints, greatly affects the quality of patients' life [1–3]. RA affects between 0.5 and 1% population worldwide, and the risk of female were two to three times more than men [4]. Related studies reported an approximately 50-60% increase in mortality in RA patients compared with healthy people [5, 6]. Active prevention and treatment are essential to improving the outcomes of RA patients.

Inflammation may be an important factor leading to premature death in RA patients [7]. It has been shown that approximately a quarter of the all-cause mortality in RA patients was mediated by inflammation mediates [8]. Several inflammation cytokines, such as tumor necrosis factor-α, interleukin-1 and 6, were significantly increased in RA patients [9–11]. These inflammatory cytokines result in the progression of RA synovitis, which leads to joint destruction and ultimately disability and premature death [12, 13]. Currently, several studies have shown the potential interaction between dietary nutrients and immune diseases [14, 15]. Several dietary patterns and supplements, such as the Mediterranean diet (MD), dietary total antioxidant capacity, vitamin D and probiotics, have proven to be associated with arthritis or osteoarthritis [16, 17]. Magnesium (Mg), a biologically active mineral, acts as a cofactor in hundreds of enzymatic reactions in the human body. Previous observations studies reported that Mg intake was inversely related to inflammatory diseases including hypertension, type 2 diabetes mellitus and cardiovascular disease (CVD) [18]. Mendes PMV et al. [19] reported the all-cause mortality risk was reduced by 6% for every 100 mg/d increase of dietary Mg intake. In addition, several studies have suggested that insufficient dietary Mg intake increase the poor prognosis risk in various cancers [20–22]. Less in known, however, the relationship of Mg intake and all-cause mortality in RA patients.

Herein, we evaluated the relationship of dietary Mg intake and RA patients' all-cause mortality based on the National Health and Nutrition Examination Survey (NHANES) database. This study was aimed to lay a theoretical foundation for the good prognosis of RA patients from the perspective of improving the diet.

Methods

Study design and RA patients

In this retrospective cohort study, data of RA patients were extracted from the NHANES 1999–2018. NHANES is a survey conducted by National Center for Health Statistics (NCHS), a part of the Centers for Disease Control and Prevention (CDC) and is responsible for assessing the health and nutritional status for the U.S. civilian. The NHANES protocols are approved by the NCHS Ethics Review Board of the US CDC and all participants provided written informed consent. According to the Ethics Review Board of Second Affiliated Hospital of Nanchang University, cross-sectional studies have been exempted from the ethical review.

The included criteria were: (1) patients diagnosed as RA; (2) patients aged≥18 years old; (3) patients with complete dietary Mg intake information. The excluded criteria were: (1) missing important covariates [white blood cell (WBC), BMI, smoking and RA medication use]; (2) missing survival data.

Outcome and follow-up

From baseline through 31 December 2019, the vital status and cause of death information were followed by the National Center for Health Statistics (obtained at: NCHS Data Linkage - Mortality Data - Public-Use Files (cdc.gov). Vital status was ascertained by probabilistic matching of subjects to the National Death Index based on social security number, name, sex and date of birth. Details of the linkage methods have been reported previously (obtained at: 2011 Linked Mortality Files Matching Metholodogy (cdc.gov). The follow-up time of the study was calculated from the NHANES 1999–2018 examination data until the last known data alive or censored through 31 December 2019 [23].

Mg intake assessment

Dietary Mg intake data were obtained through 24-h dietary recall interview. This interview was carried out via face-to-face at the Mobile Examination Center (MEC). The U.S. Department of Agriculture (USDA) Automated Multiple-Pass Method was utilized to collect dietary recall recorded during the physical health examination and participants were required to recall all of the food consumed in the 24-h prior to the interview, including information on the time of intake, amount and type food, and detailed food descriptions [24]. Then, each reported food item was linked to the USDA's Food and Nutrient Database for Dietary Studies (FNDDS) to assign 8-digit food codes. The USDA'S food codes were also used to sort the foods into the What We Eat in America (WWEIA) food categories and subcategories. The dietary Mg intake was grouped into two groups in present study according to whether meeting the recommended nutrient intake (RNI). The RNI of dietary Mg was 400 and 420 mg/d for male aged 18–30 years and 31 years and above, respectively; 310 and 320 mg/d for female aged 18–30 years and 31 years and above, respectively [25].

Potential covariates

Demographic variables included age, gender, race and marital status. Smoking status was defined as never

smokers, former smokers (smoking at least 100 cigarettes in life) and current smokers (smoking less than 100 cigarettes in life) [26]. Mg intake were calculated as the total of dietary Mg intake and Mg-supplement intake (supplements deficiency were counted as 0). Physical activity was presented as the metabolic equivalent task (MET) and calculated by the following formula: recommended MET × exercise time for corresponding activities (min/day) × the number of exercise days per week (day) [27].

The medical history data adopted in this study were determined on the basis of the medical condition questionnaires. Diabetes was defined as hemoglobin A1C (HbA1c) ≥ 6.5% or fasting glucose ≥ 126 mg/dL or 2 h oral glucose tolerance test (OGTT) blood glucose ≥ 200 mg/dL or diagnosed as diabetes by doctors [28]. Hypertension was defined as systolic blood pressure (SBP) ≥ 130 mmHg or diastolic blood pressure (DBP) ≥ 80 mmHg or taking blood pressure medication or diagnosed as hypertension by doctors [29].Subjects with total cholesterol (TC) ≥ 200 mg/dL (5.2 mmol/L), or triglyceride (TG) ≥ 150 mg/dL (1.7 mmol/L), or low density lipoprotein cholesterol (LDL-C) ≥ 130 mg/dL (3.4 mmol/L), or high density lipoprotein cholesterol (HDL-C) ≤ 40 mg/dL (1.0 mmol/L), or diagnosed as hypercholesterolemia by doctors or receiving cholesterol-lowering therapy and lipid-lowering drugs were defined as dyslipidemia [30]. Cardiovascular disease (CVD) was assessed by the question of "Ever told you had angina or heart failure or heart attack or coronary heart disease or stroke or congestive heart failure "[30]. Cancer was assessed by "Ever told you had any cancer or malignancy". The mean bone mineral density (BMD) of white females aged 20–29 years was used as the reference value. Osteoporosis was defined as participants with any BMD score of 2.5 standard deviations or more below the norm. Participants with all BMD values of 1.0 standard deviations or more above the norm were considered normal BMD, and other subjects were considered low bone mass [31]. Drugs that affect the absorption of Mg were identified based on participants' self-reported use the tetracycline and loop diuretics.

Statistical analysis

All statistical tests were performed by SAS 9.4 software (SAS Institute Inc., Cary, NC, USA). Continuous data were expressed as mean and standard error (S.E.), and the weighted t-test was used for comparison between all-cause deaths and survivor groups. Categorical data were described by case number and percentage [n (%)], and weighted χ^2 test was utilized to compare the differences between all-cause deaths and survivor groups. Missing data imputation was conducted using multivariate imputation by chained equations (MICE). Sensitivity analyses were performed to compare whether the results were different before and after imputation (Supplementary Table S1). The weighted univariable and multivariate Cox proportional hazard models were used to explore the association between dietary Mg intake and RA patients' all-cause mortality (Supplementary Table S2), described as hazard ratios (HRs), absolute risk difference (ARD) and 95% confidence intervals (CIs). Model I was a crude model. Model II was a fully adjusted model and accounted for age, race, PIR, osteoporosis, fracture, drug influence magnesium absorption, CVD, energy and calcium. The association was further explored through different subpopulations stratified by age, gender and BMI. Two-sided P-value < 0.05 was regarded as statistically significant.

Results

Characteristics of RA patients

Figure 1 shows the flow chart of population screening. A total of 2,952 RA patient were screened. Among them, 324 patients missing complete dietary Mg intake information, 384 missing WBC measurement, 56 patients missing BMI data, 2 patients missing smoking status, and 5 patients missing survival data were excluded. Finally, 2,181 patients were included, with the mean age of 57.52 (0.40) years. Of which, 825 (37.83%) were all-cause deaths. The rate of dietary Mg intake above the RNI was significant higher in the survival group than in all-cause mortality group (29.48% vs. 18.60%). Characteristics of studied RA patients were presented in Table 1. Differences were found in age, race, PIR, marital status, physical activity, the history of osteoporosis, fracture, hypertension cancer CVD, cancer, diabetes and CKD, the use of antirheumatics and drug influence Mg absorption and the intake of energy, calcium and Mg between two groups (P < 0.05).

Dietary mg intake and all-cause mortality

The relationship of dietary Mg intake and RA patients' all-cause mortality was shown in Table 2. After adjusted age, race, PIR, osteoporosis, fracture, drug influence mg absorption, CVD, energy and calcium in model II, compared with the unreached RNI of dietary Mg group, RA patients who reaching the RNI of dietary Mg had a 11.12% reduction of all-cause mortality (ARD=-11.12%; HR = 0.74, 95%CI: 0.56–0.99). After excluding the patients missing dietary Mg intake and survival information, we observed the association of dietary Mg intake and all-cause mortality in RA patients remain robust (Supplementary Table S3).

Dietary mg intake and all-cause mortality stratified by age, gender and BMI

Table 3 shown the relationship of dietary Mg intake and all-cause mortality among RA patients stratified by age, gender and BMI. After adjusted for all covariates,

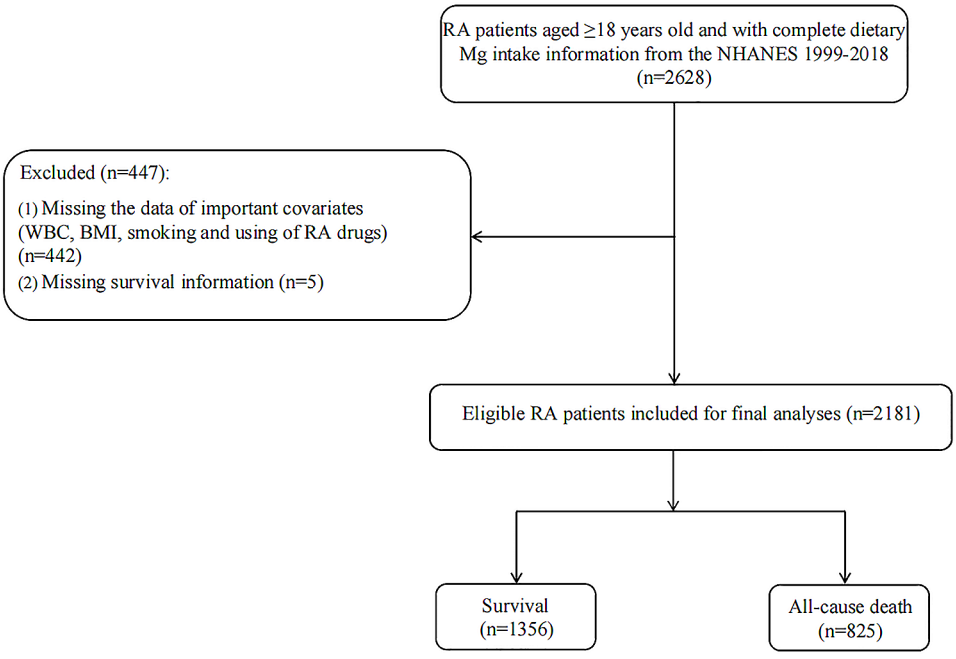

Fig. 1 The flow chart of population screening

patients who reached the RNI of Mg was also associated with the lower all-cause mortality and this relationship was pronounced in patients with female (HR=0.68, 95%CI: 0.47–0.98), aged<65 years (HR=0.59, 95%CI: 0.37–0.94) and BMI≤30 kg/m^2 (HR=0.62, 95%CI: 0.42–0.91) (all $P<0.05$), respectively.

Discussion

The association between dietary Mg intake and the all-cause mortality in RA patients was explored in this retrospective cohort study. We observed that there was a closely association of dietary Mg intake and all-cause mortality in RA patients. Keeping a higher dietary Mg intake may be a beneficial measure to improve the prognosis of RA patients.

The known pathogenesis of RA includes inflammatory cell infiltration, synovial hyperplasia, and bone destruction. A study quantifying causes of mortality of RA suggested inflammation mediated about a quarter of the excess relative RA mortality risk [8]. Therefore, reducing inflammation by all available means would be of great benefit in reducing the high mortality rate in RA population. In recent years, diet and nutrients, as potential environmental factors that affect the development and course of diseases, have attracted great attention from scholars. Nutrients regulate inflammatory states in the body, so it has emerged in nutrition science to classify the pro-inflammatory or anti-inflammatory properties of certain foods [32]. Similarly, this phenomenon applies to people with RA. More and more studies have pointed out Mg as common anti-inflammatory and antioxidant dietary nutrition has a great benefit to the alleviation of RA. Mg has a strong anti-inflammatory effect, and higher Mg intake has been proven to be related to lower inflammatory markers levels [33, 34]. Mg deficiency was related to an increased risk of many clinical diseases and mortality [35–38]. Short-term low Mg diet was related to elevated levels of inflammatory factors, which was consistent with findings proposed by Brenner et al. [38]. A prospective cohort study suggested that there was a negative relationship between high intake of antioxidant vitamins A, E, Mg and selenium and lower all-cause mortality [39]. A systematic review of Bagheri et al. [14] pointed out dietary Mg intake was related to a lower all-cause and cancer mortality, but not with CVD mortality. Moreover, no significant association was observed between supplemental and total Mg intake and the risk of all-cause, CVD and cancer mortality. In present study, dietary Mg intake was calculated as the total amount of dietary and supplement intake. The relationship between the source of dietary Mg intake and the all-cause mortality risk warrants attention in future studies.

Previous studies also shown the all-cause mortality risk was higher in female RA patients compared with male, which is consistent with our findings. RA has the characteristics of sexual dimorphism in clinical manifestations [40]. Women were 3-folds more likely to develop RA than men. Gender may show significant differences in autoimmune dysfunction and vaccination response [41, 42]. Because of differences in body composition and structure, women may be more susceptible than men to inflammation or immune burden during the process of

Table 1 Characteristics of RA patients

Variables	Total ($n=2181$)	Survivor ($n=1356$)	All-cause mortality ($n=825$)	Statistics	*P*
Age, years, Mean (S.E)	57.52 (0.40)	54.07 (0.42)	66.65 (0.75)	t = 0.01	< 0.001
Gender, n (%)				$\chi^2 = 2.239$	0.135
Male	887 (40.32)	517 (38.95)	370 (43.95)		
Female	1294 (59.68)	839 (61.05)	455 (56.05)		
Race, n (%)				$\chi^2 = 17.229$	0.002
Mexican American	368 (6.67)	242 (7.59)	126 (4.23)		
Other Hispanic	165 (4.55)	130 (5.13)	35 (3.02)		
Non-Hispanic White	949 (68.27)	512 (65.81)	437 (74.77)		
Non-Hispanic Black	615 (16.23)	407 (16.51)	208 (15.50)		
Other races	84 (4.29)	65 (4.97)	19 (2.48)		
PIR, Mean (S.E)	2.50 (0.06)	2.58 (0.07)	2.30 (0.07)	t = 2.71	0.008
Marital status, n (%)				$\chi^2 = 6.537$	0.038
Married	1799 (83.47)	1086 (81.70)	713 (88.16)		
Single	258 (10.67)	189 (12.02)	69 (7.09)		
Unknown	124 (5.86)	81 (6.28)	43 (4.75)		
Smoking, n (%)				$\chi^2 = 3.405$	0.182
Never smoker	967 (41.25)	632 (42.59)	335 (37.69)		
Former smoker	689 (32.34)	397 (31.28)	292 (35.17)		
Current smoker	525 (26.41)	327 (26.14)	198 (27.14)		
Drinking, n (%)				$\chi^2 = 0.272$	0.602
Less than once per week	2006 (87.98)	1242 (88.34)	764 (87.03)		
More than once per week	175 (12.02)	114 (11.66)	61 (12.97)		
Physical activity, n (%)				$\chi^2 = 15.184$	< 0.001
Lower	1217 (53.84)	713 (50.51)	504 (62.64)		
Higher	964 (46.16)	643 (49.49)	321 (37.36)		
BMI, kg/m^2, n (%)				$\chi^2 = 5.727$	0.057
<25	491 (23.76)	262 (21.88)	229 (28.73)		
25–30	661 (28.98)	416 (28.95)	245 (29.07)		
>30	1029 (47.27)	678 (49.18)	351 (42.21)		
Osteoporosis, n (%)				$\chi^2 = 14.134$	< 0.001
Osteoporosis	1412 (56.38)	827 (57.00)	585 (54.77)		
Low bone mass	174 (9.53)	97 (7.20)	77 (15.72)		
Normal bone mass	595 (34.08)	432 (35.80)	163 (29.52)		
Fracture, n (%)				$\chi^2 = 22.915$	< 0.001
No	2130 (97.52)	1338 (98.72)	792 (94.34)		
Yes	51 (2.48)	18 (1.28)	33 (5.66)		
WBC, 1000 cells/uL, Mean (S.E)	7.46 (0.09)	7.44 (0.11)	7.53 (0.12)	t = 0.01	0.545
Antirheumatics, n (%)				$\chi^2 = 0.751$	0.386
No	1999 (87.95)	1237 (87.48)	762 (89.19)		
Yes	182 (12.05)	119 (12.52)	63 (10.81)		
Glucocorticoid, n (%)				$\chi^2 = 1.421$	0.233
No	2034 (92.42)	1278 (93.05)	756 (90.75)		
Yes	147 (7.58)	78 (6.95)	69 (9.25)		
Drug influence Mg absorption, n (%)				$\chi^2 = 59.103$	< 0.001
No	1988 (92.59)	1289 (95.90)	699 (83.82)		
Yes	193 (7.41)	67 (4.10)	126 (16.18)		
Hypertension, n (%)				$\chi^2 = 39.213$	< 0.001
No	494 (27.46)	378 (32.01)	116 (15.38)		
Yes	1687 (72.55)	978 (67.99)	709 (84.62)		
Dyslipidemia, n (%)				$\chi^2 = 2.463$	0.117
No	526 (22.51)	343 (23.65)	183 (19.50)		
Yes	1655 (77.49)	1013 (76.35)	642 (80.50)		
CVD, n (%)				$\chi^2 = 58.476$	< 0.001

Table 1 (continued)

Variables	Total ($n=2181$)	Survivor ($n=1356$)	All-cause mortality ($n=825$)	Statistics	*P*
No	1266 (62.23)	887 (68.56)	379 (45.47)		
Yes	915 (37.77)	469 (31.45)	446 (54.54)		
Liver disease, n (%)				$\chi^2=0.053$	0.817
No	2051 (93.06)	1280 (92.97)	771 (93.31)		
Yes	130 (6.94)	76 (7.03)	54 (6.69)		
Cancer, n (%)				$\chi^2=10.304$	0.001
No	1855 (82.81)	1185 (85.26)	670 (76.34)		
Yes	326 (17.19)	171 (14.74)	155 (23.66)		
Diabetes, n (%)				$\chi^2=22.781$	< 0.001
No	1526 (75.17)	986 (78.49)	540 (66.38)		
Yes	655 (24.83)	370 (21.51)	285 (33.62)		
CKD, n (%)				$\chi^2=99.796$	< 0.001
No	1949 (90.78)	1282 (95.04)	667 (79.51)		
Yes	232 (9.22)	74 (4.96)	158 (20.49)		
COPD, n (%)				$\chi^2=0.670$	0.413
No	1712 (76.70)	1061 (77.31)	651 (75.08)		
Yes	469 (23.30)	295 (22.70)	174 (24.92)		
Total energy, kcal, Mean (S.E)	1986.56 (30.59)	2056.14 (36.21)	1802.17 (53.22)	t = 3.96	< 0.001
Calcium, mg, Mean (S.E)	1010.38 (24.38)	1049.05 (31.20)	907.88 (30.89)	t = 3.17	0.002
Mg, mg, Mean (S.E)	300.58 (7.61)	309.37 (9.67)	277.29 (12.16)	t = 2.01	0.047
Mg intake ≥ RNI, n (%)				$\chi^2=12.575$	< 0.001
No	1715 (73.50)	1025 (70.52)	690 (81.41)		
Yes	466 (26.50)	331 (29.48)	135 (18.60)		

S.E: standard error; t: t-test; χ^2: chi-squared test

RA: rheumatoid arthritis; Mg: magnesium; PIR: poverty-to-income ratio; WBC: white blood cell; CVD: cardiovascular disease; CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease; RNI: recommended nutrient intake

Table 2 Associations between dietary mg intake and all-cause mortality

Outcome	Model II			
	HR (95%CI)	*P*	Weighted all-cause mortality	ARD
Whether the dietary Mg intake reaching the RNI				
No	Ref		30.34	-11.12%
Yes	0.74 (0.56–0.99)	0.039	19.22%	

HR: hazard ratio; CI: confidence interval; ARD: absolute risk difference; Ref: reference

Mg: magnesium; RA: rheumatoid arthritis; RNI: recommended nutrient intake

Model I: crude model

Model II: adjustment for age, race, PIR, osteoporosis, fracture, drug influence magnesium absorption, CVD, energy and calcium

sex differentiation [41]. The production of gonadal-specific hormones in women during special periods such as childbearing age, pregnancy, and lactation may affect the incidence or course of RA. One study has shown that disease activity tends to improve spontaneously in 75% of pregnant women, with episodes occurring in up to 90% of cases after delivery [43]. Mg plays a vital part in regulating endothelial function and is associated with circulatory markers of systemic inflammation and endothelial dysfunction in women [44, 45]. All these results indicate that gender should be carefully considered in the clinical treatment of RA population.

In the age stratification analyzed by subgroups in this study, we found the all-cause mortality risk in RA patients also showed significant differences in the factor of age and the association between all-cause mortality and dietary Mg intake was pronounced in RA patients who aged < 65 years. It is well known CVD is the most serious complication of RA and the important cause of death in RA patients [46]. An previous study shown that the incidence of CVD events in RA patients younger than 50 were 2.6-folds higher compared to the general population, while in old age the incidence was only 1.3 times [41]. With the deepening of clinical research on RA, scholars have found that increasingly serious pathological injury can aggravate the degree of destruction of articular cartilage and bone, increase the risk of osteoporosis fracture, and affect the quality of prognosis. A cohort study also showed that RA patients have a significantly higher risk of a first fracture before aged ≤ 50 years than patients aged > 50 years, taking into account reasons such as glucocorticoid use, smoking, and alcohol [41]. The pathogenesis of RA patients in the two age groups is different, so clinical medication should also be different. Joint deformation is more obvious in the middle-aged

Table 3 Associations between dietary mg intake and all-cause mortality stratified by age, gender and BMI

Outcomes	Model I		Model II	
	HR (95%CI)	*P*	HR (95%CI)	*P*
Age < 65 years (*n* = 1243)				
No	Ref		Ref	
Yes	0.52 (0.31–0.88)	0.015	0.59 (0.37–0.94)[#]	0.026
Age ≥ 65 years (*n* = 938)				
No	Ref		Ref	
Yes	0.71 (0.51–0.99)	0.045	0.84 (0.61–1.17)[#]	0.312
Male (*n* = 887)				
No	Ref		Ref	
Yes	0.56 (0.36–0.89)	0.013	0.76 (0.47–1.23)[*]	0.259
Female (*n* = 1294)				
No	Ref		Ref	
Yes	0.68 (0.47–0.97)	0.032	0.68 (0.47–0.98)*	0.037
BMI ≤ 30 kg/m^2 (*n* = 1152)				
No	Ref		Ref	
Yes	0.55 (0.38–0.80)	0.002	0.62 (0.42–0.91)[!]	0.016
BMI > 30 kg/m^2 (*n* = 1029)				
No	Ref		Ref	
Yes	0.74 (0.53–1.05)	0.091	0.84 (0.57–1.24)[!]	0.373

HR: hazard ratio; CI: confidence interval; Ref: reference

Mg: magnesium; RA: rheumatoid arthritis; BMI: body mass index

Model I: crude model

[#]: adjustment for race, PIR, osteoporosis, fracture, drug influence magnesium absorption, CVD, energy and calcium

*: adjustment for age, race, PIR, osteoporosis, fracture, drug influence magnesium absorption, CVD, energy and calcium

[!]: adjustment for age, race, PIR, osteoporosis, fracture, drug influence magnesium absorption, CVD, energy and calcium

and elderly group, and the elderly group should pay more attention to the degree of organ involvement and other complications in addition to joint damage.

Moreover, we also found RA patients with low BMI had a higher all-cause mortality risk compared to overweight and obese patients. Studies generally believe that low BMI is a risk factor for osteoporosis [47]. Lean individuals with dietary Mg intake of < 200 mg/d versus > 200 mg/d are also at higher risk for hypertension, which may increase another key pathophysiological mediator associated with Mg deficiency.

Therefore, we provided reference for the management of RA based on the relationship of dietary Mg intake and the all-cause mortality. For clinicians and policymakers, as well as RA patients, it was essential to be aware of the benefits of dietary Mg for health management of RA. In addition, it was a beneficial move to add the Mg-enriched foods to dietary diet such as some nuts, legumes, and fiber-rich whole grains. Our study also has some limitations. First, despite the 24-h dietary recall interview was the valid method to obtain the dietary intake data, participants' memory bias may bring a difficult to acquiring the accurate evaluation. Moreover, the weighted multifactor Cox regression model included as many covariates related to RA population as possible, but the confounding effects of unconsidered or unknown factors still could not be ruled out. Finally, information such as disease history in this study obtained through questionnaires may exist the recall bias, which may affect the results of the study.

Conclusion

Higher dietary Mg intake may be an efficient measurement to improve the prognosis of RA patients, especially in RA patients with female, aged < 65 years and BMI ≤ 30 kg/m^2. Further large-scale prospective cohort study is needed to explore this beneficial effect of dietary Mg intake and health outcome of RA patients.

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

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Not applicable.

Author contributions

(1) Long Xiong, conceiving and designing the study; (2) Hantian Liu, collecting the data; (3) Hantian Liu, analyzing and interpreting the data; (4) Hantian Liu, Kui Zhang, writing the manuscript; (5) Long Xiong, providing critical revisions that are important for the intellectual content; (6) Hantian Liu, Kui Zhang, Long Xiong, approving the final version of the manuscript.

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Data availability

Publicly available datasets were analyzed in this study. This data can be found here NHANES, NHANES Questionnaires, Datasets, and Related Documentation (cdc.gov).

Declarations

Ethics approval and consent to participate

Ethical approval was not provided for this study on human participants because NHANES is a publicly available dataset. The patients/participants provided their written informed consent to participate in this study. According to the Ethics Review Board of Second Affiliated Hospital of Nanchang University, cross-sectional studies have been exempted from the ethical review.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Conflict of interest
all authors declare that they have no conflict of interests.

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