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Association Between Dietary Selenium Intake and Kidney Stones Disease Among Patients with Metabolic Syndrome: A Cross-Sectional Study from the NHANES Database

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Background: Clinically, metabolic syndrome (MetS) is associated with the formation and relapse of kidney stones diseases (KSD). In the general population, dietary selenium can reduce renal damage by reducing oxidative stress and other physiological pathways. Less is known, however, about the association between dietary selenium and KSD in patients with MetS.

Objective: The present study's purpose is to evaluate the association between dietary selenium intake and the odds of KSD in MetS populations.

Methods: Data of MetS patients aged ≥20 years were extracted from the National Health and Nutrition Examination Survey (NHANES) database (2007–2018). The information of dietary selenium intake was obtained by 24-hour dietary recall interview. Weighted univariable and multivariate logistic regression analyses were used to evaluate the association of selenium intake with KSD in MetS patients and described as odds ratios (ORs) and 95% confidence intervals (CIs). Subgroup analysis was performed to further discuss this association based on age, gender, and MetS component.

Results: In total, 6,073 patients were included, with 766 (12.61%) KSD cases. After adjusting for covariates, high dietary selenium intake was related to lower odds of KSD in MetS patients (OR = 0.70, 95% CI = 0.50–0.97), especially in females (OR = 0.61, 95% CI = 0.39–0.96), those aged <65 years (OR = 0.53, 95% CI = 0.35–0.80), without a history of hypertriglyceridemia (OR = 0.61, 95% CI = 0.40–0.93) and with a history of hypertension (OR = 0.57, 95% CI = 0.38–0.84), diabetes (OR = 0.68, 95% CI = 0.46–0.99) or central obesity (OR = 0.67, 95% CI = 0.48–0.95).

Conclusion: From this cross-sectional study, we observed that, among patients with MetS, high dietary selenium intake is associated with lower odds of KSD, implying a potential nutritional strategy for preventing KSD in this population.

Keywords: dietary selenium, metabolic syndrome, kidney stone diseases, NHANES database

Introduction

The National Cholesterol Education Program (NCEP)/Adult Treatment Panel III (ATP III) proposed the definition of metabolic syndrome (MetS), namely that MetS is a global and prevalent metabolic disorder comprised of hypertension, central obesity, hyperlipidemia, and hyperglycemia, and the weighted prevalence is 34.70% in the United States.[1–4] Insulin resistance is considered an important factor to be most commonly associated with the development of each of the MetS components, resulting in a lower urine PH and increasing the risk of uric acid stone disease.[5,6] Kidney stone disease (KSD), as a benign disease of the urinary system, is rising globally, with a lifetime prevalence of approximately 10%.[7] Studies have reported that the occurrence of KSD is closely related to the sub-characteristics of MetS, such as obesity, hypertension, dyslipidemia, and hypertension.[8–10] Active prevention and treatment are essential to maintain the renal health of MetS patients.

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Previous studies have reported that diet is believed to play an important role in the increased incidence of urinary stones. Poor eating habits can directly lead to metabolic disorders in the body, such as hypercalciuria, hyperoxaluria, and hyperuricosuria.[11,12] Among numerous dietary nutrients, the role of selenium with an important antioxidant and anti-inflammatory effect has gradually been noted. Selenium is one of the essential trace elements for the human body. It exists in the human body in the form of various selenium-containing functional proteins and is of great significance to maintain the normal function of cells. Among them, glutathione plays an important role in maintaining the oxidation–antioxidation balance in cells.[13] Patients with KSD are characterized by elevated reactive oxygen species, lipid peroxidation, pro-inflammatory cytokines, and pro-angiogenic factors.[14] Ouyang et al[15] found that high selenium intake is beneficial of reducing insulin levels. Qi et al[16] have reported that higher dietary selenium intake is associated with lower odds of KSD in adults aged over 60 years. However, the association between dietary selenium intake and KSD in the MetS population remains unclear. Based on previous studies, we speculate that dietary selenium intake is associated with the odds of KSD in patients with MetS. Herein, based on the National Health and Nutrition Examination Survey (NHANES) 2007–2018, we aimed to evaluate the association of dietary selenium intake and the odds of KSD among patients with MetS. Moreover, subgroup analyses were conducted based on MetS components to further evaluate this association in different subpopulations.

Materials and Methods

Study Design and Participants

In the present cross-sectional study, data of MetS patients were extracted from the NHANES 2007–2018. The NHANES is conducted by the National Centers for Health Statistics (NCHS), a part of the Centers for Disease Control and Prevention (CDC) to assess the health and nutritional status among civilians across the United States. It is conducted biennially and draws a representative sample across the United States. This survey contains detailed information of in-person interviews, physical examinations, laboratory values, and physical examinations by using complex, multistage, probability sampling methods based on broad population distributions. A written informed consent was obtained from all subjects before any data collection was conducted by the NCHS Ethics Review Board.

In this study, 6,819 patients diagnosed with MetS were initially extracted from the NHANES 2007–2018. Among them, 222 patients were aged <20 years, 22 patients were missing the diagnosis information of KSD, 296 patients had missing information of dietary selenium and energy intake, 206 patients were missing important variates including urine albumin and creatinine, serum creatinine education level, smoking, body mass index (BMI), uric acid (UA), lymphocyte, white blood cell (WBC), red blood cell (RBC), platelet, serum calcium, serum sodium, serum potassium, serum phosphorus, blood urea nitrogen (BUN), and were excluded. Finally, 6,073 eligible patients with MetS were included. The flow chart of population screening is shown in Figure 1.

Assessment of Dietary Selenium Intake

Data of dietary selenium and supplements intake were obtained by 24-hour dietary recall interview. The interview was conducted at a Mobile Test Center (MTC) through in-person communication. Participants were asked to recall all the types and amount of food and drink consumed in the 24 hours prior to the interview. Nutrients were calculated using the Food and Nutrient Database for Dietary Studies published by the United States Department of Agriculture (USDA).[17]

Potential Covariates

The potential covariates in this study included age, gender, race, education level, BMI, physical activity, and poverty-to-income ratio (PIR). 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).[18] Drinking status was measured by the questionnaire of "alcohol use" and defined as heavy drinker (drinking ≥8 times per week), moderate drinker (drinking 1–8 times per week), occasional drinker (drinking <1 time per week) and never drinker (drinking 0 times per week).[19] PIR was categorized as <1, ≥1, and unknown. BMI ≥30 kg/m^2 was categorized as obesity, 25–29.9 kg/m^2 as overweight, 18.5–24.9 kg/m^2 as normal, and BMI <18.5 kg/m^2 as underweight.[20]

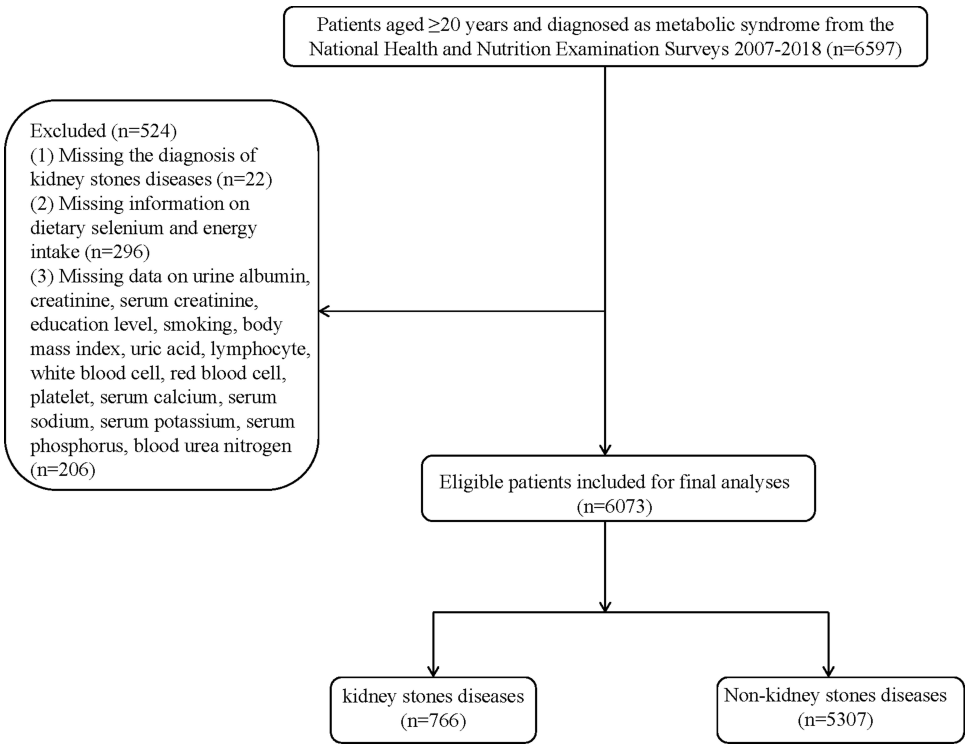

Figure 1 The flow chart of population screening.

The medical history was determined by the medical condition questionnaire (MSQ). Chronic kidney disease (CKD) was defined as urine albumin-to-creatinine ratio (UACR) >30 mg/g or estimated glomerular filtration rate (eGFR) <60 mL/min 1.73 m^2.[21] Cardiovascular disease (CVD) was assessed by the question of "Ever told you had angina or heart failure/heart attack/coronary heart disease/stroke/congestive heart failure?".[22] Physical activity was expressed as the metabolic equivalent task (MET) and calculated as follows: physical activity (met·min/week) = recommended MET × exercise time for corresponding activities (min/day) × the number of exercise days per week (day).[23] The total of energy, sodium, and protein intake were calculated as food and supplements, and supplements deficiency was calculated as 0.

MetS and KDS Definitions

Subjects were diagnosed as MetS if they met three of the following diseases: (1) hypertension [systolic blood pressure (SBP) ≥130 mmHg, diastolic blood pressure (DBP) ≥80 mmHg];[24] (2) high triglycerides [total cholesterol (TC) ≥150 mg/dL]; (3) low level of high density lipoprotein cholesterol (HDL-C) (HDL-C ≤50 mg/dL for females and HDL-C ≤40 for males);[25] central obesity (waistline >88 cm for females and waistline >102 cm for males);[26] and hyperglycemia (fasting glucose ≥100 mg/dL).[27]

The outcome was diagnosed as KSD. KSD was assessed by the question "Do you have kidney stones?". Subjects were defined as having KSD if they answered positively to this question. Previous studies have validated the accuracy of self-reported KSD.[28,29]

Statistical Analysis

Using the proc surveyfreq in SAS software, the final sample size was weighted with SDMVPSU, SDMVSTRA and WTMEC4YR. SDMVPSU means that the masked variance unit pseudo-stratum is sdmvstra, and the masked variance unit pseudo-primary sampling unit (PSU) is sdmvpsu. SDMVSTRA refers to the confidence interval (CI) being applied to assess the reliability of an estimate. WTDRD1 is a set of weights used for the day 1 dietary recall data.

Continuous data were expressed as mean and standard error (SE), and the weighted *t*-test was used for comparison between groups. Categorical data were described by the number of cases and percentage [n (%)], and the chi-square test was used for comparison. The weighted univariable and multivariable logistic regression models were used to assess the

association between dietary selenium intake and KSD in MetS, with odds ratio (ORs) and 95% confidence intervals (CIs). Dietary selenium intake levels were categorized into tertiles: <89.22 mcg, 89.22–140.56 mcg, and ≥140.56 mcg. Model 1 was a crude model without adjusting any covariate. Model 2 adjusted for race, anti-hypertension drug use, anti-diabetic drug use, potassium intake, serum calcium, and serum phosphorus. The associations were further explored stratified by age, gender, and MetS components.

All statistical analyses were performed by SAS 9.4 (SAS Institute Inc., Cary, NC, USA). SDMVPSU, SDMVSTRA, and WTMEC2TR were weighted for the final sample size using the proc surveyfreq in SAS software. P <0.05 was considered as statistically significant. Visualization of forest map was conducted by R version 4.2.2 (2022–10-31 ucrt).

Results

Characteristics of MetS Patients

After the screening, 6,073 participants were included, with a mean age of 53.62 (±0.32) years and a mean dietary selenium intake of 127.31 (±1.43) mcg. Of these, 766 (12.61%) patients had KSD. Characteristics of the included participants are shown in Table 1. Differences were found in age, race, the level of alcohol consumption, BMI, serum calcium and serum phosphorus,

Table 1 Characteristics of MetS Patients

Variables	Total	Non-KSD	KSD	Statistics	P
Number (weighted %)	6,073 (100)	5,307 (86.70)	766 (13.30)		
Age, years, Mean (S.E)	53.62 (0.32)	53.20 (0.34)	56.36 (0.56)	t = −5.07	<0.001
Gender, n (%)				χ^2= 2.030	0.154
Female	3,263 (52.12)	2,897 (52.69)	366 (48.44)		
Male	2,810 (47.88)	2,410 (47.31)	400 (51.56)		
Race, n (%)				χ^2= 40.799	<0.001
White	2,674 (68.22)	2,239 (66.83)	435 (77.30)		
Black	1,097 (9.61)	1,021 (10.45)	76 (4.12)		
Others	2,302 (22.17)	2,047 (22.72)	255 (18.59)		
Education level, n (%)				χ^2= 3.619	0.057
High school graduate or below	3,289 (45.98)	2,910 (46.69)	379 (41.35)		
Some college or above	2,784 (54.02)	2,397 (53.31)	387 (58.65)		
PIR, n (%)				χ^2= 2.156	0.340
<1	1,247 (14.51)	1,101 (14.81)	146 (12.54)		
≥1	4,270 (78.52)	3,715 (78.31)	555 (79.84)		
Unknown	556 (6.97)	491 (6.87)	65 (7.62)		
Smoking, n (%)				χ^2= 4.171	0.124
Current smoker	1,235 (19.93)	1,092 (20.41)	143 (16.80)		
Former smoker	1,727 (30.21)	1,470 (29.45)	257 (35.15)		
Never smoker	3,111 (49.86)	2,745 (50.13)	366 (48.05)		
Alcohol consumption, n (%)				χ^2= 9.840	0.043
Heavy	491 (9.72)	444 (10.11)	47 (7.19)		
Moderate	1,162 (20.17)	995 (19.82)	167 (22.45)		
Occasional	853 (15.82)	752 (16.36)	101 (12.31)		
Never	1,577 (21.86)	1,385 (21.40)	192 (24.86)		
Unknown	1,990 (32.43)	1,731 (32.32)	259 (33.19)		
Physical activity, MET·min/week, n (%)				χ^2= 5.719	0.057
<450	1,694 (28.14)	1,511 (28.86)	183 (23.48)		
≥450	2,360 (42.46)	2,077 (42.46)	283 (42.40)		
Unknown	2,019 (29.40)	1,719 (28.68)	300 (34.13)		
CKD, n (%)				χ^2= 10.943	<0.001
No	4,727 (82.11)	4,171 (82.97)	556 (76.55)		
Yes	1,346 (17.89)	1,136 (17.04)	210 (23.45)		

(*Continued*)

Table 1 (Continued).

Variables	Total	Non-KSD	KSD	Statistics	*P*
Anti-hypertension drug use, n (%)				χ^2= 34.707	<0.001
No	2,673 (47.17)	2,425 (49.34)	248 (33.08)		
Yes	3,400 (52.83)	2,882 (50.66)	518 (66.92)		
Anti-dyslipidemia drug use, n (%)				χ^2= 6.616	0.010
No	3,497 (59.48)	3,129 (60.53)	368 (52.66)		
Yes	2,576 (40.52)	2,178 (39.47)	398 (47.34)		
CVD, n (%)				χ^2= 23.891	<0.001
No	4,151 (70.70)	3,702 (72.33)	449 (60.05)		
Yes	1,922 (29.30)	1,605 (27.67)	317 (39.95)		
Anti-diabetic drug use, n (%)				χ^2= 40.563	<0.001
No	4,628 (79.69)	4,118 (81.40)	510 (68.53)		
Yes	1,445 (20.31)	1,189 (18.60)	256 (31.47)		
Diuretics use, n (%)				χ^2= 7.985	0.005
No	4,686 (79.88)	4,130 (80.75)	556 (74.18)		
Yes	1,387 (20.12)	1,177 (19.25)	210 (25.82)		
BMI, n (%)				χ^2= 6.303	0.043
Obesity	3,931 (66.84)	3,403 (66.16)	528 (71.31)		
Overweight	1,763 (27.70)	1,558 (28.07)	205 (25.32)		
Underweight/Normal	379 (5.46)	346 (5.78)	33 (3.38)		
Energy intake, kcal, Mean (SE)	2,119.48 (18.03)	2,121.96 (20.52)	2,103.30 (51.38)	t = 0.32	0.749
Protein intake, gm, Mean (SE)	81.80 (0.90)	82.57 (0.98)	76.76 (1.91)	t = 2.72	0.008
Calcium intake, mg, Mean (SE)	1,100.40 (15.86)	1,109.22 (18.08)	1,042.88 (27.46)	t = 1.94	0.055
Potassium intake, mg, Mean (.E)	2,632.45 (28.40)	2,654.09 (32.50)	2,491.34 (57.07)	t = 2.35	0.021
Phosphorus intake, mg, Mean (SE)	1,371.25 (14.85)	1,382.36 (16.66)	1,298.79 (32.31)	t = 2.24	0.028
Fiber intake, gm, Mean (SE)	16.31 (0.21)	16.32 (0.22)	16.26 (0.51)	t = 0.11	0.916
Sodium intake, mg, Mean (SE)	3,558.34 (40.89)	3,561.60 (42.74)	3,537.10 (103.12)	t = 0.23	0.820
Moisture intake, gm, Mean (SE)	3,038.74 (33.81)	3,046.82 (35.35)	2,986.09 (73.43)	t = 0.80	0.423
UA, mg/dL, Mean (SE)	5.91 (0.03)	5.92 (0.03)	5.88 (0.08)	t = 0.49	0.624
Lymphocyte, 1,000 cells/μL, Mean (SE)	2.23 (0.02)	2.24 (0.02)	2.17 (0.03)	t = 1.78	0.078
WBC, 1,000 cells/μL, Mean (.E)	7.69 (0.05)	7.66 (0.05)	7.90 (0.13)	t = −1.86	0.066
RBC, million cells/μL, Mean (SE)	4.77 (0.01)	4.78 (0.01)	4.74 (0.03)	t = 1.50	0.136
Platelet, 1,000 cells/μL, Mean (SE)	246.24 (1.42)	246.20 (1.45)	246.52 (3.85)	t = −0.08	0.935
Serum calcium, mg/dL, Mean (SE)	9.36 (0.01)	9.36 (0.01)	9.32 (0.02)	t = 2.66	0.009
Serum sodium, mmol/L, Mean (SE)	139.28 (0.11)	139.27 (0.11)	139.35 (0.16)	t = −0.62	0.539
Serum potassium, mmol/L, Mean (SE)	4.03 (0.01)	4.04 (0.01)	4.03 (0.02)	t = 0.32	0.751
Serum phosphorus, mg/dL, Mean (SE)	3.66 (0.01)	3.67 (0.01)	3.60 (0.03)	t = 2.29	0.024
BUN, mg/dL, Mean (SE)	14.28 (0.11)	14.20 (0.12)	14.84 (0.32)	t = −1.97	0.051
MetS components, n (%)				χ^2= 2.034	0.362
3	4,180 (68.41)	3,662 (68.34)	518 (68.90)		
4	1,475 (24.55)	1,291 (24.85)	184 (22.63)		
5	418 (7.04)	354 (6.82)	64 (8.47)		
Selenium, mcg, Mean (SE)	127.31 (1.43)	128.63 (1.59)	118.72 (2.84)	t = 3.02	0.003
Selenium, mcg, n (%)				χ^2= 11.716	0.003
<89.22	2,268 (32.97)	1,970 (32.27)	298 (37.56)		
89.22–140.56	1,982 (34.01)	1,727 (33.52)	255 (37.16)		
≥140.56	1,823 (33.02)	1,610 (34.21)	213 (25.28)		

Note: χ^2, chi-square test; t, *t*-test; SE, standard error.

Abbreviations: MetS, metabolic syndrome; KSD, kidney stones disease; PIR, poverty-to-income ratio; CKD, chronic kidney disease; CVD, cardiovascular disease; BMI, body mass index; UA, uric acid; WBC, white blood cell; RBC, red blood cell; MET, metabolic equivalent task. BUN, blood urea nitrogen.

a history of CKD and CVD, intake of protein, potassium, phosphorus and selenium, and the use of anti-hypertension drugs, anti-dyslipidemia drugs, anti-diabetic drugs, and diuretics between two groups (P <0.05).

Association Between Dietary Selenium Intake and KSD in Patients with MetS

Table 2 shows the association between dietary selenium intake and KSD among MetS patients. After adjusting for race, anti-hypertension drugs, anti-diabetic drugs, potassium intake, serum calcium, and serum phosphorus, compared with low dietary selenium intake (<89.22 mcg), high dietary selenium intake (≥140.56 mcg) was related to lower odds of KSD among MetS patients (OR = 0.70, 95% CI = 0.50–0.97).

Dietary Selenium Intake and KSD Stratified by Age, Gender, and MetS Components

Figure 2 shows the association between dietary selenium intake and KSD in MetS individuals stratified by age, gender, and the components of MetS. After adjusting for all covariates, compared with low dietary selenium intake (<89.22 mcg), high dietary selenium intake (≥140.56 mcg) was related to low odds of KSD patients, especially in patients who were female (OR = 0.61, 95% CI = 0.39–0.96), aged <65 years (OR = 0.53, 95% CI = 0.35–0.80), without hypertriglyceridemia (OR = 0.61, 95% CI = 0.40–0.93), had a history of hypertension (OR = 0.57, 95% CI = 0.38–0.84), diabetes (OR = 0.68, 95% CI = 0.46–0.99), and those with central obesity (OR = 0.67, 95% CI = 0.48–0.95) (all P <0.05).

Discussion

Based on NHANES 2007–2018, we evaluated the association between dietary selenium intake and KSD among MetS patients. Our findings show that high dietary selenium intake is associated with lower odds of KSD in MetS patients, especially in females, those aged <65 years, with hypertension, diabetes, and central obesity, and those without hypertriglyceridemia.

Several studies have suggested that MetS components are associated with KSD.[30–32] Liu et al[33] reported a significantly increased risk of KSD associated with MetS. When the MetS components increased from three to five, the risk of KSD increased significantly, and the OR value increased from 1.520 to 2.986. A previous study showed that reactive oxygen species (ROS) are generated during the formation of calcium oxalate and calcium phosphate kidney stones.[34] In addition, hypocitraturia is related to defective renal acid excretion and insulin resistance, while increased urinary uric acid and oxalic acid excretion are currently considered as potential causes of calcium stone formation in MetS patients.

Our findings suggest that high dietary selenium intake is related to lower odds of KSD in MetS patients. Considering that diabetes, as a key component of MetS, is associated with oxidative stress, selenium may be beneficial for diabetes.[35,36] Several studies have pointed out that participants with type 2 diabetes (T2D) have lower selenium concentrations than the general population,[37–39] which was consistent with our results. However, several studies have also drawn controversial associations between dietary selenium intake and T2D.[40,41] Dias et al[42] reported that dietary selenium intake was not associated with the prevalence of T2D, despite the high intake of selenium in the participants. Another study, focused on north Chinese adults,

Table 2 Association Between Dietary Selenium Intake and KSD Among MetS Patients

Variables	Model 1		Model 2	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Selenium, mcg				
<89.22	Ref		Ref	
89.22–140.56	0.95 (0.71–1.27)	0.740	0.99 (0.73–1.35)	0.972
≥140.56	0.63 (0.48–0.83)	0.001	0.70 (0.50–0.97)	0.033

Note: Ref, reference; OR, odds ratio; CI, confidence interval; KSD, kidney stones disease; MetS, metabolic syndrome. Model 1: crude model; Model 2: adjusted for race, anti-hypertension drug, anti-diabetic drug, potassium intake, serum calcium, and serum phosphorus.

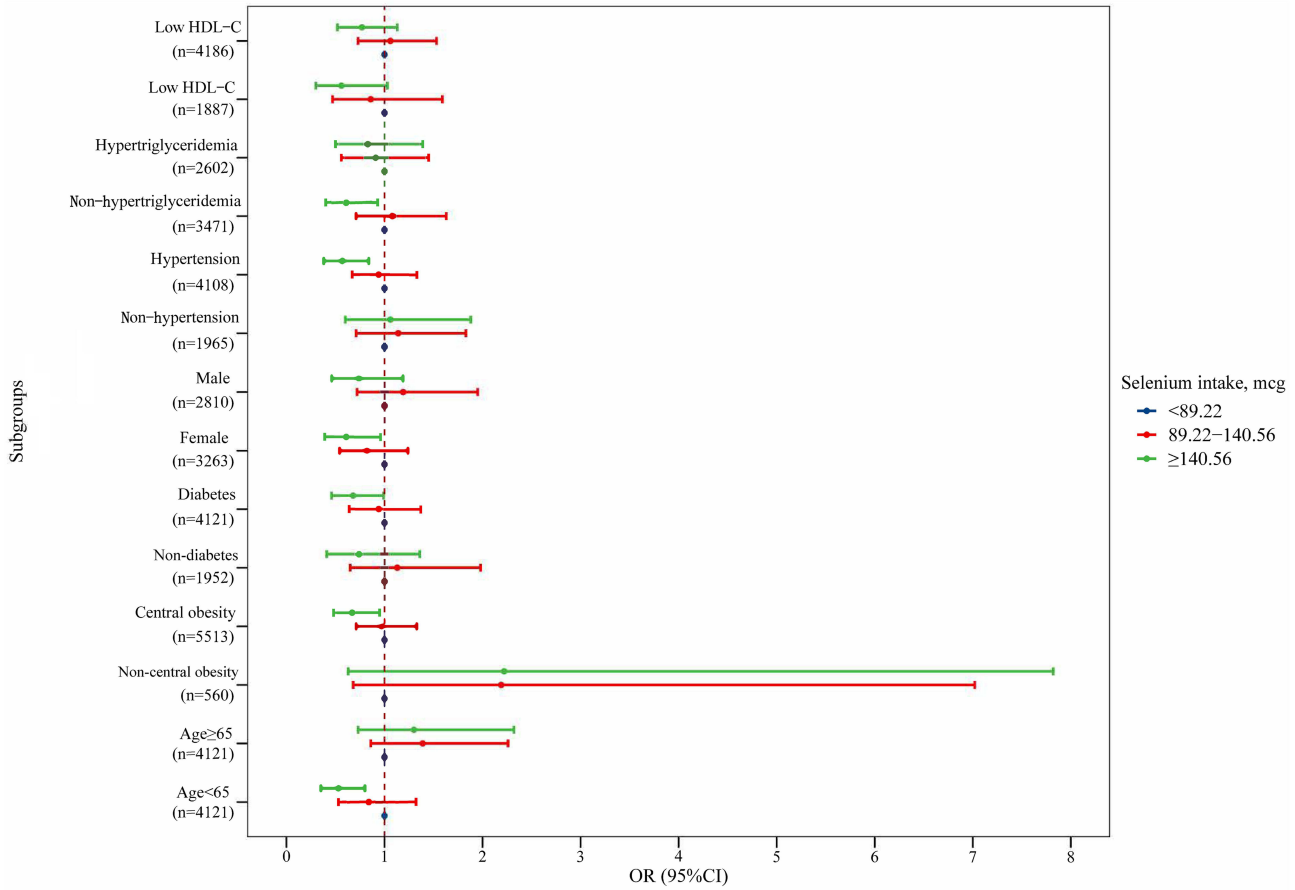

Figure 2 Association between dietary selenium intake and KSD among MetS patients based on age, gender, and MetS components.
Abbreviations: KSD, kidney stones diseases; MetS, metabolic syndrome.

suggested that there exists a positive correlation between dietary selenium intake and T2D.[43] The potential relationship of dietary selenium intake and exposure of T2D needs to be explored further.

We also observed the relationship of dietary selenium intake and KSD was prominent in hypertension patients. Selenium is an essential cofactor for glutathione peroxidase, an enzyme that protects the body from reactive oxygen species and free radical mediated cell membrane damage, so increasing selenium intake can prevent hypertensive diseases and cardiovascular diseases caused by oxidative stress response.[44] Selenium deficiency can lead to hypertension, and chronic selenium deficiency can even lead to Keshan disease and fibrotic heart disease.[45,46] A cross-sectional study reported that high dietary selenium intake in females was associated with lower odds of hypertension.[47] However, the association between selenium and hypertension remains controversial. McKinney et al[48] suggested that high selenium level was associated with a high risk of hypertension among US adults. More studies are needed before recommending high dietary selenium intake in the management of kidney health among hypertensive patients.

Moreover, we found high dietary selenium intake was associated with lower odds of KSD in central obesity. Previous studies suggested selenium was related to energy metabolism.[49,50] Selenium over-removes hydrogen peroxide, one of the key messengers in the insulin signaling pathway, by promoting the production of glutathione peroxidase 1. Appropriate selenium supplementation may be vital to maintain the stability of body weight and energy metabolism. Obese individuals have higher levels of inflammation and chronic oxidative stress, which are closely related to metabolic diseases and insulin resistance. Zhang et al[51] reported that selenium intake is positivity correlated with microalbuminuria in females with obesity, but not in males with obesity. Similar results were found in female MetS. Several studies have revealed a gender-specific effect of selenium for human health.[52,53] In addition, a previous study demonstrated that adult males needed 80 mcg selenium to maintain the balance of selenium per day, whereas

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females needed 57 mcg per day.[54] This indicated that, compared with males, selenium may be easily accumulated in females with a higher selenium intake, suggesting females may be more susceptible to selenium intake-related adverse effects.[55]

Strengths

We have provided evidence for early kidney health management in a MetS population based on the association of dietary selenium and KSD. For clinicians and policymakers, as well as people with MetS, it is essential to be aware of the benefits of dietary selenium for kidney health management. In addition, it is a beneficial move to add selenium-enriched foods and selenium supplements to a daily diet.

Limitations

Several limitations are still visible in this study. First, the information of dietary selenium intake was obtained by 24-hour dietary recall interview, which may include memory bias; second, the type of KSD was not recorded in the NHANES database, so the relationship between the risk of different types of KSD and dietary selenium intake needs to be further explored; finally, the cross-sectional study design could not establish a causal relationship between dietary selenium intake and KSD in MetS populations. Future studies should consider using more accurate methods of assessing participants' dietary selenium intake to address these limitations and obtain accurate and precise results. Also, considering the type of KSD may help clinicians develop more precise dietary interventions for specific patients. Finally, longitudinal studies would help support our results and elucidate the potential mechanisms behind the observed relationships.

Conclusion

High dietary selenium intake was related to lower odds of KSD among patients with MetS, especially in females, those aged <65 years old, with hypertension, diabetes, and central obesity, and without hypertriglyceridemia. Sufficient selenium supplementation and selenium-rich foods intake may be beneficial for kidney health in MetS patients. However, further large and well-designed prospective cohort studies are needed to verify the association between dietary selenium and KSD in patients with MetS.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

Not applicable, because NHANES belongs to public databases, the patients involved in the database have provided ethical approval, users can download relevant data for free for research and publish relevant articles, and our study is based on open-source data, and the Beijing Friendship Hospital, Capital Medical University, do not require research using publicly available data to be submitted for review to their ethics committee, so there are no ethical issues or other conflicts of interest.

Consent for Publication

Not applicable, because this paper did not reveal any personal information of patients.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis, and interpretation, or in all these areas; took part in drafting, revising, or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

All authors declare that they have no conflict of interests.

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