Lower Geriatric Nutritional Risk Index Are Associated With A Higher Incidence Of Osteoporosis In Northern China Type 2 Diabetes

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Abstract

Introduction Geriatric Nutrition Risk Index（GNRI）assesses the risk of malnutrition and complications associated with nutritional status in older patients and is also an important predictor of many diseases. Osteoporosis is very general in the elderly population and can cause fractures and disability. Malnutrition is associated with osteoporosis. Therefore, we investigated the relationship between GNRI and osteoporosis in T2DM.

Methods Study population of 610 patients with T2DM, collect general and laboratory data on the patient and measure BMD; GNRI is calculated based on serum albumin levels, actual weight and ideal weight. Spearman's correlation analysis, logistic regression analysis, receiver operating characteristic (ROC) curves were used to explore the relationship between GNRI and bone metabolism-related indicators.

Results Spearman's correlation analysis yielded a positive correlation between GNRI and BMD, and a negative correlation between GNRI and ALP and PINP. After adjustment of age, duration of illness, TC, TG and uric acid et al confounding factors, a positively correlated with 25(OH)D and PTH (r=0.224, r=0.136, respectively) and negative correlation was found between GNRI and ALP and PINP (r=-0.178, r=-0.120, respectively). Regression analysis yields, GNRI was significantly associated with osteoporosis (in the age < 65 years group, odds ratio was 0.917, P < 0.05 and in the age≥65 years group, was 1.062, P<0.05). The area under the curve of the GNRI recipient operating characteristic curve was 0.584, and the optimal threshold for GNRI to assess the occurrence of osteoporosis was 107.2, the sensitivity was 89.63% and the specificity was 24.63%.

Conclusions: Lower Geriatric Nutritional Risk Index among type 2 diabetic patients in northern China are associated with a higher Incidence of osteoporosis.

Introduction

Osteoporosis is a prevalent disease among the older adults. As we age, osteoporosis and the increased risk of falls can lead to fractures, which can severely impact on people's quality of life and significantly increase the risk of hospitalization and death [1]. The incidence of diabetes is increasing as people's dietary patterns shift towards higher energy levels. Hyperglycemia itself increases the production of advanced glycation end products and negatively affects bone mineralization, bone remodeling, also bone strength [2]. Diabetic complications can also make the risk of osteoporosis much higher [3]. The findings show that the prevalence of diabetic osteoporosis accounts for approximately more than 50% of diabetic patients [4]. Moreover, the adverse outcomes after fracture are more severe in diabetic patients than in normoglycemic patients, so it is important to identify and detect high-risk groups early in elderly T2DM patients [5].

Risk factors for osteoporosis include age, gender, vitamin D levels, muscle strength and nutritional status. The elderly are prone to malnutrition due to their specific metabolic characteristics and disease, and much evidence that malnutrition is an independent risk factor for elderly patients with osteoporosis; studies have shown that low body weight, reduced albumin, and prealbumin can lead to an increased incidence of osteoporotic fractures [6, 7].GNRI is calculated based on the ratio between serum albumin and actual weight to ideal weight, assessing the nutritional condition of older adults; According to the results, there are 4 levels: GNRI < 82 indicates high nutritional risk, GNRI:82 to < 92 is moderate nutritional risk, GNRI:92 to ≤ 98 is low risk, and GNRI > 98 is no risk [8]. GNRI allows for early detection and diagnosis of malnutrition, timely and appropriate interventions, as well as the identification of conditions at risk for adverse effects, including cancer prognosis, postoperative complications, and mortality in dialysis patients and cardiovascular disease [9–13], and is highly accurate and easy to use clinically.

To our knowledge there are fewer studies on the correlation between GNRI and osteoporosis. Liang Wang et al [14] showed that GNRI is associated with osteoporosis as well as BMD in T2DM patients. Their study population consisted of patients from the Second Hospital of Wenzhou Medical University and Yu Ying Children's Hospital, who had adequate vitamin D. However, in northern China, Majority of population is vitamin D insufficient or deficient; therefore, the study by Wang Liang et al. is not fully representative of the type 2 diabetic population in China. This paper investigates the relationship between GNRI and the development of osteoporosis in northern T2DM and assesses the predictive properties of GNRI for osteoporosis.

Topics And Materials

2.1 Topics

This study was a cross-sectional observation of all included patients, 610 patients with T2DM which aged > 50 (317 were men, 293 were postmenopausal women) treated at the Hebei General Hospital from January 2018 to December 2020.The diagnostic criteria of T2DM was based on the 1999 WHO. Exclusion criteria for participants were as follows: (1) individuals with diseases affecting bone metabolism or affect nutritional status, such as malignancies, severe liver diseases, kidney diseases, pituitary-related diseases, thyroid and parathyroid diseases,

adrenal diseases, rheumatoid arthritis, acute inflammatory diseases, etc. (2) individuals who are bedridden for long periods of time; (3) individuals who are also taking drugs that affect bone metabolism, such as vitamin D, calcium, bisphosphonates, glucocorticoids, etc. The study was approved by the Ethics Committee of the Hebei Provincial People's Hospital and complies with the Declaration of Helsinki.

2.2 Clinical Information Collection and Laboratory Measurements

Patient demographics and clinical characteristics, including information on gender, age, disease duration, and comorbidities, were collected from medical records. Weight was measured while wearing light clothing and height was measured without shoes. Body weight divided by height squared (kg/m^2) was used to calculate the body mass index for each patient.

Serum samples were collected after fasting (at least eight hours). Triglycerides, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and albumin levels, glucose metabolism indicators, fasting blood glucose, glycosylated hemoglobin; bone metabolism indicators, alkaline phosphatase, β-CTX, collagen type I N-peptide, 25-hydroxyvitamin D, PTH were measured. In addition, other biochemical markers, such as uric acid, blood creatinine and calcium were tested.

Bone densitometry was performed using a dual-energy X-ray bone densitometer to measure bone density values in the lumbar spine (L1-4), femoral neck, and total hip. According to the criteria for the definition of osteoporosis in the 1994 WHO, T values ≤ 2.5 standard deviations were obtained for any part of the lumbar spine, femoral neck or total hip.

2.3 Calculating GNRI

The GNRI calculation formula is as follows:

GNRI = [1.489 × albumin (g/dL)] + [41.7 × (body weight/WL0)].

WL0 represented the ideal body weight (kg) is calculated as follows :

For men: height (cm)- 100- [(height (cm) − 150)/4]

For women: height (cm)-100- [(height (cm)-150)/2.5]

Actual weight divided by ideal weight was set to 1 when actual weight exceeded the ideal weight [15].

3. Statistical analysis

All data were statistically analyzed using SPSS 25. Data distribution was evaluated with the Kolmogorov-Smirnov test. Mean ± standard deviation was used to indicate that the data were subject to a normal distribution, analysis of variance (ANOVA) was used for comparisons between groups. The median (25th percentile,75th percentile) was used to indicate that the data did not conform to a normal distribution; analysis of Kruskal-Wallis was used for comparisons between groups. Categorical data were expressed as frequencies (%), and differences between groups were determined using the $\chi 2$ test, Spearman correlation analysis was applied to determine the relationship between GNRI and BMD and each clinical index. Logistic regression analysis was performed to assess the relationship between GNRI and osteoporosis. ROC curves were applied to assess the predictive properties of GNRI for osteoporosis and calculate the area under the receiver operating characteristic curve (AUC).

Results

4.1 Table 1 lists the general characteristics of the 610 patients.

As seen in Table 1, all patients were grouped according to GNRI scores, where the differences in gender, duration of disease, age, BMI, albumin, HbA1c, TC, TG, LDL-c, HDL-c, uric acid; Ca, 25(OH)D3, PTH, Total lumbar BMD, Femur neck BMD and prevalence of osteoporosis were statistically significant in different groups.

Table 1
Baseline characteristics of patients stratified by GNRI

	GNRI < 95	95 < GNRI < 100	100 ≤ GNRI < 105	105 ≤ GNRI < 110	GNRI ≥ 110	P
	(n = 56)	(n = 124)	(n = 218)	(n = 169)	(n = 43)	
male/female	29/27	47/77	117/101	93/76	44561	0.01
Diabetes duration	18(10,20)	12(5,17)	10(6,18)	10(4,15.5)	10(3,14)	0.000
Age (years)	67.5(59,70.5)	66(60,73)	63(56,70)	60(55,66.5)	57(55,67)	0.000
BMI (kg/m2)	24.28±3.98	25.93±3.51	26.4±3.54	26.3±3.41	26.04±3.09	0.002
Laboratory findings						
Albumin (g/L)	34.8(32.5,35.5)	38.02(37.3,38.7)	41.0(40.2,41.9)	44.2(43.4,44.9)	46.9(46.4,47.6)	0.000
HbA1c(mmol/L)	9.8(7.9,11.3)	8.8(7.4,10.8)	8.5(7.2,9.5)	7.7(6.7,8.9)	8.1(6.5,9.7)	0.000
TC (mmol/L)	4.0(3.5,4.7)	4.1(3.1,5.0)	4.18(3.2,5.1)	4.5(3.5,5.8)	4.7(3.9,6.0)	0.002
TG (mmol/L)	1.1(0.8,1.3)	1.2(0.92,1.5)	1.25(0.99,1.7)	1.2(1.0,1.8)	1.4(1.2,2.5)	0.000
HDL-c(mmol/L)	1.1(0.9,1.4)	1.2(0.9,1.4)	1.2(1.0,1.4)	1.3(1.0,1.6)	1.1(0.9,1.3)	0.034
LDL-c(mmol/L)	2.6(2.0,3.3)	2.77(1.84,3.53)	2.61(1.78,3.19)	2.41(2.10,3.40)	2.41(2.10,3.40)	0.002
Uric(mmol/L)	250.6(188.3,321)	247.1(176.1,319.3)	275.9(204.2,275.9)	294.8(229.9,357.6)	314.9(256.5,364.6)	0.002
Ca(mmol/L)	2.14(2.06,2.27)	2.24(2.19,2.28)	2.29(2.21,2.34)	2.35(2.27,2.42)	2.43(2.32,2.51)	0.000
ALP	74.0(56.1,9.6)	72.2(44.6,87.8)	69.3(47.9,87.9)	76.3(55.1,94.6)	73.4(56,100)	0.147
25(OH)D(ng/mL)	14.31(11.76,19.31)	15.34(12.17,20.61)	16.93(13.41,21.45)	19.44(15.21,26.21)	22.05(16.446,25.84)	0.000
BGP (ng/mL)	12.38(8.58,15.02)	13.32(10.26,16.69)	12.11(9.64,15.93)	13.07(10.24,17.18)	12.63(8.48,15.94)	0.124
β-CTX (ng/mL)	0.36(0.21,0.48)	0.37(0.25,0.53)	0.33(0.24,0.49)	0.36(0.24,0.54)	0.33(0.19,0.56)	0.427
P1NP (ng/mL)	40.91(28.83,51.53)	43.03(33.26,54.82)	38.68(30.10,51.67)	40.34(29.72,52.36)	37.32(27.7,51.17)	0.178
PTH (ng/mL)	29.84(22.76,40.05)	34.88(27.27,47.60)	38.17(26.08,47.05)	38.1(30.84,49.9)	35.46(30.8,48.9)	0.004
BMD						
Total lumbar(g/cm2)	0.88(0.78,0.94)	0.78(0.71,0.91)	0.92(0.79,1.02)	0.84(0.78,0.99)	0.88(0.80,0.98)	0.003
Femur neck (g/cm2)	0.56(0.50,0.66)	0.56(0.50,0.64)	0.63(0.55,0.71)	0.64(0.57,0.72)	0.65(0.60,0.73)	0.004
Total hip (g/cm2)	0.75(0.69,0.81)	0.80(0.68,0.88)	0.83(0.73,0.94)	0.85(0.75,0.96)	0.82(0.73,0.94)	0.117
Osteoporosis%	28.6%	29%	22.9%	16.6%	16.6%	0.031

Data was shown as median (25th percentile, 75th percentile) or mean ± SD. ALP （alkaline phosphatase）, BGP （osteocalcin）, β-CTX（β-collagen specific sequence）, P1NP （procollagen type I amino-terminal peptide）, PTH （parathyroid hormone）, BMD（bone mineral density）

4.2 Spearman's correlations between the Geriatric Nutritional Risk Index and bone metabolism indicators, and correlations adjusted for age, duration of disease, TC, TG, and uric acid

(Table 2) GNRI was found to be positively correlated with BMD of all bone sites ,25(OH)D and PTH and negatively correlated with PINP; after adjustment of age, duration of disease, TC, TG, and uric acid, Spearman's partial correlation analysis showed that GNRI was positively correlated with 25(OH)D and PTH, with Pearson's correlation coefficients (r) of 0.224 and 0.136; and negatively correlated with ALP and PINP with correlation coefficients (r) of -0.178 and − 0.120.

Table 2
Correlation between Geriatric Nutritional Risk Index and BMD bone and metabolic markers, and correlation after adjustment for age, duration of disease, TC, TG, and uric acid

Variables	□□□		□□□	
	r	P	r	P
ALP	0.030	0.460	-0.178	0.000
25(OH)D	0.273	0.000	0.224	0.000
OC	0.012	0.761	-0.045	0.271
β-CTX	-0.033	0.414	-0.039	0.334
PINP	-0.085	0.035	-0.120	0.003
PTH	0.132	0.001	0.136	0.001
Total lumbar BMD	0.170	0.009	0.110	0.104
Total hip BMD	0.162	0.014	0.050	0.458
Femur neck BMD	0.239	0.000	0.042	0.540

4.3 Logistic regression analysis of participants with osteoporosis

Analysis of the association between GNRI and osteoporosis using logistic regression, and the results are shown in Table 3. After adjusting for sex, age, duration of diabetes, TC, TG, uric acid, and 25(OH)D, a significant association between GNRI and osteoporosis was observed in the age < 65 years group (Odds ratio 0.917, P < 0.05), and in the age ≥ 65 years group (Odds ratio 1.062, P < 0.05).

Table 3

Logistic regression analysis of participants with osteoporosis

Variables	Age□65			Age≥65		
	SE	Odds ratio (95% CI)	P	SE	Odds ratio (95% CI)	P
Sex□male	0.365	3.102 (1.516,6.348)	0.002	0.343	6.465 (3.299,12.672)	0.000
Age	0.044	1.035 (0.949,1.128)	0.435	0.025	1.081 (1.030,1.135)	0.002
Diabetes duration	0.025	1.014 (0.966,1.064)	0.574	0.018	1.025 (0.991,1.061)	0.156
TC	0.119	0.854 (0.676,1.079)	0.186	0.108	0.823 (0.666,1.018)	0.072
TG	0.176	1.021 (0.723,1.442)	0.907	0.120	1.297 (1.025,1.642)	0.031
Uric	0.002	1.001 (0.997,1.004)	0.722	0.001	1.000 (0.997,1.003)	0.937
25□OH□D	0.021	1.031 (0.990,1.074)	0.145	0.020	0.985 (0.947,1.024)	0.443
GNRI	0.031	0.917 (0.864,0.974)	0.005	0.029	1.062 (1.003,1.125)	0.040

SE, standard error

4.4 Predictive properties of GNRI for osteoporosis

In Fig. 1, ROC curve analysis was performed with GNRI as the test variable and the presence or absence of osteoporosis as the status variable. The area under the ROC curve for GNRI was calculated to be 0.584 (P < 0.05); the optimal GNRI threshold for pre-osteoporosis was 107.2, the sensitivity was 89.63% and the specificity was 24.63%.

Discussion

Originally used as an indicator to assess nutritional status in the elderly, GNRI is calculated from albumin, weight and height and is a dual assessment of serum albumin and BMI that complements and improves the accuracy of diagnosis. Good nutritional status plays a good role in bone metabolism, Similarly, malnutrition increases the incidence of osteoporosis and fragility fractures [16]. In the present study we looked at

the correlation between GNRI and osteoporosis in northern T2DM patients and showed that there is a positive correlation between GNRI and BMD, the group with lower GNRI values had a higher prevalence of osteoporosis. Divided the participants into osteoporotic and non-osteoporotic groups, and low GNRI values in the osteoporotic group compared to those in the non-osteoporotic group, and the difference was statistically significant. In the logistic regression analysis, Significant association of GNRI with osteoporosis (dominance ratio of 0.917, $P < 0.05$ in the age < 65 group and 1.062, $P < 0.05$ in the age ≥ 65 group). Similar to the findings of Liang Wang et al, our results suggest a significant positive relationship between GNRI and osteoporosis and provide a theoretical basis for screening for osteoporosis in clinical practice.

The current mechanism of association between GNRI and osteoporosis is considered to be possibly related to the following. First, malnutrition affects calcium and vitamin D intake, which may increase bone mineral loss in patients, making it difficult to mineralize bone and leading to the development of osteoporosis. Secondly, hypoalbuminemia is a marker of both nutritional status and chronic inflammatory response; hypoalbuminemia activates osteoclasts and inhibits osteoblasts through NF-κB factors, other inflammatory cytokines [17]; hypoalbuminemia causes a decrease in insulin-like growth factor-1 synthesis, which also leads to a decrease in the number of osteoblasts and a decrease in cellular activity, increased osteoclast lifespan, increased bone resorption, and bone decreased remodeling [18].

Finally, hypoproteinemia leads to inadequate muscle synthesis and decreased skeletal muscle mass, resulting in decreased balance and gait capacity, which can cause falls as well as the occurrence of fractures [19, 20].

We evaluated the predictive effect of GNRI on osteoporosis using Roc curves. The analysis yielded a GNRI cut-off value of 107.2, whereas Liang Wang et al. derived an optimal GNRI cut-off value of 98.2 for predicting osteoporosis in men and 99.5 for predicting osteoporosis in women, and we consider that the difference in results may be due to the fact that our subjects were northern type 2 diabetic patients with a mean level of 25(OH)D of 17.26 mmol/L and were mostly vitamin D deficient or lacking, offsetting some of the positive regulatory effect of GNRI. The correlation between GNRI and 25(OH)D was found to be positive, and GNRI was independently correlated with 25(OH)D after adjusting for age, duration of disease, TC, TG, uric acid, and other influencing factors; this result may partially demonstrate that high levels of GNRI have a protective effect on bone metabolism in patients. In contrast, no correlation between GNRI and 25(OH)D in the study by Liang Wang et al. Possible reasons for our different results are that in the south even though nutritional status leads to reduced vitamin intake, vitamin D levels can still be ensured with adequate sun exposure, while in the north insufficient sun exposure combined with nutritional barriers makes it more likely to cause vitamin D deficiency and deficiency. Studies show a close relationship between latitude sunlight deficiency, skin coverage and vitamin D. In China, there is a clear geographical division of vitamin D deficiency, with populations in northern, northeastern and northwestern China north of 35 degrees north latitude being more severely undernourished, while vitamin D levels are adequate in areas south of 25 degrees north latitude and in the middle of the country [21, 22].

Our study also found that GNRI was positively correlated with PTH and negatively correlated with PINP; after adjusting for age, duration of disease, TC, TG, uric acid, and other influencing factors, GNRI was negatively correlated with PINP and ALP and positively correlated with PTH. No significant association of GNRI with osteocalcin or β-CTX was observed before or after adjustment. Our results are contrary to the findings of Liang Wang et al. During bone conversion, bone formation and bone resorption are tightly coupled. Our results yielded a negative correlation between GNRI and bone turnover markers, thus it reduces the rate of bone turnover and thus bone loss. Among alkaline phosphatases, bone-specific alkaline phosphatase is closely related to normal bone growth and development, and is a marker of maturation and activity of osteoblasts. However, the specificity of our current assay is not good, and there is some crossover with liver-derived ALP, and we measure total ALP and not BALP; therefore, the relationship of ALP does not accurately reflect the level of bone metabolism. For the relationship between GNRI and PTH, the study found a positive correlation between serum PTH and BMI, fat mass, and Mehrotra indicated that reduced PTH is a risk factor for malnutrition[23, 24]. PTH can promote the inward flow of calcium ions into adipocytes and stimulate adipose synthesis, and accordingly, low levels of PTH inhibit adipose synthesis and cause protein depletion. In this study, the lower GNRI group had lower BMI and lower PTH, and when we further performed regression analysis, we found that PTH was not a risk factor for bone loss. Therefore, PTH did not affect the predictive effect of GNRI on osteoporosis in the present study.

In this study, we found that diabetic patients with osteoporosis were older, had a longer duration of diabetes, higher glycated hemoglobin, and most patients had substandard overall glycemic control, glycated hemoglobin is not a risk factor for osteoporosis. In the regression analysis, we still concluded that GNRI was independently associated with osteoporosis after adjusting for age and duration of diabetes. Furthermore, in our regression analysis of GNRI and osteoporosis, we concluded that uric acid was not associated with osteoporosis and was neither a protective nor a risk factor for osteoporosis. Several studies have concluded that higher UA levels are protective for osteoporosis [25–27]. Our difference with the results of these studies may be due to gender, region, ethnicity, study methodology and sample size. Finally, the association between serum UA and osteoporosis may be directly or indirectly confounded by the fact that many older adults suffer from two or more chronic diseases, such as obesity, diabetes mellitus, etc.

This study has some limitations, firstly, the retrospective nature of this study, it does not provide a mechanism-related explanation for the observed association; and the study is a cross-sectional study and doesn't indicate a causal relationship between GNRI and bone mineral density. Second, the serum data of the patients with the disease, of which only one was collected, and the BMD of each location was also

collected once, thus leading to bias. Third, some relevant parameters affecting the study results may have been overlooked in this study, such as history of smoking, alcohol consumption, hormone levels, dietary habits, exercise situation, and history of previous fractures.

In summary, study results demonstrated that a lower GNRI is associated with increased osteoporosis and that GNRI is a convenient way to assess nutritional status and osteoporosis in patients with T2DM. Nutritional supplementation therapy may Reducing the incidence of osteoporosis in patients with T2DM.

Declarations

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Discloser

No conflict of interest between the authors, including Yuanyuan Ji, Nan Geng and Yingchun Niu, Hang Zhao, Wenjie Fei, Shu Chun Chen, Lu Ping Ren.

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Figures

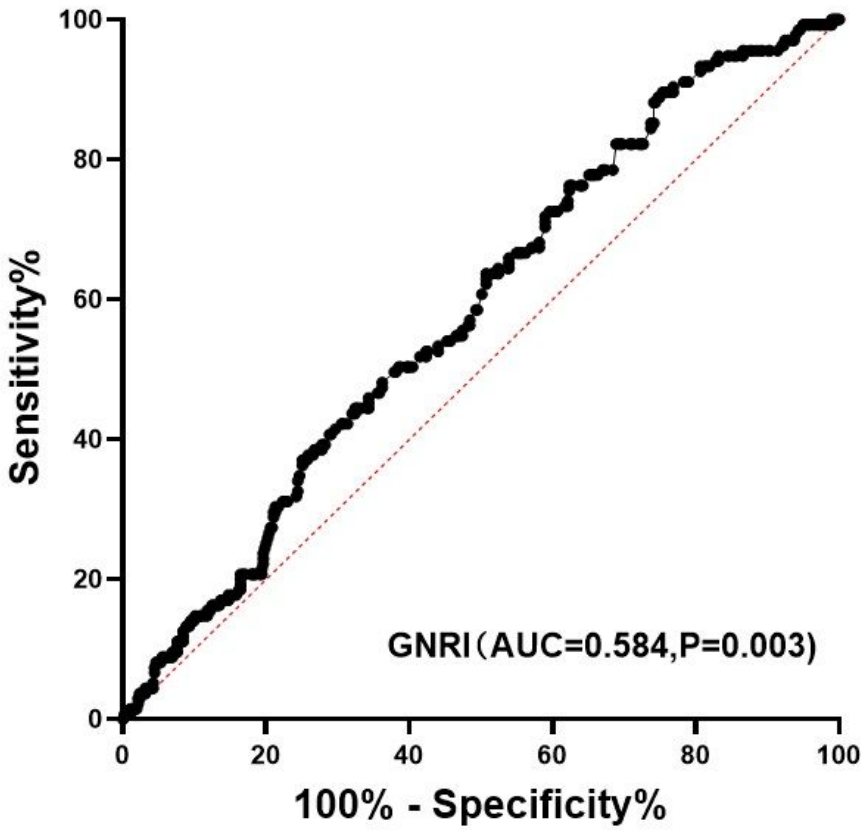

Figure 1

ROC curve analysis was performed with GNRI as the test variable and the presence or absence of osteoporosis as the status variable.