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ORIGINAL RESEARCH

Association of Central Obesity and Diabetes with Thyroid Nodules: A 3.2-year Prospective Study in Chinese Adults

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Purpose: We investigated the association of central obesity and glucose metabolism with thyroid nodule (TN) risk and explored whether diabetes/central obesity altered TN risk.

Methods: Data were collected in the Risk Evaluation of cAncers in Chinese diabeTic Individuals: a lONgitudinal study, which involved cross-sectional (May–August 2011) and longitudinal cohorts (July–October 2014; 3.2-year follow-up). In all, 8995 subjects were enrolled in the baseline survey; the 5655 subjects evaluated during the 3.2-year follow-up were included in this study. All subjects underwent body measurements, laboratory tests, and thyroid ultrasonography.

Results: Among 5655 participants, 420 had TNs. Participants with central obesity or abnormal glucose metabolism had significantly higher TN risk than participants without central obesity (OR: 1.233; 95% CI: 1.009–1.507) or abnormal glucose metabolism (OR: 1.257; 95% CI: 1.025–1.541). After adjustments for age and sex, this significance was retained for central obesity. Among participants with central obesity, prediabetes (OR: 1.483, 95% CI: 1.016–2.163) and diabetes (OR: 1.622, 95% CI: 1.078–2.407) were associated with TNs, when compared to the euglycemic subgroup (*P* trend = 0.040). No similar result was observed among participants without central obesity. During follow-up, the incidence rate ratio (IRR) for TNs was 1.026 (95% CI: 1.006–1.050) among those with new-onset central obesity and 1.025 (95% CI: 1.006–1.049) among those with chronic central obesity, as compared with those without central obesity. No significant associations were found among groups based on dynamic changes in glucose metabolism. Only in the chronic central obesity subgroup, new-onset prediabetes (IRR: 1.092, 95% CI: 1.020–1.169) and diabetes (IRR: 1.103, 95% CI: 1.031–1.189) significantly increased TN risk, as compared with euglycemia.

Conclusion: TNs are associated with central obesity and abnormal glucose metabolism. Chronic/new-onset central obesity increased TN risk. New-onset prediabetes/diabetes amplified TN risk among people with chronic central obesity.

Keywords: diabetes, glucose metabolism, obesity, thyroid nodule

Introduction

Thyroid nodules are extremely common lesions that are diagnosed in approximately 50%–60% of healthy individuals, and their incidence is growing at an alarming rate.[1] In 2020, a cross-sectional survey across China revealed that the overall prevalence of thyroid nodules (TNs) was 20.43%.[2] Advances in diagnostic tools and the widespread application of ultrasound tests partially explain the increasing incidence of thyroid nodules.[3,4] Additionally, risk factors for the development and progression of thyroid nodules in the general population include[5] age, sex, and metabolic disorders.[1,6]

Obesity and diabetes mellitus are associated with insulin resistance, especially in those with the central accumulation of body fat.[7,8] Both thyroid volume and the occurrence rate of thyroid nodules are higher in persons with insulin resistance than in persons without insulin resistance.[9,10] Furthermore, the rates of obesity and diabetes are higher among individuals with thyroid nodules,

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and both rates progressively increase with the number of nodules.[6,11,12] A recent retrospective cohort study involving Chinese participants showed that central obesity and impaired fasting glucose were risk factors for thyroid nodules.[13] However, little evidence is available on the association of diabetes mellitus and obesity with thyroid nodules, and it remains unknown whether the presence of prediabetes or diabetes modifies the association between obesity and thyroid nodules.

Therefore, we aimed to determine the association of central obesity and glucose metabolism status with the risk of thyroid nodules in a baseline cross-sectional study. As the glucose metabolism and obesity status change dynamically, we also explored whether the development of prediabetes/diabetes or central obesity is associated with an altered risk of thyroid nodules in a 3.2-year prospective longitudinal cohort study.

Methods

Study Design and Participant Selection

The present study analyzed the data collected in the Risk Evaluation of cAncers in Chinese diabeTic Individuals: a lONgitudinal study (REACTION).[14] The REACTION study was a cross-sectional study of a cohort of 10,140 individuals aged ≥ 40 years, that was conducted from May to August 2011. Three years later, the same cohort was followed up in a longitudinal study performed between July and October 2014. A flow chart of the subject-selection process is shown in Figure 1. Of the 10,140 individuals who were enrolled in the baseline survey, 8995 subjects had complete information. At the 3.2-year follow-up, another 3340 subjects were excluded due to missing data (eg, thyroid ultrasound results). Thus, finally, 5655 subjects were included in the present study.

Data Collection, Body Measurements, and Laboratory Tests

All subjects completed a standard questionnaire with the guidance of a staff member trained in conducting epidemiological screening. Body weight, height, and waist circumference (WC) were measured on a calibrated scale. The body mass index (BMI) was determined by dividing the weight in kilograms by the square of the height in meters. Blood pressure was measured 3 times with an electric sphygmomanometer (OMRON) after a resting period of at least 15 min, and the average of the 3 measurements was used in the subsequent analyses.

Blood samples were drawn from the subjects after 10 h of fasting, and stored at −80°C until analysis. Oral glucose tolerance tests were performed with 75 g of glucose, and the fasting plasma glucose (FPG) and 2-h plasma glucose (2hPG) concentrations were measured using the hexokinase method. In addition, the fasting insulin level and the lipid profile, including triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) levels, were measured on an auto-analyzer (c16000 system, ARCHITECT ci16200 analyzer, Abbott Laboratories, Abbott Park, Illinois, USA). The Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) score was derived using the following equation: FPG (mmol/L) × FINS (mU/mL)/22.5, where FINS stands for the fasting insulin level.[15] Thyroid ultrasonography was performed with a 7.5-MHz linear probe. Experienced physicians conducted the examination on patients lying in a supine position with the neck hyperextended, and recorded the number of thyroid nodules and their locations.

Study Definitions

According to the 2020 Guidelines for the Prevention and Treatment of Type 2 Diabetes Mellitus in China,[16] we defined diabetes mellitus as an FPG concentration ≥ 7.0 mmol/L, a 2hPG concentration ≥ 11.1 mmol/L, or a prior diagnosis of diabetes. We defined prediabetes as FPG ≥ 6.1 mmol/L but < 7.0 mmol/L and/or 2hPG ≥ 7.8 mmol/L but < 11.1 mmol/L. Glucose metabolism was considered to be normal in subjects with FPG < 6.1 mmol/L, 2hPG < 7.8 mmol/L, and no prior diagnosis of diabetes. Glucose metabolism was deemed as abnormal in subjects with FPG ≥ 6.1 mmol/L, 2hPG ≥ 7.8 mmol/L, or a prior diagnosis of diabetes. Central obesity was defined as WC ≥ 90 cm in men and ≥ 85 cm in women.

In the longitudinal cohort study, the participants were stratified according to their central obesity and glucose metabolism status at the baseline and at the 3.2-year follow-up. The groups based on central obesity were as follows: no central obesity (participants without central obesity throughout the study), new-onset central obesity (participants who newly developed central obesity during the study), central obesity recovery (participants who recovered from preexisting

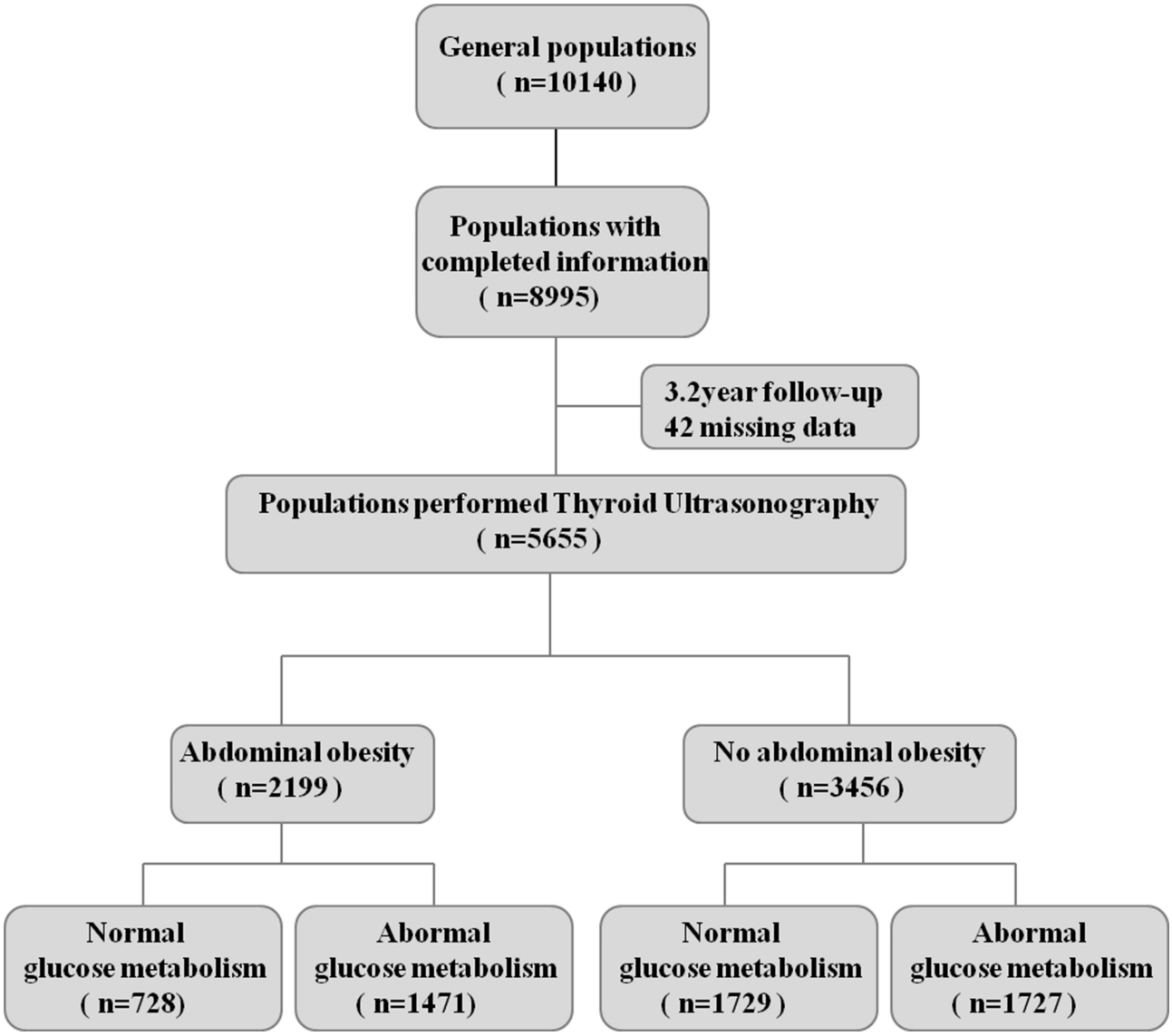

Figure 1 Flow chart showing the participant-selection process in the present study.

central obesity), and chronic central obesity (participants with central obesity throughout the study). The groups based on glucose metabolism were as follows: normal glucose metabolism (participants with consistently normal glucose metabolism throughout the study), prediabetes recovery (participants who recovered from preexisting prediabetes), chronic prediabetes or diabetes (participants with prediabetes or diabetes throughout the study), new-onset prediabetes (participants who newly developed prediabetes during the study), and new-onset diabetes (participants who newly developed diabetes during the study).

Statistical Analysis

All statistical analyses were conducted using SPSS (version 17.0). The baseline characteristics of the study subjects stratified according to glucose metabolism and central obesity status were expressed as means and standard deviations in the case of continuous variables and compared between groups by using analysis of variance, whereas categorical variables were presented as numbers with percentages and compared using the χ^2 test.

In the baseline cross-sectional cohort study, the multivariate linear regression model was used to determine whether the prevalence of thyroid nodules was correlated with various clinical parameters. Binary logistic regression was used to

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determine the odds ratios (ORs) with 95% confidence intervals (CIs) for the association of central obesity and glucose metabolism status with the prevalence of thyroid nodules. In the longitudinal cohort study, Poisson regression was used to determine the incidence rate ratios (IRRs) of the association of central obesity and glucose metabolism status with the risk of thyroid nodules. Multiplicative interaction models were used to determine the effects of the interaction between central obesity and glucose metabolism status on the incidence of thyroid nodules. P (two-tailed) < 0.05 was considered to indicate statistical significance.

Results

General Characteristics of the Subjects and Risk Factors for Thyroid Nodules in the Baseline Cross-Sectional Cohort

The baseline characteristics of the subjects stratified by their central obesity and glucose metabolism statuses are listed in Table 1. Of the 5655 subjects, 728 subjects had central obesity and normal glucose metabolism, 1471 subjects had central obesity and abnormal glucose metabolism, 1729 subjects had no central obesity and normal glucose metabolism, and 1727 subjects had no central obesity and abnormal glucose metabolism. A total of 420 (7.4%) subjects had thyroid nodules. Among the above 4 groups, the group with central obesity and abnormal glucose metabolism showed high FPG, 2hPG, fasting insulin, TC, TG, and LDL-C levels; increased BMI, WC, systolic blood pressure (SBP), diastolic blood pressure (DBP), and HOMA-IR scores; and low HDL-C levels. This group also showed the highest proportion of thyroid nodules. Multivariate logistic regression analysis showed that in the baseline cross-sectional cohort, age, WC, and 2hPG level were independently associated with the presence of thyroid nodules (Table 2).

Next, binary logistic regression was performed to determine the association of central obesity and glucose metabolism status with the prevalence of thyroid nodules. Compared with subjects without central obesity, those with central obesity were at a significantly higher risk for thyroid nodules (OR: 1.233; 95% CI: 1.009–1.507), and this risk remained significant after adjustments for age and sex (Table 3). Subjects with abnormal glucose metabolism also had

Table 1 Baseline Characteristics of Participants Stratified by Central Obesity and Glucose Metabolism Status

Glucose Metabolism	Central Obesity		No Central Obesity		*P**
	Normal	Abnormal	Normal	Abnormal	
Number of Subjects, %	728 (12.8)	1471 (26.0)	1729 (30.6)	1727 (30.6)	
Sex, F (%)	595 (81.7)	1094 (74.4)	1357 (78.5)	1069 (61.9)	<0.001
Age, y	56.20 ± 7.71	60.17 ± 7.80	55.59 ± 7.56	59.50 ± 7.64	<0.001
FPG, mmol/L	5.5 ± 0.3	6.8 ± 1.9	5.5 ± 0.3	6.6 ± 1.9	<0.001
2hPG, mmol/L	6.4 ± 0.9	10.8 ± 1.6	6.3 ± 0.9	10.1 ± 3.8	<0.001
BMI, kg/m^2	25.9 ± 2.6	26.5 ± 2.7	22.0 ± 2.3	22.8 ± 2.4	<0.001
WC, cm	91.1 ± 4.9	92.6 ± 5.5	77.9 ± 5.6	79.9 ± 5.5	<0.001
SBP, mm Hg	119.5 ± 17.4	128.3 ± 17.1	113.8 ± 16.6	124.0 ± 18.2	<0.001
DBP, mm Hg	77.8 ± 10.5	80.5 ± 10.5	73.1 ± 20.2	77.5 ± 10.3	<0.001
Fasting insulin, mU/mL	8.8 ± 3.6	11.3 ± 9.1	6.4 ± 2.7	8.3 ± 6.6	<0.001
HOMA-IR score	2.0 ± 0.1	2.5 ± 0.7	2.0 ± 0.1	2.4 ± 0.7	<0.001
TC, mmol/L	4.5 ± 1.2	4.7 ± 1.2	4.4 ± 1.2	4.6 ± 1.3	<0.001
TG, mmol/L	1.7 ± 1.2	2.1 ± 1.3	1.4 ± 0.9	1.7 ± 1.3	<0.001
LDL-C, mmol/L	2.6 ± 0.8	2.7 ± 0.9	2.5 ± 0.9	2.6 ± 0.9	<0.001
HDL-C, mmol/L	1.2 ± 0.4	1.2 ± 0.3	1.3 ± 0.4	1.2 ± 0.4	<0.001
TNs, N (%)	46 (6.3)	137 (9.3)	113 (6.5)	124 (7.1)	<0.001

Note: *P values for comparisons among the 4 groups.

Abbreviations: F, female; FPG, fasting plasma glucose; 2hPG, plasma glucose concentration at 2 h after a 75-g oral glucose tolerance test; BMI, body mass index; WC, waist circumference; SBP, systolic blood pressure; DBP, diastolic blood pressure; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; TC, total cholesterol; TG, triglyceride; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol; TN, thyroid nodule.

Table 2 Multivariate Analysis of Potential Risk Factors for Thyroid Nodules in the Baseline Cross-Sectional Cohort Study

	OR (95% CI)	*P*
Age	1.121 (1.022–1.236)	0.005
WC	1.061 (1.006–1.068)	0.021
2hPG	1.014 (1.003–1.064)	0.047

Abbreviations: OR, odds ratio; CI, confidence interval; WC, waist circumference; 2hPG, plasma glucose concentration at 2 h after a 75-g oral glucose tolerance test.

Table 3 Association of Glucose Metabolism with Prevalence of Thyroid Nodules Among Participants with and without Central Obesity in the Baseline Cohort Study

	Subjects	Cases	Unadjusted Model		Adjusted Model	
			OR (95% CI)	*P*	OR (95% CI)	*P*
No central obesity	3456		1.00 (ref)		1.00 (ref)	
Central obesity	2199		1.233 (1.009–1.507)	0.031	1.119 (1.006–1.478)	0.049
Normal glucose metabolism	2457	161	1.00 (ref)		1.00 (ref)	
Abnormal glucose metabolism	3198	259	1.257 (1.025–1.541)	0.028	1.155 (0.935–1.427)	0.182
No central obesity and normal glucose metabolism	1729	113	1.00 (ref)		1.00 (ref)	
Central obesity and normal glucose metabolism	728	48	1.009 (0.712–1.423)	0.925	0.993 (0.700–1.409)	0.969
No central obesity and abnormal glucose metabolism	1727	124	1.106 (0.849–1.441)	0.454	1.022 (0.779–1.339)	0.454
Central obesity and abnormal glucose metabolism	1471	137	1.445 (1.114–1.875)	0.006	1.306 (1.000–1.706)	0.050

Note: Adjusted for age and sex in all participants.
Abbreviations: OR, odds ratio; CI, confidence interval.

a significantly higher risk of thyroid nodules than those with normal glucose metabolism (OR: 1.257; 95% CI: 1.025–1.541), but the significance of this risk disappeared after adjustments for age and sex. Individuals with both central obesity and abnormal glucose metabolism had the highest risk of thyroid nodules among the 4 groups (OR: 1.306; 95% CI: 1.000–1.706). A significant interaction effect of central obesity and glucose metabolism status was identified on the prevalence of thyroid nodules. Among persons with central obesity, the ORs for thyroid nodules in those with prediabetes or diabetes, as compared to those with normal glucose metabolism, were 1.483 (95% CI: 1.016–2.163) and 1.622 (95% CI: 1.078–2.407) respectively (p for trend = 0.040; Figure 2). A similar result was not observed among persons without central obesity.

Risk of Thyroid Nodules According to Dynamic Changes in Central Obesity and Glucose Metabolism Status

In the 3.2-year follow-up study, the IRRs for thyroid nodules were 1.026 (95% CI: 1.006–1.050) in those with new-onset central obesity and 1.025 (95% CI: 1.006–1.049) in those with chronic central obesity, as compared with subjects with no central obesity throughout the study (Table 4). Similar results were obtained after adjustments for age and sex. No significant results were found among the groups based on dynamic changes in glucose metabolism.

The associations of the different changes in central obesity and glucose metabolism status with the incidence of thyroid nodules are presented in Table 5. A significant interaction effect was found only among subjects with chronic central obesity. In this subgroup, those who developed prediabetes or diabetes had a significantly higher risk of developing thyroid nodules than did those who had normal glucose metabolism throughout the study (prediabetes: IRR = 1.092, 95% CI = 1.020–1.169; diabetes: IRR = 1.103, 95% CI = 1.031–1.189). Moreover, the above findings were not altered after adjustments for age and sex. Notably, a similar risk of abnormal glucose metabolism for the incidence of

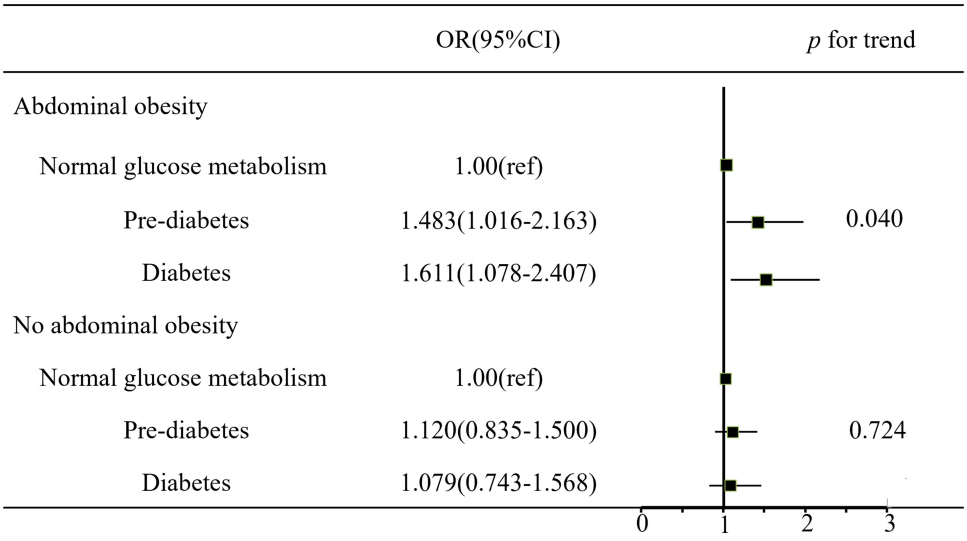

Figure 2 Interactional association of glucose metabolism and central obesity with thyroid nodules.

thyroid nodules was not observed in the no central obesity, new-onset central obesity, and central obesity recovery subgroups.

Discussion

In the cross-sectional cohort study, we found that central obesity and pre-diabetes/diabetes were associated with a high risk of thyroid nodules, which is consistent with previous studies.[6,9,10,12] We additionally found that abnormal glucose metabolism increases the risk of thyroid nodules among individuals with central obesity. In the longitudinal cohort study, we confirmed that new-onset central obesity and chronic central obesity increase the incidence of thyroid nodules. An interactional relationship between chronic central obesity and new-onset prediabetes/diabetes on the risk of the incidence of thyroid nodules was also found in the present study. In the chronic central obesity subgroup, subjects who developed prediabetes or diabetes during the study had a significantly higher risk of developing thyroid nodules.

Obesity is linked to thyroid disease, as it promotes an increased thyroid volume and higher serum levels of thyroid-stimulating hormone.[4,8,17,18] Numerous clinical studies have demonstrated that thyroid nodules are strongly associated with markers of obesity, such as body weight, BMI, and WC.[19] BMI is commonly used to evaluate obesity, but WC appears to be a more relevant parameter for assessing metabolic health.[20] Therefore, we used the WC to analyze the

Table 4 Risk of Thyroid Nodules According to Dynamic Changes in Central Obesity and Glucose Metabolism Status in the Longitudinal Cohort Study

	Subjects	Cases	Unadjusted Model IRR (95% CI)	Adjusted Model IRR (95% CI)
No central obesity	1973	250	1.00 (ref)	1.00 (ref)
New-onset central obesity	1483	226	1.026 (1.006–1.050)	1.022 (1.001–1.046)
Central obesity recovery	592	69	0.990 (0.961–1.020)	0.987 (0.958–1.017)
Chronic central obesity	1607	244	1.025 (1.006–1.049)	1.026 (1.002–1.050)
Normal glucose metabolism	1503	191	1.00 (ref)	1.00 (ref)
Prediabetes recovery	847	115	1.009 (0.980–1.038)	1.003 (0.974–1.032)
Chronic prediabetes/diabetes	2016	289	1.016 (0.994–1.040)	1.008 (0.984–1.032)
New-onset prediabetes	701	110	1.030 (0.998–1.064)	1.011 (0.978–1.045)
New-onset diabetes	588	84	1.016 (0.983–1.050)	1.028 (0.996–1.062)

Note: Adjusted for age and sex in all participants.
Abbreviations: IRR, incidence rate ratios; CI, confidence interval.

Table 5 Association of Changes in Central Obesity and Glucose Metabolism Status with the Incidence of Thyroid Nodules in the Longitudinal Cohort Study

	Subjects	Cases	Unadjusted Model IRR (95% CI)	Adjusted Model IRR (95% CI)
No central obesity				
Normal glucose metabolism	710	82	1.00 (ref)	1.00 (ref)
New-onset prediabetes	213	29	1.021 (0.927–1.075)	1.016 (0.964–1.071)
New-onset diabetes	124	12	0.981 (0.807–1.029)	0.966 (0.911–1.026)
New-onset central obesity				
Normal glucose metabolism	388	56	1.00 (ref)	1.00 (ref)
New-onset prediabetes	249	40	1.016 (0.960–1.077)	1.019 (0.962–1.080)
New-onset diabetes	222	30	0.991 (0.936–1.049)	0.995 (0.940–1.054)
Central obesity recovery				
Normal glucose metabolism	114	11	1.00 (ref)	1.00 (ref)
New-onset prediabetes	65	8	1.027 (0.932–1.131)	1.026 (0.931–1.131)
New-onset diabetes	45	8	1.056 (0.945–1.179)	1.062 (0.945–1.193)
Chronic central obesity				
Normal glucose metabolism	291	38	1.00 (ref)	1.00 (ref)
New-onset prediabetes	174	33	1.092 (1.020–1.169)	1.089 (1.017–1.165)
New-onset diabetes	189	38	1.103 (1.031–1.189)	1.095 (1.022–1.172)

Note: Adjusted for age and sex in all participants.
Abbreviations: IRR, incidence rate ratio; CI, confidence interval.

association between central obesity and thyroid nodules, and found that WC was an independent risk factor for thyroid nodules, which is consistent with previous results.[8,12,18] Furthermore, during the 3.2-year follow-up, we confirmed an elevated risk of new-onset thyroid nodules among subjects with chronic or newly developed central obesity. Central obesity is associated with the development of metabolic syndrome, the central link of which is insulin resistance, which is often accompanied by elevated insulin levels. The association between central obesity and a higher risk of thyroid nodules may be attributable to elevated circulating levels of insulin. Insulin stimulates follicular-cell proliferation in the thyroid and promotes the development and growth of thyroid nodules.[10,21,22] Nevertheless, no significant decrease in the risk of thyroid nodules was detected among subjects who recovered from central obesity, possibly because they continued to exhibit relatively higher insulin concentrations than those who never had central obesity.

Abnormal glucose metabolism and type-2 diabetes are associated with insulin resistance. Diabetes has been reported to show a significant positive association with thyroid nodules.[8,23–25] Patients with type-2 diabetes often exhibit insulin resistance and hyperinsulinemia, and the abnormal glucose metabolism and changes in hormone levels in these patients lead to abnormal thyroid morphology.[26] Patients with insulin resistance have an increased thyroid volume, which promotes thyroid-cell proliferation and leads to the formation of thyroid nodules.[27] In middle-aged and elderly type-2 diabetes patients, age, gender, and insulin resistance are independent risk factors for thyroid nodules.[26,28] Consistent with previous results, we found that participants with abnormal glucose metabolism showed a higher prevalence of thyroid nodules, and the 2hPG level was an independent risk factor for thyroid nodules. However, during the longitudinal cohort study, we did not any association between thyroid nodules and dynamic changes in the glucose metabolism status, such as chronic and new-onset prediabetes/diabetes. A longer follow-up may be needed to evaluate the long-term effects of the dynamic changes in glucose metabolism status on the risk of thyroid nodules.

Metabolic syndrome is characterized by a cluster of metabolic risk factors, such as hyperglycemia, dyslipidemia, central obesity, and hypertension.[22] Studies have revealed that metabolic syndrome is associated with a high risk of thyroid nodules, which suggests that obesity, which is associated with metabolic abnormalities, would have an even greater influence on the risk of thyroid nodules.[22,24] However, few studies have focused on the effects of glucose metabolism and central obesity, and especially their interaction, on the risk of thyroid nodules. In this study, we identified that among participants with both central obesity and prediabetes/diabetes, the deleterious effects of each factor on the

risk of thyroid nodules were amplified. In the longitudinal cohort study, new-onset prediabetes or diabetes amplified the risk of chronic central obesity on thyroid nodules. The precise mechanism underlying our observations is as yet unclear. Both diabetes and obesity contribute to the incidence of thyroid nodules by increasing the insulin concentration. Therefore, we suggest that high insulin concentrations among people with chronic central obesity may be a plausible explanation. Thus, we believe that it is important to assess obesity-related factors, such as insulin-like growth factor and leptin, in patients who develop diabetes in a follow-up study. Moreover, in this study, we did not find a correlation between low HDL-C and thyroid nodules, which is inconsistent with other conclusions.[29]

The strengths of this study include its prospective study design and the analysis of the interactional association of central obesity and glucose metabolism with thyroid nodules. The limitations of this study include the relatively small sample size, especially in the longitudinal cohort study; the lack of assessment of obesity-related factors to explain the observed results; and the relatively older age of the participants as the occurrence of thyroid nodules is related to age. Most subjects were middle-aged or elderly Chinese people, which may have caused some deviation in the results.

Conclusion

In conclusion, the prevalence of thyroid nodules in our study was 7.4%. Age, WC, and 2hPG level were risk factors for thyroid nodules. During the 3.2-year follow-up, we found that chronic and new-onset central obesity increased the risk of thyroid nodules, and new-onset prediabetes/diabetes amplified the risk of thyroid nodules among people with chronic central obesity.

Data Sharing Statement

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

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

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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