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ORIGINAL RESEARCH

MRI-Measured Pancreatic Fat Correlates with Increased Arterial Stiffness in Patients Who are Overweight and Obese

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Purpose: Arterial stiffness is often increased in overweight or obese individuals before the development of hypertension (HT). This study aimed to determine the connection between pancreatic fat and atherosclerosis in overweight and obese people without HT.

Patients and methods: We included 128 patients who were non-hypertensive and overweight or obese in a study between December 2019 and November 2022. Medical history was collected, and all participants underwent a physical examination and blood tests. Pancreatic fat content was measured by magnetic resonance imaging (MRI) and was grouped into quartiles based on pancreatic fat fraction (PFF). The upper three quartiles (PFF≥10.33%) were defined as non-alcoholic fatty pancreas disease (NAFPD) and the first quartile (PFF<10.33%) as non-NAFPD. High baPWV (H-baPWV) and low baPWV (L-baPWV) were classified according to the median baPWV (1159 cm/s). The effect of NAFPD on baPWV was examined using binary logistic regression. The study population consisted of 96 NAFPD and 32 non-NAFPD cases.

Results: Participants with NAFPD had significantly higher levels of baPWV than people without. The rates of NAFPD and the PFF values varied significantly in the L-baPWV and H-baPWV groups. Logistic regression analysis suggested that the presence of NAFPD was independently correlated with increased baPWV after adjusting for age, smoking, body mass index, blood pressure, lipid profiles, and glycemic index.

Conclusion: NAFPD is an independent risk factor for increased baPWV in individuals with overweight and obesity but no HT, suggesting that the presence of NAFPD may be a warning signal of early atherosclerosis.

Keywords: obesity, non-alcoholic fatty pancreas disease, overweight, arterial stiffness, brachial-ankle pulse wave velocity

Introduction

Obesity is an independent risk factor for cardiovascular disease (CVD) and influences the process of other cardiovascular risk factors such as dyslipidemia, hypertension (HT), and type 2 diabetes mellitus (T2DM).[1] An expanding corpus of studies indicates that compared with subcutaneous fat, visceral fat is a more reliable indicator of cardiovascular risk.[2,3] Non-alcoholic fatty pancreas disease (NAFPD), a type of visceral obesity, is also correlated with CVD risk factors.[4] However, whether NAFPD directly contributes to an increased risk of CVD is unknown.

Few studies have shown that NAFPD is related to subclinical atherosclerosis. Kim et al found that computed tomography (CT)-assessed fatty pancreas was directly correlated with higher carotid intima-media thickness (IMT) and brachial-ankle pulse wave velocity (baPWV) in individuals with no obesity and T2DM but not in patients with obesity and T2DM.[5] Ozturk et al and Sahin et al reported that pancreatic fat accumulation measured using

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transabdominal ultrasonography was associated with higher carotid IMT among people with non-alcoholic fatty liver disease (NAFLD).[6,7] Ultrasonography-determined NAFPD and elevated aortic IMT were also reported to be independently associated in a Turkish study.[8] Moreover, a Chinese study suggested that CT-estimated pancreatic fat content correlated with carotid plaque in patients with T2DM.[9] Yet another study found a direct link between carotid artery calcification and pancreatic fat.[10] However, the direct connection between NAFPD and atherosclerosis became weaker after adjusting for confounding factors. Additionally, this association is further complicated by differences in the methods for quantifying pancreatic fat and the characteristics of the individuals. Hence, further research in this field is required.

Aim

In the present research, we sought to analyze the connection between baPWV and pancreatic fat quantified using magnetic resonance imaging (MRI) in individuals with non-hypertension and overweight or obesity. MRI, which is non-invasive, nonradiative, and highly accurate, is regarded as the best imaging tool for quantifying intrapancreatic fat deposition in clinical practice.[11,12] Arterial stiffness, one of the early markers of increased CVD risk, frequently increases in patients who are overweight or obese prior to the onset of hypertension.[13] Therefore, our study population included individuals without hypertension to exclude the interference of hypertension in atherosclerosis.[14,15]

Methods

Study Population

This research adopted baseline data of patients with overweight and obesity attending the weight-loss clinic of the affiliated Wuxi People's Hospital of Nanjing Medical University for individualized multidisciplinary weight management (ChiCTR1900022948). This research was authorized by the hospital's ethics committee and carried out in line with the Declaration of Helsinki (KS2019020). Each participant provided written informed consent.

One hundred twenty-eight eligible participants were enrolled in this study between December 2019 and November 2022. The requirements for inclusion were: age between 18–65 years, body mass index (BMI) ≥ 28.0 kg/m^2 or ≥ 24.0 kg/m^2 with one or more comorbidities (dyslipidemia, sleep apnea, or abnormal glucose metabolism), and steady weight over the past 3 months. The Working Group on Obesity in China recommends a BMI of 24.0 kg/m^2 as the cutoff point for overweight and a BMI of 28.0 kg/m^2 as the cutoff point for obesity.[16] Individuals with the following conditions were excluded: (1) medical history of HT or a baseline systolic blood pressure (SBP) ≥140 mmHg or diastolic blood pressure (DBP) ≥90 mmHg; (2) secondary obesity (such as Cushing's syndrome and hypothyroidism) or drug usage to change fat metabolism; (3) consumption of alcohol per day: intake >10g in females and >20g in males (Supplementary Table); (4) pancreatitis, pancreatic cancer, or any other types of pancreatic illnesses; (5) medical history of malignancy, cardiovascular disease, or severe liver or kidney dysfunction; and (6) inappropriate for MRI examination (metallic substance implanted in the body, a history of claustrophobia, or pregnancy).

Medical history was gathered from each participant, as well as physical examination, blood tests, arterial stiffness testing, and pancreatic MRI. These tests were completed within 7 days of the first visit.

Anthropometric and Laboratory Measurements

All clinical evaluations were conducted by qualified personnel following recognized procedures. Body weight and height were recorded using a calibrated scale with participants dressed in light attire and barefoot. BMI was computed as follows: weight (kg)/height squared (m^2). Waist circumference (WC) was determined by measuring the midpoint of the lowest rib and the superior border of the iliac crest with a regular tape measure. Waist-to-height ratio (WHtR) was computed as follows: WC (cm)/height (cm). After a 15-min rest period, SBP and DBP were assessed twice using a mercury gravity manometer with the individual in the seated position.

After fasting for at least 10 h overnight, venous blood samples were gathered to measure total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), alanine aminotransferase(ALT); aspartate aminotransferase(AST), low-density lipoprotein cholesterol (LDL-C), and fasting blood glucose (FBG) levels using photometric assays (Chemistry Immunoanalyzer AU5800, Beckman Coulter, USA). Glycosylated hemoglobin (HbA1c)

levels were measured using high-pressure liquid chromatography (VARIANT II Hemoglobin Testing System, BIORAD, USA).

Assessment of baPWV

baPWV, which can be measured by a straightforward and nonintrusive technique, reflects arterial stiffness and was determined using a BP-203RPE III detection device (Omron Health Medical Co., Ltd., China). The detection procedure was performed by trained nurses following the manufacturer's recommendations. Cigarettes, alcohol, tea, and coffee were prohibited for 30 min before testing. After more than 5 min of rest, the participants underwent baPWV measurements in the supine position. Both arms and ankles were fitted with blood pressure cuffs that were linked to plethysmographic and oscillometric pressure sensors. A heart sound detector was positioned at the left margin of the sternal border. This procedure was performed twice for each participant, and the results from the second examination were valuable. The mean baPWV values of the left and right sides were used in the analysis. Participants were separated into high baPWV (H-baPWV) and low baPWV (L-baPWV) groups based on the median value of baPWV (1159 cm/s).

Assessment of NAFPD

The six-point Dixon technique was used to determine pancreatic fat content using a 3T MRI system (SIEMENS 3.0T MAGNETOM Prisma) with an eighteen-channel body array coil. The imaging parameters were as follows: TE1, 2.38 ms; TE2, 4.76 ms; TE3, 7.15 ms; TE4, 9.53 ms; TE5, 11.91 ms; TE6, 14.29 ms; TR, 15.60 ms; flip angle, 4°; FOV, 420 mm × 420 mm, and a 3.5-mm slice thickness. The Philips IntelliSpace Portal software was used with MRI workstations for post-processing of the mDixon sequence images. A trained radiologist manually drew the outline of the entire pancreas at each level of the proton density fat fraction (PDFF) maps to calculate the average pancreatic fat fraction (PFF). Based on the PFF, pancreatic fat was divided into quartiles. The 25th percentile corresponds to a PFF value of 10.33%. The first quartile of PFF (PFF<10.33%) was considered non-NAFPD and the upper three quartiles (PFF≥10.33%) were defined as NAFPD.

Statistical Analysis

In this cross-sectional study, the descriptive data are displayed as means ± standard deviations or medians (first quartile, third quartile). The chi-square statistical test was used for categorical variables, whereas Student's *t*-test or the Mann–Whitney *U*-test was used for continuous variables. Spearman correlation analysis was used to evaluate the relationship between baPWV and many variables. To assess the effect of NAFPD on baPWV, odds ratios (OR) and 95% confidence intervals (CI) were calculated by binary logistic regression analysis. Statistical analyses were conducted using SPSS software version 27.0 (Armonk, NY: IBM Corp). A *P*-value of less than 0.05 was considered significant.

Results

Demographic and Clinical Characteristics of Study Participants with the Presence or Absence of NAFPD

As shown in Table 1, individuals with NAFPD tended to be male, smokers, and older. In the NAFPD group, the levels of ALT and AST were 1.7 and 1.3 times greater, respectively, than those in the non-NAFPD group. This group also had higher BMI, WC, WHtR, and HbA1c. Moreover, compared with individuals without NAFPD, those with NAFPD had higher baPWV values (1159.68 [1087.00, 1249.62] vs 1109.50 [1032.37, 1167.79] cm/s, P=0.032). However, lipid levels (TG, TC, LDL-C, HDL-C, TG/HDL-C), FBG levels, HOMA-IR, prevalence of diabetes, and blood pressure (SBP, DBP) did not differ significantly between the two groups.

Demographic and Clinical Characteristics of Study Participants According to baPWV Value

As shown in Table 2, the proportion of patients with diabetes in the H-baPWV group was nearly four times higher than that in the L-baPWV group; further, the proportion of smokers in the H-baPWV group was twice that in the L-baPWV

Table 1 Characteristics of Study Participants with and without NAFPD

Variables	Non-NAFPD (n = 32)	NAFPD (n = 96)	P
Age (years)	29.88±6.24	33.44±7.74	0.022
Male (n, %)	8 (25.0%)	51 (53.1%)	0.006
DM (n, %)	2 (6.3%)	19 (19.8%)	0.073
Currently smoking (n, %)	2 (6.3%)	25 (26.0%)	0.027
BMI (kg/m^2)	30.73±4.12	32.68±4.55	0.035
WC (cm)	95.63±11.34	103.99±11.62	<0.001
WHtR	0.58±0.06	0.62±0.07	0.008
SBP (mmHg)	122.53±11.30	123.73±9.62	0.559
DBP (mmHg)	72.44±8.14	74.09±7.51	0.293
TG (mmol/L)	1.43 (1.00,2.89)	1.89 (1.39,2.43)	0.122
TC (mmol/L)	4.92 (4.57,5.79)	5.11 (4.46,5.71)	0.834
LDL-C (mmol/L)	2.87 (2.42,3.38)	3.01 (2.54,3.52)	0.425
HDL-C (mmol/L)	1.11 (0.92,1.33)	1.05 (0.94,1.17)	0.162
TG/HDL-C	1.45(0.69,3.08)	1.80(1.19,2.52)	0.112
ALT (U/L)	21.45(16.68,43.05)	36.95(22.15,57.13)	0.018
AST (U/L)	20.00(18.00,28.00)	25.5(19.00,38.00)	0.045
FBG (mmol/L)	5.27 (4.80,5.79)	5.37 (4.89,6.59)	0.198
HbA1c (%)	5.33 (5.10,5.68)	5.60 (5.30,6.21)	0.006
HOMA-IR	4.39(3.13, 7.02)	5.21(3.53,7.87)	0.235
BaPWV (cm/s)	1109.50 (1032.37, 1167.79)	1159.68 (1087.00, 1249.62)	0.032

Abbreviations: NAFPD, non-alcoholic fatty pancreas disease; DM, diabetes mellitus; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triacylglycerol; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase; FBG, fasting blood glucose; HbA1c, glycosylated hemoglobin; HOMA-IR, Homeostasis Model Assessment of Insulin Resistance; baPWV, brachial-ankle pulse wave velocity.

group. Participants in the H-baPWV group were 1.2 times older than those in the L-baPWV group, and TG and AST levels were both 1.3 times higher than those in the L-baPWV group. The H-baPWV group also had higher SBP, DBP, TG/HDL-C, HbA1c, and FBG levels. However, the difference in sex distribution, BMI, WC, WHtR, TC, LDL-C, HDL-C, ALT, or HOMA-IR between the two groups was not statistically significant.

The univariate analysis showed a positive association between baPWV and age (r=0.317, P<0.001), SBP (r=0.239, P=0.007), DBP (r=0.303, P<0.001), HbA1c (r=0.194, P=0.029), and FBG (r=0.225, P<0.011). However, there was no association between baPWV and BMI, ALT, AST, HOMA-IR, and lipid profiles. (Table 3)

Relationship Between NAFPD and baPWV

Comparisons between the H-baPWV and L-baPWV groups revealed that individuals with H-baPWV had higher PFFs (14.76 [11.85,17.52] vs.12.43 [8.64,15.74] %, *P*=0.003) as well as a greater prevalence of NAFPD (85.1 vs 63.9%, *P*=0.006) (Figure 1A). Furthermore, the presence of NAFPD was strongly related with baPWV (r=0.191, P=0.031) (Figure 1B).

Impact of NAFPD on baPWV After Adjusting for Confounding Factors

The impact of pancreatic fat on atherosclerosis was evaluated using logistic regression analysis. Individuals with NAFPD, compared with those without, had an OR of 3.22 (95% CI: 1.38–7.53, *P*=0.007) for H-baPWV. After adjusting for age, smoking, BMI, BP, lipid profiles, hepatic enzymes, FBG, HOMA-IR, and HbA1c, the ORs of NAFPD on baPWV were slightly attenuated, but remained significant (Table 4).

Discussion

This study demonstrated that people with overweight and obesity but no HT, with MRI-proven NAFPD, had increased baPWV compared with those without NAFPD. In contrast, individuals with H-baPWV had a higher PFF than those with

Table 2 Characteristics of Study Participants According to baPWV

Variables	L-baPWV (n=61)	H-baPWV (n=67)	P
Age (years)	30.07±6.21	34.81±8.13	<0.001
Male (n, %)	24 (39.3%)	35 (52.2%)	0.114
DM (n, %)	4 (6.6%)	17 (25.4%)	0.004
Current smoking (n, %)	8 (13.1%)	19 (28.4%)	0.035
BMI (kg/m^2)	32.55±5.03	31.85±3.99	0.385
WC (cm)	101.34±14.09	102.42±9.97	0.622
WHtR	0.60±0.07	0.61±0.07	0.576
SBP (mmHg)	120.77±10.65	125.86±8.84	0.004
DBP (mmHg)	70.95±7.22	76.16±7.26	<0.001
TG (mmol/L)	1.55 (1.09,2.24)	2.01 (1.55,2.81)	0.008
TC (mmol/L)	4.88 (4.52,5.48)	5.15 (4.47,6.16)	0.120
LDL-C (mmol/L)	3.06 (2.46,3.48)	2.90 (2.54,3.60)	0.762
HDL-C (mmol/L)	1.09 (0.96,1.23)	1.03 (0.93,1.19)	0.296
TG/HDL-C	1.44(0.98,2.19)	2.04(1.20,3.14)	0.009
ALT (U/L)	28.80(18.45,46.50)	40.00(20.00,60.70)	0.068
AST (U/L)	21.00(18.00,28.50)	28.20(18.00,44.00)	0.019
FBG (mmol/L)	5.04 (4.78,5.46)	5.75 (5.01,6.92)	<0.001
HbA1c (%)	5.40 (5.20,5.70)	5.70 (5.30,6.70)	0.010
HOMA-IR	4.86(3.23,7.18)	5.15(3.59, 8.00)	0.170
PFF (%)	12.43 (8.64,15.74)	14.76 (11,85,17.52)	0.003

Abbreviations: baPWV, brachial-ankle pulse wave velocity; DM, diabetes mellitus; BMI, body mass index; WC, waist circumference; WHtR, waist-to-height ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triacylglycerol; TC, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase; FBG, fasting blood glucose; HbA1c, glycosylated hemoglobin; HOMA-IR, Homeostasis Model Assessment of Insulin Resistance; PFF, pancreatic fat fraction.

Table 3 Univariate Correlation Coefficients of baPWV

	r	P
Age (years)	0.317	<0.001
BMI (kg/m^2)	−0136	0.126
SBP (mmHg)	0.239	0.007
DBP (mmHg)	0.302	<0.001
TG (mmol/L)	0.161	0.07
TC (mmol/L)	0.163	0.06
HDL-C (mmol/L)	0.031	0.729
LDL-C (mmol/L)	0.056	0.527
TG/HDL-C	0.140	0.114
ALT (U/L)	0.099	0.268
AST (U/L)	0.137	0.124
FBG (mmol/L)	0.225	0.011
HbA1c (%)	0.194	0.029
HOMA-IR	0.056	0.528

Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TG, triacylglycerol; TC, total cholesterol; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase; FBG, fasting blood glucose; HbA1c, glycosylated hemoglobin; HOMA-IR, Homeostasis Model Assessment of Insulin Resistance.

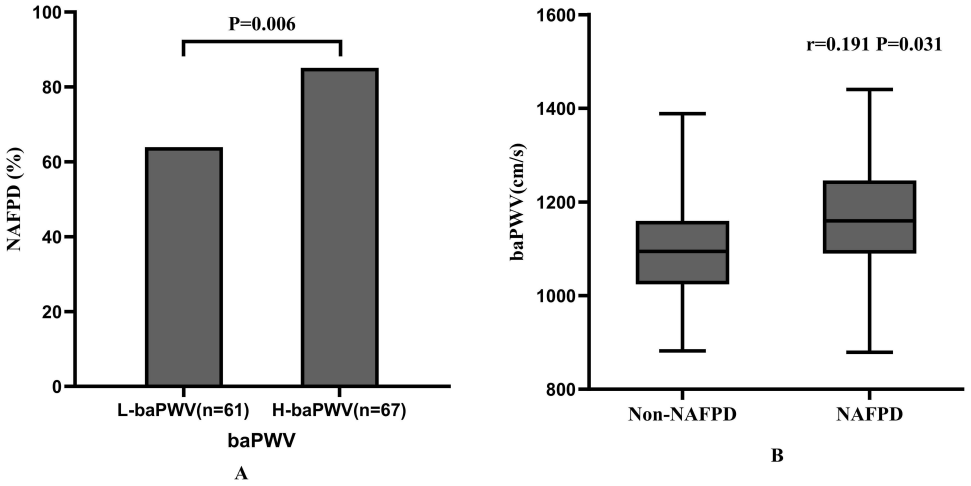

Figure 1 (A) Incidence of NAFPD according to baPWV groups; (B) Association of baPWV with the presence of NAFPD.

L-baPWV. Furthermore, logistic regression analysis demonstrated that the presence of NAFPD was independently correlated with increased baPWV. These results indicate that the likelihood of developing CVD can be increased by the presence of NAFPD, suggesting that individuals with NAFPD require a more proactive approach to assess their cardiovascular risk.

There are several risk factors for atherosclerosis, including age, blood pressure, and smoking.[17] In addition, a stronger correlation between physical activity and arterial stiffness than other risk factors has been observed in older people.[18] Therefore, the effect of old age should be taken into account. Studies have shown that age also affects the amount of fat in the pancreas.[19,20] A cross-sectional study showed that the volume of pancreatic fat plateaus between the ages of 20 and 60 years.[19] The age range of our actual enrollment population was 18 to 53 years old, and the average age was 32 years old. Therefore, age grouping was not performed. Furthermore, smoking and alcohol consumption are also important confounding factors. Smoking and alcohol consumption are strongly associated with subclinical atherosclerosis[21,22] and studies have shown that smoking is a more influential factor in NAFPD than alcohol consumption.[23] In this study, we excluded people who consumed excessive amounts of alcohol. Consequently, to clarify the confounding effect of these factors, we performed multiple logistic analysis adjusted for age, smoking, and other confounding factors.

Table 4 Unadjusted and Adjusted Odds Ratios (ORs) of baPWV by NAFPD

NAFPD	OR (95% CI)	P
Unadjusted	3.22 (1.38–7.53)	0.007
Age-adjusted	2.63 (1.09–6.37)	0.032
Model 1	3.02 (1.13–8.06)	0.027
Model 2	3.26(1.13–9.42)	0.029
Model 3	3.17(1.05–9.58)	0.041

Notes: Model 1: Further adjustment for BMI, SBP, DBP, and smoking on the basis of adjustment for age. Model 2: Adjustment for FBG, HbA1c, and HOMA-IR on the basis of Model 1. Model 3: Adjustment of TG, LDL, ALT, and AST on the basis of Model 2.

Abbreviations: baPWV, brachial-ankle pulse wave velocity; NAPFD, non-alcoholic fatty pancreas disease; CI, confidence interval; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FBG, fasting blood glucose; HbA1c, glycosylated hemoglobin; HOMA-IR, Homeostasis Model Assessment of Insulin Resistance; TG, triacylglycerol; LDL-C, low-density lipoprotein-cholesterol; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

Some studies have suggested that NAFPD is correlated with different non-invasive indicators of atherosclerosis, such as IMT, carotid plaque, carotid-femoral PWV (cfPWV), and carotid artery calcification.[5–10] Most of these studies reported that pancreatic steatosis is independently correlated with atherosclerosis markers after adjusting for confounders,[7–9] which is consistent with our observations. However, in a study by Ozturk et al, the association of fatty pancreas with cfPWV and carotid IMT ceased to be significant after adjusting for confounding factors.[6] Kim et al indicated that a significant correlation between fatty pancreas and carotid atherosclerosis remained in non-obese individuals with T2DM, but not in the obese group, after adjusting for age, sex, and BMI.[5] A large community-based cohort study that evaluated the association between pancreatic fat and systemic calcified atherosclerosis demonstrated that NAFPD was only independently linked to carotid calcification.[10] Thus, it is clear that conventional risk factors have some effect on the relationship between fatty pancreas and atherosclerosis. In addition, the study populations in previous studies mainly involved patients with NAFLD[6,7] and T2DM,[5,9] whereas the participants selected for this study were patients with overweight and obesity.

To the best of our knowledge, this is the first study to assess the relationship between MRI-determined NAFPD and atherosclerosis; previous studies used ultrasound or CT to measure NAFPD. Comparing the echogenicity of the pancreas with that of the liver or kidney is somewhat subjective; therefore, pancreatic fat cannot be quantitatively measured using ultrasound.[24] While pancreatic fat can be visualized with CT, it is challenging to accurately measure the tissue fat content of the pancreas with CT.[25] In addition, the radioactivity of CT restricts it from being widely used in clinical studies. Therefore, MRI is the ideal imaging modality for precisely quantifying pancreatic fat because of its nonradiative nature and high accuracy.[12,26]

In this study, we used baPWV to evaluate arterial stiffness. As stated in earlier reports, cfPWV is considered the highest standard for evaluating arterial stiffness;[27] however, cfPWV is not suitable for routine clinical use because of its high cost and inconvenience associated with measuring devices. Based on the current evidence,[28] baPWV and cfPWV exhibit similar associations with atherosclerotic CVD. Because of its simplicity and convenience, baPWV is an ideal method for assessing early atherosclerosis in clinical practice.[29] Carotid IMT and plaques are also reliable indicators of atherosclerosis.[30,31] Compared with carotid IMT or arterial stiffness, carotid plaques signify the advanced stages of atherosclerosis.[32] Carotid IMT quantitatively assesses arterial morphology composed of intimal lesions,[33] whereas baPWV reflects central arterial stiffness.[29] However, carotid IMT measurements are influenced by the operating physician.

Our study has some limitations. First, owing to the strict inclusion criteria, the sample size was small. Second, the cross-sectional design made it impossible to identify causal connections. Third, we did not examine the pancreatic histology; pancreatic biopsy is highly invasive and cannot be performed ethically in healthy individuals. Therefore, we used MRI which is the best non-invasive test for pancreatic fat. Finally, all of the participants were Chinese, aged between 18 and 65, and were overweight or obese. Studies have shown racial differences in the prevalence of NAFPD and propensity for high PFF. The prevalence of NAFPD was 16.1% in Hong Kong,[34] 30.7% in mainland China,[35] 35% in Indonesia,[36] and 27.8% in America.[37] The differences are partly influenced by the method of examination and ethnicity. Several studies have looked into the likelihood of having high PFF in different racial and/or ethnic groups and found that PFF occurrence was highest among individuals of East/South-East/South Asian descent and lowest among Black African people.[38–41] All published investigations, however, were restricted to comparing no more than two or three racial or ethnic groups. Combined with previous studies, Sun et al's study in Asia supported the role of NAFPD as an independent predictor of atherosclerosis,[9] similar to our conclusion. Therefore, more research across more ethnic groups is required to draw more definitive conclusions.

Conclusion

In summary, this research demonstrated that the presence of NAFPD is associated with an increased risk of atherosclerosis in individuals with no HT but with overweight and obesity. The findings we obtained support the potential role of pancreatic fat in the development of atherosclerosis. Therefore, it is necessary to assess cardiovascular risk in patients with NAFPD for the early prevention of CVD.

Data Sharing Statement

All Data has been provided in the manuscript and no further Data will be shared.

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Author Contributions

Wenjun Wu used to work at The Affiliated Wuxi People's Hospital of Nanjing Medical University and now works at Jinshan Branch of Shanghai Sixth People's Hospital.

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Disclosure

The authors report no conflicts of interest in this work.

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