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Strong association between atherogenic index of plasma and obesity in college students

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Abstract

Background The issue of obesity is becoming more and more prominent. Understanding the metabolic profile of obese young adults and finding possible risk markers for early prediction and intervention is of great importance.

Methods A total of 13,082 college students with an average age of 20 years were enrolled in this cross-sectional study. The lipid composition was measured and novel lipid profiles such as AIP, AI, LCI, Non-HDL-C, TC/HDL-C, LDL-C/HDL-C and TyG were calculated. Participants were then assessed as normal weight, overweight or obese based on their BMI. Pearson correlation analysis, multivariate logistic analysis, and predictive analysis were used to assess the association and discriminative power between lipid profile and obesity.

Results The prevalence of obesity with dyslipidemia was 61.0% in males and 38.7% in females. Most obese patients were associated with only one dyslipidemia component, with the highest proportion having low HDL-C. We found a positive correlation between all lipid profiles except HDL-C and BMI. Multivariate logistics regression shows, AIP were strongly associated with obesity, which shows the largest OR = 12.86, 95%CI (9.46,17.48).

Conclusions In the youth population, higher AIP levels were positively and strongly associated with obesity. AIP may be a novel and better risk biomarker for predicting obesity.

Keywords AIP, Obesity, Young adult

Background

Obesity is a systemic disease characterized by excessive and abnormal accumulation of adipose tissue in the body [1]. Nowadays, the number of obese people is as high as 700 million, and it shows a rapid growth trend, which brings a huge economic burden to society [2]. The prevalence of overweight and obesity is peaking at younger ages [3]. According to estimates, 1% rate of overweight and obesity rates reduce among 16- to 17-year-olds, there would be 52,821 fewer obese people in the future and an average of $586 million less in health care costs after age 40 [4]. Therefore, understanding the metabolic signatures

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of obesity in youth and finding risk markers for early identification and intervention is of great social value.

Previous studies have shown that abnormal changes in blood lipids are heavily associated with the occurrence of obesity [5], which is also commonly observed in children or adolescents [6, 7]. It was previously suggested that lipid metabolism disorders are more prevalent in obese children and adolescents. Additional evidence has been demonstrated that children and adolescents with severe lipid metabolism disorders are at elevated risk for obesity and early atherosclerosis [8, 9]. Several studies suggested that abnormalities in the metabolism of individual lipid components or variations in some non-traditional significant lipid features, or changes in the joint ratio of lipid components, or in lipid-related derived indices, are strongly correlated with obesity in children and adolescents. For example, triglyceride/high-density lipoprotein cholesterol (TG/HDL-C), triglyceride glucose (TyG), visceral adiposity index (VAI) and height-corrected lipid accumulation product (HLAP) and atherogenic index of plasma (AIP) can be a useful predictor of obese adolescents [10–14].

Although known associations between excess weight and abnormal lipid concentrations have been concluded, it is worth paying close attention and considering that blood lipids are modified over time. Consideration of lipid profiles may provide a basis for assessing risk and monitoring the development of obesity and atherosclerosis, but additional evidence on the metabolic signatures of obesity in adolescents is still needed. Moreover, there is a temporal trend in lipid disorders over time [15] and previous study has shown that changes in blood lipids in youthful people have their own characteristics [16]. We therefore conducted this study to further elucidate the characteristics of abnormal lipid metabolism in obese young college students and to seek risk markers for early identification of obesity.

Methods

A total of 13,082 college students from Xinjiang Medical University were included in this study from 2018 to 2021. According to China's diagnostic criteria for obesity, individuals with a body mass index (BMI) ranging from 24 to 27.9 are classified as having overweight, while those with a BMI of 28 or higher are categorized as obese [12]. Ultimately, 8,366 subjects were classified as normal weight, 3340 as having overweight, and 1,376 as obese. This study received approval and support from the Ethics Review Committee of the First Affiliated Hospital of Xinjiang Medical University. As the research was retrospective and based on real-world situations, obtaining informed consent from the students was not required. The study adheres to the ethical principles outlined in the 1975 Declaration of Helsinki.

After fasting for one night, relevant indicators were measured and recorded by a trained professional. Weight (kg) and height (m) were assessed with participants barefoot and wearing light clothing. Body Mass Index (BMI) was calculated by dividing weight by the square of height (kg/m^2). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured in a calm state following a period of rest. Venous blood samples were collected and stored in tubes under low temperature and anticoagulant conditions, with biochemical tests performed within 24 h. The results for Total Cholesterol (TC), Triglyceride (TG), High-Density Lipoprotein Cholesterol (HDL-C), Low-Density Lipoprotein Cholesterol (LDL-C), and other lipid indices were obtained using equipment for chemical analysis (Dimension AR/AVL Clinical Chemistry System, Newark, NJ, USA). All laboratory examinations were conducted at the Laboratory Center of the First Affiliated Hospital of Xinjiang Medical University.

Computational formula

The novel lipid parameters were calculated as follows:

Non-HDL-C = TC - HDL-C.

arteriosclerosis index(AI)= (TC-HDL)/HDL.

Atherogenic index of plasma (AIP) = lg (TG/HDL-C).

Lipoprotein combine index (LCI) = TC × TG × LDL-C/HDL-C.

Triglyceride glucose index(TyG) = Ln(TG*FBG/2).

Statistic

The data were analyzed using SPSS 26.0 for Windows statistical software (SPSS Inc., Chicago, IL, USA) and R (version 4.0.3). Categorical variables were expressed as numbers and percentages, while continuous variables were presented as means (SD) or medians (IQR). Student's t-test or Mann-Whitney U-test was used to compare normally distributed numeric variables or non-normally distributed continuous variables, respectively. BMI, AIP, and various lipid components (such as HDL-C, TC, TG, LDL-C) were assessed using the Pearson coefficient. Multivariate logistic regression analyses were conducted to examine the association between AIP, conventional lipid composition, and obesity. The area under the curve (AUC) value of the receiver operating characteristic (ROC) curve was calculated to compare the predictive value of AIP and three lipid components (TC, TG, LDL-C) in relation to obesity. All statistical tests were two-tailed with significance set at $p < 0.05$.

Results

13,082 young people took part in our study. Of those studied, 8,366 were of normal weight, 3,340 were overweight and 1,376 were obese. Table 1 shows the laboratory data of the three groups. Compared to the normal-weight group, subjects with excess weight had

Table 1 Baseline characteristics of all participants

Variables	Normal weight (*n* = 8366)	Excess weight(*n* = 4716)	*P* value	Excess weight(*n* = 4716)		*p* value
				Overweight (*n* = 3340)	Obesity (*n* = 1376)	
Male(n,%)	4588(54.8%)	2827(59.9%)	< 0.001	1903(56.98%)	924(67.15%)	< 0.001
Age(year)	20.78 ± 2.19	20.85 ± 2.37	0.112	20.98 ± 2.37	20.53 ± 2.33	< 0.001
BMI(kg/m^2)	21.19 ± 1.49	27.16 ± 3	< 0.001	25.59 ± 1.11	30.95 ± 2.74	< 0.001
SBP(mmHg)	114.83 ± 11.94	120.81 ± 12.4	< 0.001	118.87 ± 12.01	125.5 ± 12.08	< 0.001
DBP(mmHg)	71.71 ± 8.46	74.81 ± 9.25	< 0.001	73.6 ± 8.8	77.74 ± 9.64	< 0.001
WBC(10^9/L)	6.5(5.57,7.56)	7.07(6.11,8.28)	< 0.001	6.92 (5.95,8.03)	7.56 (6.53,8.8)	< 0.001
FBG(mmol/L)	4.51 ± 0.52	4.57 ± 0.63	< 0.001	4.55 ± 0.59	4.61 ± 0.71	< 0.001
Hb(g/dl)	147.7 ± 17.08	149.72 ± 16.81	< 0.001	148.68 ± 17.08	152.24 ± 15.87	< 0.001
PLT(10^9/L)	265(228,306)	278(240,319)	< 0.001	274 (236,315)	287 (248,332)	< 0.001
BUN(mmol/L)	4.37 ± 1.20	4.39 ± 1.17	0.352	4.39 ± 1.19	4.39 ± 1.12	0.521
Cr(μmol/L)	72.91 ± 13.96	74.71 ± 14.8	< 0.001	74.19 ± 14.7	75.97 ± 14.96	< 0.001
ALT(IU/L)	13.3 (9.97,18.5)	18.58 (12.5,29.7)	< 0.001	16.9 (11.8,25.41)	24.5 (15.17,39.75)	< 0.001
AST(IU/L)	17.47 (15.3,20.4)	18.84 (15.9,23.05)	< 0.001	18.4 (15.7,22.1)	20.3 (16.8,26.19)	< 0.001
TBIL(mmol/L)	12.58 (9.13,17.1)	11.82 (8.62,16.2)	< 0.001	12 (8.7,16.26)	11.5 (8.42,16.1)	< 0.001
Traditional lipid profiles						
HDL-C(mmol/L)	1.34 ± 0.26	1.24 ± 0.29	< 0.001	1.27 ± 0.29	1.19 ± 0.27	< 0.001
LDL-C(mmol/L)	2.53 (2.12,2.9)	2.63 (2.19,3)	< 0.001	2.56 (2.13,2.97)	2.79 (2.37,3.13)	< 0.001
TG(mmol/L)	0.9 (0.72,1.15)	1.01(0.76,1.41)	< 0.001	0.96 (0.73,1.32)	1.16 (0.86,1.67)	< 0.001
TC(mmol/L)	4.06 ± 0.79	4.16 ± 0.84	< 0.001	4.09 ± 0.82	4.33 ± 0.87	< 0.001
Non-traditional lipid profiles						
AIP	-0.17 (-0.29,-0.04)	-0.08 (-0.24,0.1)	< 0.001	-0.11 (-0.26,0.06)	0 (-0.17,0.18)	< 0.001
Non-HDL-C	2.65 (2.25,3.12)	2.81 (2.35,3.39)	< 0.001	2.73 (2.27,3.26)	3.02 (2.54,3.65)	< 0.001
TC/HDL-C	3.01 (2.63,3.45)	3.33 (2.8,4.04)	< 0.001	3.22 (2.73,3.87)	3.66 (3.02,4.45)	< 0.001
LDL-C/HDL-C	1.89 (1.58,2.24)	2.13 (1.75,2.68)	< 0.001	2.04 (1.67,2.56)	2.33 (1.87,2.98)	< 0.001
AI	2.01 (1.63,2.45)	2.33 (1.8,3.04)	< 0.001	2.22 (1.73,2.87)	2.66 (2.02,3.45)	< 0.001
LCI	6.76 (4.85,9.8)	8.8 (5.13,15.88)	< 0.001	7.82 (4.67,13.68)	12.1 (6.7,21.03)	< 0.001
TyG	0.7 (0.46,0.96)	0.83 (0.52,1.18)	< 0.001	0.77 (0.48,1.1)	0.98 (0.65,1.35)	< 0.001
TG/HDL-C	0.67 (0.52,0.91)	0.84 (0.58,1.27)	< 0.001	0.78 (0.55,1.14)	1.01 (0.68,1.53)	< 0.001

Method: Categorical variables were expressed as numbers and percentages, while continuous variables were presented as means (SD) or medians (IQR). Student's t-test or Mann-Whitney U-test was used to compare normally distributed numeric variables or non-normally distributed continuous variables, respectively. *P* for excess weight vs. normal weight. *p* for obesity vs. normal weight

Abbreviation: SBP: systolic blood pressure; DBP: diastolic blood pressure; Hb: hemoglobin; PLT: platelet; WBC: white blood Cell; BUN: blood urea nitrogen; ALT: alanine transaminase; AST: aspartate transaminase; TC: total cholesterol; Cr: creatinine; FBG: fasting blood glucose; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TG: triglyceride; TBIL: total bilirubin; AIP: atherogenic index of plasma; AI: atherogenic index; LCI: lipoprotein combined index. TyG: Triglyceride-Glucose Index

higher SBP, DBP, white blood cell(WBC), fasting blood glucose (FBG), hemoglobin(Hb), platelet(PLT), creatinine (Cr), alanine transaminase (ALT), aspartate transaminase (AST) and higher rate of dyslipidemia. The values of LDL-C, TG, TC, AIP, Non-HDL-C, TC/HDL-C, LDL-C/HDL-C, AI, LCI， TyG and TG/HDL-C were also higher in excess weight (Ps < 0.001). However, there were no significant differences in age and blood urea nitrogen (BUN) between normal weight and excess weight.

In Table 2, distribution of dyslipidemia in different genders were shown. The prevalence of obesity with dyslipidemia was 61.0% in men and 38.7% in women. Most obese patients were associated with only one dyslipidemia component, and the proportion of LHDL-C was the highest. More dyslipidemia was incidence in obesity compared to normal weight. To be specific, higher rates of HTC, HTG, HLDL-C and LHDL-C in total and male subjects with obesity. Only elevated rates of HTG and LHDL-C difference exist in females.

Figure 1 shows the significant correlation between clinical variables, lipid profiles (including HDL-C, LCI, TG, AIP, LDL-C/HDL-C, AI, TC/HDL-C, LDL-C, TC and Non-HDL-C) and BMI in all subjects. ALL lipid components and novel lipid variables beside HDL-C showed weak positive correlations with BMI (Ps < 0.01). HDL-C was negatively associated with BMI (r=-0.227, $p < 0.01$). SBP, DBP, WBC, AST and ALT also showed weak positive correlations with BMI (Ps < 0.01).

Table 3 shows the area under the ROC curve of each lipid index for obesity. In the overall subjects, the area under the curve of AIP, TC/HDL-C, LDL-C/HDL-C, AI,

Table 2 Distribution of dyslipidemia in different gender

Variables		Total($n=13082$)			P	Male($n=7415$)			P	Female($n=5667$)			P
		Normal weight	Excess weight			Normal weight	Excess weight			Normal weight	Excess weight		
			Obesity	Overweight			Obesity	Overweight			Obesity	Overweight	
High TC		86(1.0%)	33(2.4%)*	41(1.2%)	< 0.001	27(0.6%)	25(2.7%)*	26(1.4%)	< 0.001	59(1.6%)	8(1.8%)^{ns}	15(1.0%)	0.307
High TG		142(1.7%)	156(11.3%)*	159(4.8%)	< 0.001	86(1.9%)	131(14.2%)*	130(6.8%)	< 0.001	56(1.5%)	25(5.5%)*	29(2.0%)	< 0.001
Low HDL-C		1924(23%)	683(49.6%)*	1205(36.1%)	< 0.001	1299(28.3%)	525(56.8%)*	863(45.3%)	< 0.001	625(16.5%)	158(35.0%)*	342(23.8%)	< 0.001
High LDL-C		79(0.9%)	44(3.2%)*	61(1.8%)	< 0.001	37(0.8%)	37(4.0%)*	45(2.4%)	< 0.001	42(1.1%)	7(1.5%)^{ns}	16(1.1%)	0.724
Dyslipidemia		2096(25%)	739(53.7%)*	1287(38.5%)	< 0.001	1363(29.7%)	564(61.0%)*	913(48.0%)	< 0.001	733(19.4%)	175(38.7%)*	374(26.0%)	< 0.001
Number of lipid disorders	1	1973(23.6%)	587(42.7%)*	1133(33.9%)	< 0.001	1286(28.0%)	431(46.6%)*	784(41.2%)	< 0.001	687(18.2%)	156(34.5%)*	349(24.3%)	< 0.001
	2	112(1.3%)	130(9.4%)*	136(4.1%)		69(1.5%)	115(12.4%)*	114(6.0%)		43(1.1%)	15(3.3%)*	22(1.5%)	
	3	10(0.1%)	19(1.4%)*	11(0.3%)		7(0.2%)	15(1.6%)*	8(0.4%)		3(0.1%)	4(0.9%)*	3(0.2%)	
	4	1(0.0%)	3(0.2%)*	7(0.2%)		1(0.0%)	3(0.3%)*	7(0.4%)		-	-	-	

Method: Categorical variables were expressed as numbers and percentages.chi-square test was used to analysis. P for excess weight vs. normal weight. * means: $P<0.001$ for obesity vs. normal weight. ^{ns} means: Not significant for obesity vs. normal weight

LCI and TG/HDL-C were all greater than 0.7 (AIP: 0.708, 95%CI (0.692,0.724), $p<0.001$.

In Table 4, multivariate logistic regression was conducted to clarify the relationship between the lipid variables and obese. After adjusting for sex, age, SBP, WBC, ALT, AST, TBIL, FBG and Cr, AIP were firmly associated with obesity, OR = 12.86, 95%CI (9.46,17.48). In subgroup analysis, after adjusting for age, SBP, WBC, ALT, AST, TBIL, FBG and Cr, AIP showed significant stronger predictive power for obesity in males, OR = 29, 95%CI (19.65,42.8),$P<0.001$; in female, OR = 2.09, 95%CI (1.19,3.69), $P=0.011$.

Discussion

In this study, we examined the prevalence of dyslipidemia and obesity， and the association between obesity and dyslipidemia in a large sample of Chinese adults in their 20s. It was additionally suggested that increases in AIP were most strongly associated with elevated rates of obesity and that AIP may be an independent risk factor for obesity. In view of the trend of obesity and overweight at a younger age, we thought it would be useful to evaluate the ability of AIP to predict overweight and obesity in adolescents in order to predict obesity earlier. Early detection of blood lipid indicators and attention to dyslipidemia may capture the risk of future obesity early.

Obesity is a major public health problem of the 21st century. Studies show that BMI in children and adolescents has been on the rise for the past 40 years [17]. Studies have estimated that 398,000 children aged 6–9 years in 21 European countries are severely obese [18]. As a result, rising obesity rates have attracted increasing attention. The close relationship between obesity and blood lipids has been gradually recognized in recent years. Atherogenic index of plasma (AIP), as a fresh lipid index, is suggested to be a significant predictor of cardiovascular risk and widely recommended as a potential biomarker of atherosclerosis and cardiovascular disease (CVD) [19–22]. For example, Karadağ MK et al. proposed that the area under the curve of AIP was 0.66, and the sensitivity of AIP in diagnosing heart failure was 68% when the critical value was 0.47 [19]. In addition, numerous studies have demonstrated that AIP can be used as a marker for screening for subclinical atherosclerosis [20, 21] Moreover, Khosravi A et al. proposed in their study that when the critical point level was 0.62, the sensitivity of AIP to the diagnosis of unstable plaques in coronary artery disease (CAD) diseases was close to 90% [22]. Not incidentally, it has also shown a great ability to predict obesity in recent studies. Shen et al. found AIP is closely related to abdominal obesity and the detection rate of abdominal obesity increased as the AIP quartile increased [23]. Zhang et al. also found that AIP was significantly associated with obesity [24]. Zhu et al. also suggested that using

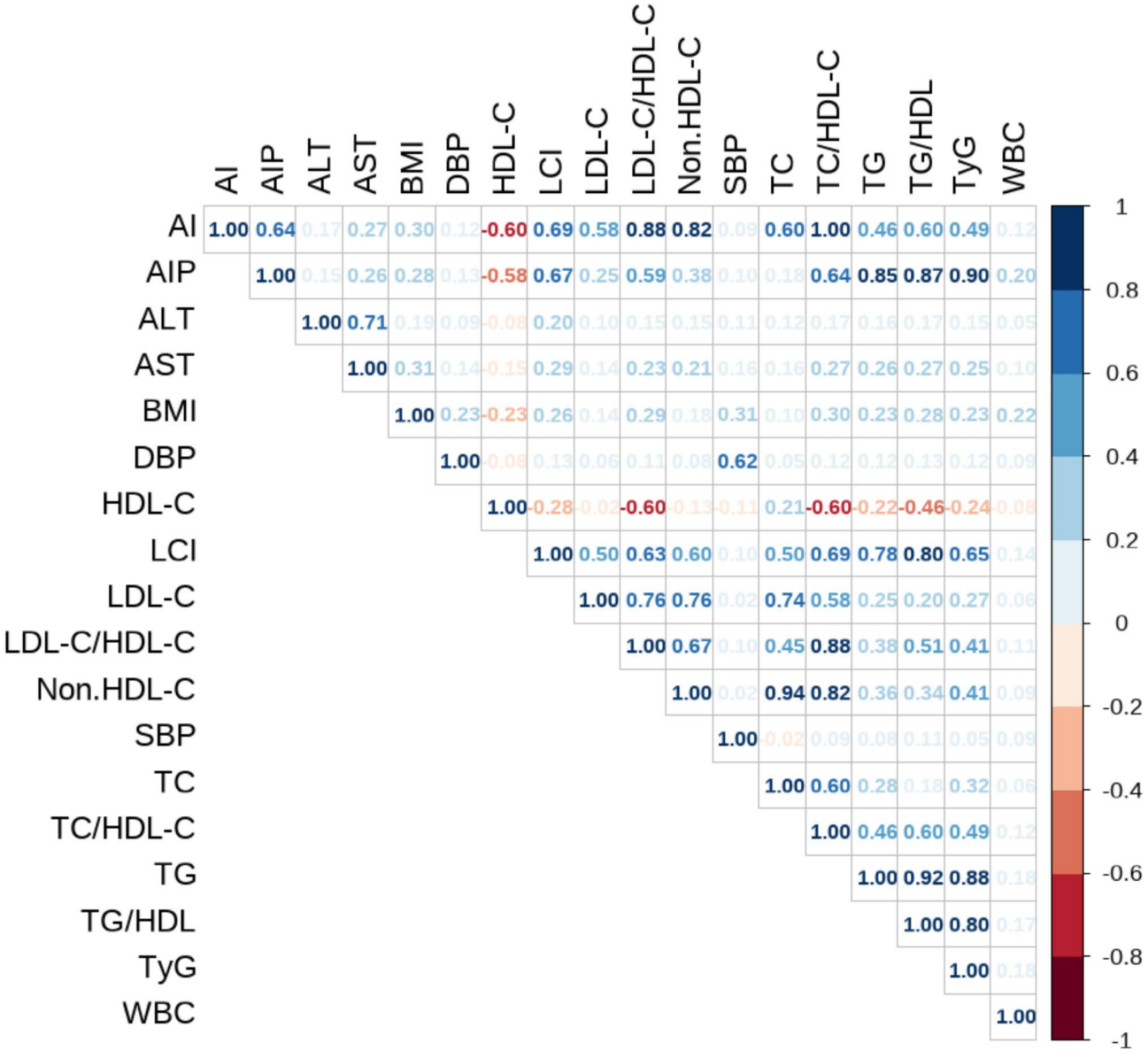

Fig. 1 The correlation matrix graph to assess the correlation of lipid components to BMI

AIP instead of HDL-C and TG significantly improved the risk prediction of obesity (AUC improvement = 0.011, $P = 0.011$) [25].

While AIP, as a composite indicator combining TG and HDL-C, it is reasonable to suspect that it is more closely related to obesity. Increasing evidence suggests that insulin resistance, the most common metabolic disorder in obesity, is the most significant driving force for obesity-related metabolic dyslipidemia [26]. In the physiological state, insulin promotes the hydrolysis of TG in very low density lipoprotein(VLDL) particles driven by lipoprotein lipase (LPL) and promotes hepatic lipase (HL) activity. However, a state of insulin resistance leads to a slowing of the clearance of TG-rich lipoproteins in plasma, ultimately leading to hypertriglyceridemia [27]. In addition, insulin resistance leads to increased lipolysis and excessive release of free fatty acids. They will not only promote the synthesis of lipids in the liver, but also promote the formation of VLDL [28]. The result of increased secretion and decreased clearance of VLDL particles is hypertriglyceridemia [29]. In addition, obesity and insulin resistance accelerate the accumulation of low-density lipoprotein (LDL) and high-density lipoprotein (HDL) particles by TG through cholesteryl ester transfer proteins (CETP) [30]. At this time, the enrichment of HDL in TG and the increase in HL activity resulted in faster clearance of TG-rich HDL produced by CETP-mediated transfer. Finally, they are hydrolyzed by HL to form small dense LDL (sdLDL) and dysfunctional HDL particles,

Table 3 The area under the ROC curve of all lipids index for obese

Variables	All subjects			Males			Female		
	AUC	AUC[95%CI]	*p*	AUC	AUC[95%CI]	*p*	AUC	AUC[95%CI]	*p*
AIP	0.708	[0.692,0.724]	< 0.001	0.762	[0.744,0.78]	< 0.001	0.593	[0.56,0.626]	0.000
Non-HDL-C	0.650	[0.636,0.664]	< 0.001	0.746	[0.726,0.766]	< 0.001	0.518	[0.485,0.551]	0.202
TC/HDL-C	0.710	[0.694,0.726]	< 0.001	0.769	[0.753,0.785]	< 0.001	0.580	[0.547,0.613]	0.000
LDL-C/HDL-C	0.706	[0.692,0.720]	< 0.001	0.761	[0.743,0.779]	< 0.001	0.584	[0.553,0.615]	0.000
AI	0.710	[0.694,0.726]	< 0.001	0.769	[0.751,0.787]	< 0.001	0.580	[0.551,0.609]	0.000
LCI	0.708	[0.692,0.724]	< 0.001	0.789	[0.771,0.807]	< 0.001	0.536	[0.505,0.567]	0.013
TC(mmol/L)	0.594	[0.578,0.610]	< 0.001	0.702	[0.684,0.72]	< 0.001	0.567	[0.536,0.598]	0.000
TG(mmol/L)	0.673	[0.655,0.691]	< 0.001	0.745	[0.729,0.761]	< 0.001	0.533	[0.504,0.562]	0.020
LDL-C(mmol/L)	0.619	[0.603,0.635]	< 0.001	0.707	[0.689,0.725]	< 0.001	0.528	[0.501,0.555]	0.049
HDL-C(mmol/L)	0.678	[0.664,0.692]	< 0.001	0.682	[0.662,0.702]	< 0.001	0.649	[0.622,0.676]	0.000
TyG	0.671	[0.653,0.689]	0.009	0.736	[0.718,0.754]	0.009	0.545	[0.51,0.58]	0.002
TG/HDL-C	0.708	[0.692,0.724]	0.008	0.762	[0.744,0.78]	0.009	0.593	[0.562,0.624]	0.000

Method: By employing the ROC curve, the area under the curve (AUC) is computed to ascertain the diagnostic efficacy of the lipid components

Abbreviations: TG: triglyceride; TC: total cholesterol; HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; TyG: triglyceride glucose; AIP: atherogenic index of plasma; AI: arteriosclerosis index; LCI: lipoprotein combine index;

Table 4 Multivariate logistics regression of traditional and novel lipid indexes for obese

Variable	B	SE	Z	*P*	OR[95%CI]
lipid indexes as independent risk factor for obesity in all subjects*					
TC(mmol/L)	0.256	0.039	6.496	< 0.001	1.29[1.2,1.39]
TG(mmol/L)	0.609	0.053	11.499	< 0.001	1.84[1.66,2.04]
LDL-C(mmol/L)	0.504	0.054	9.352	< 0.001	1.66[1.49,1.84]
HDL-C(mmol/L)	-2.092	0.15	-13.969	< 0.001	0.12[0.09,0.17]
Non-HDL-C	0.443	0.443	10.865	< 0.001	1.56[1.44,1.69]
TC/HDL-C	0.712	0.040	17.778	< 0.001	2.04[1.88,2.2]
LDL-C/HDL-C	0.93	0.052	18.004	< 0.001	2.54[2.29,2.81]
LCI	0.05	0.003	14.684	< 0.001	1.05[1.04,1.06]
AIP	2.554	0.157	0.157	< 0.001	12.86[9.46,17.48]
AI	0.712	0.040	17.778	< 0.001	2.04[1.88,2.2]
TyG	1.005	0.079	12.647	< 0.001	2.73[2.34,3.19]
TG/HDL-C	0.819	0.056	14.74	< 0.001	2.27[2.03,2.53]
AIP as independent risk factor for prediction in different gender#					
Obesity (males)	3.367	0.199	16.959	< 0.001	29[19.65,42.8]
Obesity(females)	0.738	0.289	2.554	0.011	2.09[1.19,3.69]

Method: Multivariate logistics regression, *Adjusted for Sex, Age, SBP, WBC, ALT, AST, TBIL, FBG, Cr; #Adjusted for Age, SBP, WBC, ALT, AST, TBIL, FBG, Cr;

Abbreviations: TC: total cholesterol; TG: triglyceride; LDL-C: low density lipoprotein cholesterol; HDL-C: high density lipoprotein cholesterol; LCI: lipoprotein combine index; AIP: atherogenic index of plasma; AI: arteriosclerosis index; TyG: triglyceride glucose;

which further lead to reduced plasma HDL-C levels [31, 32].

Our results confirmed a strong relationship between AIP and obesity among young students. Not only that, the AIP test is much cheaper and the test procedure is very simple. Therefore, large-scale AIP screening can be carried out in community and primary hospitals to find people who are likely to have elevated BMI and trend to be obese.

Limitation

Nevertheless, some shortcomings in our study must be acknowledged. First, it is not known whether the subjects' diet, lifestyle and other factors that affect blood lipid levels changed in the short term before the blood samples were collected. In addition, We have not been able to conduct continuous follow-up to understand whether the changes in blood lipids are directly related to BMI. Finally, this is a cross-sectional study, and further high-quality cohort studies are needed to determine whether AIP can truly predict the development of obesity.

Conclusion

Our study confirms that AIP is an independent risk factor of abnormal weight. It can indeed be used as a novel predictor of obesity and has the ability to predict people with high risk of overweight and obesity early.

Abbreviations

AI	Arteriosclerosis index
AIP	Atherogenic index of plasma
ALT	Alanine transaminase
AST	Aspartate transaminase
BMI	Body mass index
BUN	Blood urea nitrogen
CETP	Cholesteryl ester transfer protein
Cr	Creatinine
DBP	Diastolic blood pressure
FBG	Fasting blood glucose
Hb	Hemoglobin
HDL-C	High density lipoprotein cholesterol
HDL	High density lipoprotein
HL	Hepatic lipase
HLAP	Height-corrected lipid accumulation product
LCI	Lipoprotein combine index
LDL-C	Low density lipoprotein cholesterol
LDL	Low density lipoprotein
Lp	Lipoprotein
LPL	Lipoprotein lipase
Plt	Platelet
SBP	Systolic blood pressure

sdLDL	Small dense LDL
TBIL	Total bilirubin
TC	Total cholesterol
TG	Triglyceride
TyG	Triglyceride glucose
VAI	Visceral adiposity index
VLDL	Very low density lipoprotein
WBC	white blood cell

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Author contributions
WZL and JML made substantial contributions to study conception and design and to the drafting and critical revision of the manuscript for important intellectual content; SCH and XYZ collected data and undertook the statistical analyses; XY , YTL and YYZ gave critical comments on the draft and contributed to the manuscript writing; TTW and XX made substantial contributions to study conception and design, drafting and critical revision of the manuscript for important intellectual content, including study supervision. All authors read and approved the final manuscript.

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Data availability
The datasets during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The research protocol was approved by the ethics committee or review committee of the First Affiliated Hospital of Xinjiang Medical University. The approval number is K202403-43. Because the study was a retrospective study based on real-world situations, there was no need to obtain informed consent from the students.

Consent for publication
Not Applicable.

Competing interests
The authors declare no competing interests.

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