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Dietary and plasma atherogenic and thrombogenic indices and cardiometabolic risk factors among overweight and individuals with obesity

Reyhaneh Mokhtari[1] and Mahdieh Abbasalizad Farhangi[1*]

Abstract

Background Obesity and hyperlipidemia are the two central metabolic disorders linked to non-communicable diseases (NCDs) that increase the risk of cardiovascular disease (CVD). Apart from dyslipidemia, the Atherogenic Index of Plasma (AIP), which is associated with dietary consumption, is another marker for predicting the risk of CVD. Healthy fat quality indicators may impact AIP. The purpose of this study is to ascertain whether there is any connection between Iranian obese people's plasma and dietary indices and cardiometabolic risk factors.

Methods This cross-sectional study, consisted of 645 overweight and obese participants. The study included assessments of body composition and anthropometric measurements. Dietary fatty acid consumption was evaluated using a validated Food Frequency Questionnaire (FFQ) containing 168 items. Additionally, biochemical parameters, including serum total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), fasting serum glucose (FSG), and insulin levels, were measured using enzymatic methods. The lipid profile was quantified.

Results For participants in higher tertiles of the AIP, the percentage of men was significantly higher than women (men: 48.1%, women: 51.7%, $p < 0.001$). Additionally, individuals in higher tertiles of AIP had a higher waist-to-hip ratio (WHR) (mean WHR: 0.92 ± 0.05 vs. 0.86 ± 0.04 in lower tertile, $p < 0.001$). Participants in the highest tertile of AIP had higher systolic blood pressure (SBP: 132 ± 8 mmHg vs. 118 ± 6 mmHg in lower tertile, $p < 0.001$), total cholesterol (TC: 210 ± 15 mg/dL vs. 185 ± 12 mg/dL, $p < 0.001$), triglycerides (TG: 180 ± 20 mg/dL vs. 120 ± 15 mg/dL, $p < 0.001$), and glucose concentrations (fasting glucose: 105 ± 10 mg/dL vs. 90 ± 8 mg/dL, $p < 0.001$). Participants in the lower tertile of AIP had higher HDL cholesterol levels (HDL: 60 ± 5 mg/dL vs. 45 ± 4 mg/dL in higher tertile, $p < 0.001$). In the model for Thrombogenicity Index (TI), participants in the higher tertile had higher glucose concentrations (glucose: 110 ± 12 mg/dL vs. 95 ± 9 mg/dL in lower tertile, $p = 0.04$).

Conclusion This research introduces a novel field of investigation and emphasizes the possible importance of TI, AI, and AIP indices in regulating cardiometabolic risk factors.

*Correspondence:
Mahdieh Abbasalizad Farhangi
abbasalizadm@tbzmed.ac.ir; abbasalizad_m@yahoo.com
[1]Department of Community Nutrition, Faculty of Nutrition, Tabriz University of Medical Sciences, Attar Neyshabouri Street, Daneshgah Blv, Tabriz, Iran

Background

The most significant worldwide health problem currently is non-communicable diseases (NCDs). Obesity and hyperlipidemia are the two main metabolic conditions linked to NCDs that increase the risk of cardiovascular disease (CVD) [1]. As an inflammatory state with increased adipose tissue and decreased adiponectin levels, obesity is a chronic disease [2, 3]. Obesity inhibits the body's control of inflammatory processes and exacerbates the inflammatory condition [4–6]. Additionally, especially in individuals with obesity, perivascular adipose tissue compromises endothelial function and local inflammation. Obesity lowers vascular elasticity, resulting in hypertension because it increases intravascular inflammation, interstitial arterial thickness, and arterial lumen diameter [7]. The arterial stiffness results are lower diastolic blood pressure (DBP) and higher systolic blood pressure (SBP). A higher risk of myocardial infarction and other coronary heart diseases (CHD) results from these effects and increased pulse pressure, which puts more load on the left ventricle [8–10]. As a result of pandemic obesity, the rate of CVD is predicted to reach 23.6 million worldwide by 2030 [11]. According to the World Health Organization's most recent data from 2018, there was a sharp rise in the global obesity rate. Over 2 billion persons over the age of 18 were overweight. Over 650 million persons worldwide are obese (WHO, 2018), with the US leading the way with over 35% of men and 40% of women who meet the criteria for obesity with a body mass index (BMI) of 30 kg/m 2 or more [12, 13]. However, it was frequently seen that people who were obese had anomalies in their metabolism of fat. The evidence was substantial, showing an inverse association between high-density lipoprotein cholesterol (HDL-C) and a direct or indirect relationship with high total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG) to high BMI. It has been suggested that high levels of LDL-C and low levels of HDL-C, which are associated with a higher risk of CVD in individuals with obesity, are strongly correlated with BMI [14]. The atherogenic index of plasma (AIP) is one of the best indicators for estimating the risk of CVD. An effective indication of dyslipidemia and related conditions, including cardiovascular illnesses, AIP is a unique index [15] that has been used to quantify blood lipid levels [16–18]. The leading causes of obesity and weight gain are imbalances in the amount of calories consumed and the types and quantities of nutrients ingested. Research has shown that the kinds and quality of food ingested, as well as the individual macronutrients—fat, carbohydrates, and protein—impact changes in body weight [19]. For example, throughout the past few decades, research has suggested indicators of the quality of dietary fat, such as the thrombogenicity index (TI) and the atherogenicity index (AI) [20]. Therefore, it is crucial to consider the dietary and plasma indices. However, the association between AIP, TI, and AI and various demographic, anthropometric, and biochemical variables has yet to be evaluated. Thus, the present study aimed to identify the relationship between dietary and plasma indices and investigate their relationships with cardiometabolic risk factors.

Materials and methods

This cross-sectional study carried out in the Iranian cities of Tabriz and Tehran, involved 645 overweight and obese participants. These data are a combination of two previously approved projects in Tabriz University of Medical Sciences with 339 participants [21, 22] and a currently approved recruiting project (MS thesis of RM; identifier; IR.TBZMED.REC. 1402.071) (Fig. 1). The first project in Tabriz focused on investigating the association between the inflammatory potential of a diet, the + 405 VEGF C/G (rs2010963) polymorphism, and metabolic components in patients with metabolic syndrome. This study assessed 150 patients with metabolic syndrome and 50 healthy individuals using a semi-quantitative food frequency questionnaire (FFQ) to calculate the dietary inflammatory index (DII) and various biochemical markers [21]. The second project, also conducted in Tabriz, evaluated the association of adherence to the Healthy Eating Index (HEI)-2015 with sociodemographic factors, psychological characteristics, metabolic syndrome (MetS), and other cardio-metabolic risk factors among 188 healthy obese adults (96 males and 92 females). Structural equation modeling (SEM) was used to analyze interrelationships among these factors, revealing that adherence to HEI could mediate the effects of socio-demographic and psychological factors on cardio-metabolic risk markers [22].

Inclusion-exclusion criteria

Public announcements and posters were distributed to recruit patients from the outpatient clinics. People with a BMI of 25 kg/m2 or more and an age range of 20 to 50 years old met the study's inclusion requirements. Exclusions from the study included those with particular conditions such as menopause, pregnancy, cancer, hepatic or renal disease, diabetes mellitus, recent bariatric surgery, breastfeeding, or a history of CVD.

Additionally, individuals with diabetes mellitus without distinguishing between Type 1 and Type 2 diabetes were excluded. Participants receiving chronic treatment with oral lipid-lowering agents or using dietary fiber supplements and omega-3, omega-6, and omega-9 supplements were also excluded. Finally,

individuals who had followed weight-reduction diets or used supplements within the three months before study involvement were excluded.

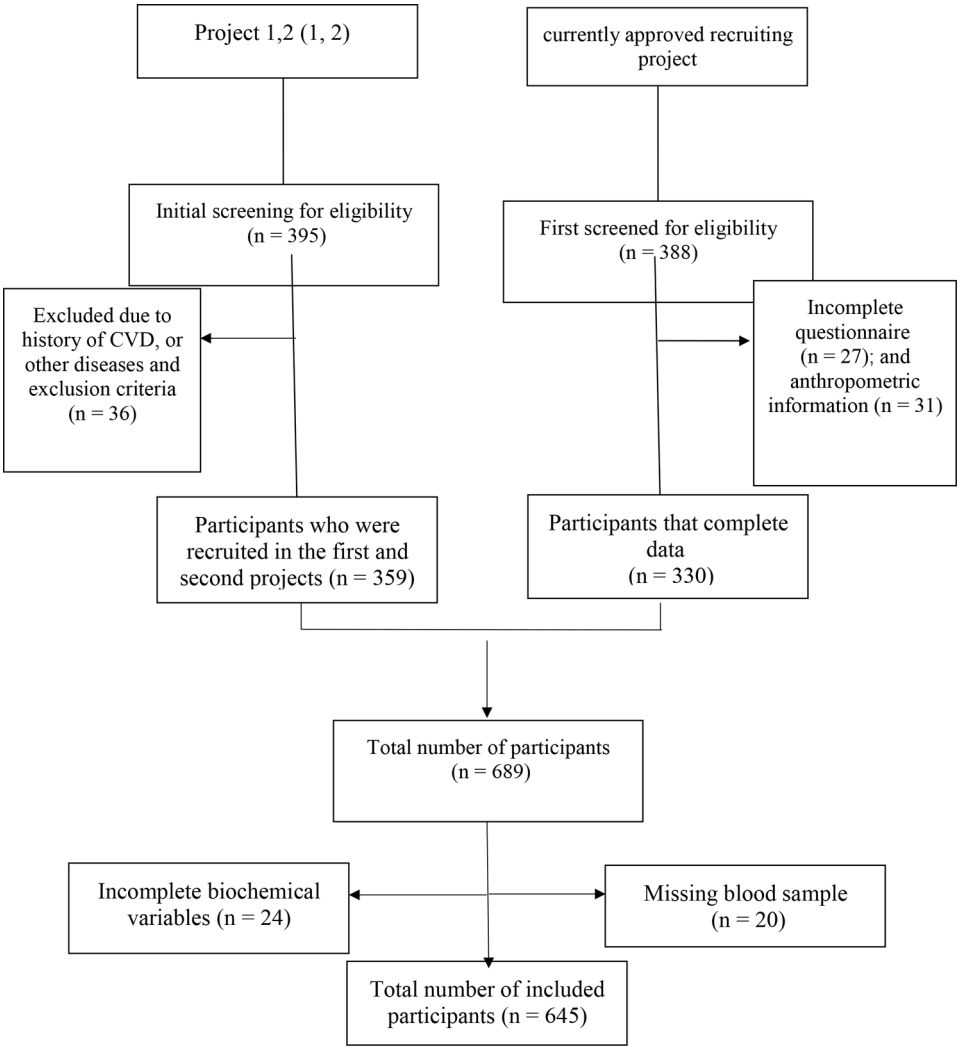

Fig. 1 Study Flowchart [21, 22]

Demographics and anthropometric evaluations

We used a questionnaire to collect participants' sociodemographic data, including age, gender, smoking habits, education level, marital status, employment, medical history, and family size. Education level was categorized using ordered categorical variables, ranging from illiterate (0) to higher education [5]. Similarly, occupational status was recorded using categories such as housewife, worker, student, freelancer, etc., for females, and without a job, rancher, farmer, worker, etc., for males. Family size was assigned scores of 1, 2, or 3 based on the number of family members. Participants were also given a score of 1 if they did not own a house and 2 if they did. We used bioelectrical impedance analysis (BIA) with the InBody 770 system (InBody Co., Ltd., Seoul, South Korea) specific equipment to assess body composition. Before measuring BIA, participants must follow several specific prerequisites to obtain accurate and reliable results from BIA: Avoid drinking large amounts of water for at least 4 h before the test. Avoid dehydration, which can affect the results. Do not eat a large meal within 4 h before the test. Avoid strenuous exercise for at least 12 h before the test as it can temporarily change body composition. Refrain from consuming alcohol for 48 h before the test [23]. Additionally, to adhere to established criteria, we conducted the test under standardized conditions, including performing the test at the same time of day for all participants. All subjects were required to fast before the test, and women were tested outside their menstrual periods. We also ensured that participants were normohydrated before testing to minimize variability in body composition measurements.

Height and weight were measured using a wall-mounted stadiometer and a Seca scale (Seca GmbH & Co. KG, Hamburg, Germany). The stadiometer had an accuracy of ± 0.1 cm, while the Seca scale used for weight measurement had an accuracy of ± 0.1 kg. These devices were regularly calibrated to ensure precise and consistent

measurements during the study. Hip circumference (HC) was measured at the widest part of the buttocks, and waist circumference (WC) was measured at the midpoint between the lowest rib and the hip bone [24]. We also calculated the waist-to-hip ratio (WHR) and BMI. A calibrated mercury sphygmomanometer was used to measure blood pressure twice at 15-minute intervals, and the average of the two readings was used for analysis. The participants' physical activity levels were assessed using the short form of the International Physical Activity Questionnaire (IPAQ) [17].

Dietary assessments and their reliability and validity

A validated semi-quantitative Food Frequency Questionnaire (FFQ) consisting of 168 questions was employed to gather dietary information from the Iranian population [18]. Participants maintained diaries in which they recorded the frequency and quantity of each food item consumed daily, weekly, monthly, and yearly. Participants were asked to keep these diaries for one week before completing the FFQ to ensure accurate recall and capture variations in their typical dietary patterns. The FFQs were completed through face-to-face interviews conducted by trained nutritionists, ensuring accuracy and consistency in responses. The amount of food consumed was converted into grams per day using standard portion sizes, cooking factors, and edible portions as defined in the Iranian household measures manual [19]. The Nutritionist IV software (N Squared Computing, California, USA) analyzed daily dietary intakes, including total energy, carbohydrates, fiber, proteins, fats, vitamins, and minerals. Given its reasonable relative validity and reproducibility correlations, this FFQ serves as a reliable tool for evaluating food group consumption and accurately ranking individuals based on their intake levels for each food group. The food items in the FFQ were categorized according to the nutrients they provided, including whole grains, refined grains, potatoes, dairy products, vegetables, fruits, legumes, meats, nuts and seeds, solid fat, liquid oil, tea and coffee, salty snacks, simple sugars, honey and jam, soft drinks, and desserts and snacks. Food items from the FFQ were converted into dietary fatty acids using the following formula: the intake of dietary fatty acids in food of each item = the intake of food of each item (g/d) × the content of dietary fatty acids in the edible part of the food (100 g)/100 g. The reference for fatty acids and energy content is based on the USDA food database [25].

The atherogenic index (AI) formula

The atherogenic index shows a relationship between the total amount of unsaturated and saturated fatty acids. Is the sum of C12:0 = Lauric acid, C14:0 = Myristic acid, C16:0 = Palmitic acid, ∑MUFA = sum of monounsaturated fatty acids, ∑ω-6 = sum of omega-6 polyunsaturated fatty acids, ∑ω-3 = sum of omega-3 polyunsaturated fatty acids [20].

$$(\mathrm{AI}) = \frac{[(C12:0 + (4xC14:0) + C16:0)]}{\left(\sum^{M} UFA + \sum^{\omega} -6 + \sum^{\omega} -3\right)}$$

Thrombogenic index (TI) formula

n-3 PUFA has greater anti-atherogenic properties than MUFA and n-6 PUFA. ∑Sn-6 = total omega-6 fatty acids, ∑Sn-3 = total omega-3 fatty acids, ∑MUFA = sum of monounsaturated fatty acids, and C14:0 = myristic acid, C16:0 = palmitic acid, and C18:0 = stearic acid [20].

$$(\mathrm{TI}) = \frac{[(C14:0 + C16:0 + C18:0)]}{\left[\begin{array}{l}(0.5 \times \sum^{M} UFA) + (0.5 \times \sum^{\omega} -6 \\ + \left(3 \times \sum^{\omega} -3\right) + \left(\sum^{\omega} -3 / \sum^{\omega} -6\right)\end{array}\right]}$$

Atherogenic index of plasma (AIP) formula

To calculate the logarithm of the ratio of triglyceride to HDL-C plasma concentration [15].

$$\mathrm{AIP} = \log\left[\frac{(TG)}{(HDL\_C)}\right]$$

Biochemical evaluation

For the biochemical analysis, 10 milliliters of fasting venous blood were taken from each participant. A commercial kit (Pars Azmoon, Tehran, Iran) measured the following parameters: TC, TG, HDL-C, and fasting blood glucose (FBG). Samples of plasma and serum were separated using centrifugation at 4,500 rpm for 10 min at 4 degrees Celsius. Aliquots were frozen at − 70 degrees C before the examination. The Friedewald equation was also used to determine the amount of LDL-C [26]. The blood's insulin levels were measured using enzyme-linked immunosorbent assay (ELISA) kits from Shanghai Korean Biotech, Shanghai City, China (Bioassay Technology Laboratory). Fasting insulin levels were assessed, and participants had to fast for at least 8 h before blood sample collection to ensure accurate measurements.

Statistical analysis

The data was analyzed using IBM SPSS version 19.0 software with a significance level of 0.05. The mean [standard deviation (SD)] and frequency (%) were used to characterize continuous variables and categorical variables, respectively. There were 645 participants in the final sample, 48.1% of whom were men and 51.7% of whom were women. To assess the relationship between AIP, TI, and AI and cardiometabolic risk variables, analysis of variance (ANOVA) was employed with *Tuke'y* post hoc test.

The effect of confounding variables (age, sex, BMI, total energy consumption) on the relationship between AIP, TI, AI, and cardiometabolic risk factors was controlled for using analysis of covariance (ANCOVA). The sample size was calculated with $\alpha = 0.05$ and $\beta = 0.2$. Therefore, the power was 80%. According to the power of 80%, categorizing the AIP, TI, and AI into tertiles was the best choice to avoid false positives due to multiple comparisons and false negatives due to inadequate power [27, 28].

Results

The general demographic and anthropometric features of study participants are represented in Table 1. As shown, for participants in higher tertiles of the AIP, the percentage of men was higher than women ($p < 0.001$). Also, in a crude model, individuals in higher tertiles of AIP had higher WHR, FFM, and BMR ($p < 0.001$, 0.04, and 0.02, respectively). However, FFM and BMR lost their significance level after adjusting for confounders, and WHR remained significant ($p < 0.001$). Also, those with higher dietary AI tertiles had higher weight and WC in a crude model ($p = 0.02$). These differences did not remain significant after adjustment for confounders. There was no significant difference in general characteristics and anthropometric variables among different tertiles of the TI group.

Table 2 compares biochemical variables across different AIP, TI, and AI tertiles in crude and energy, age, gender, and BMI-adjusted models. In the crude model of AIP, participants in the higher tertile had higher SBP, DBP, TC, TG, and glucose concentrations ($p < 0.001$, 0.02, and < 0.001 For all three variables respectively), and those in the lower tertile of AIP had higher HDL. In the adjusted model, all remained significant ($p < 0.001$ For all variables) except DPB. In the crude model of the TI, participants in the higher tertile had higher glucose concentrations ($p = 0.03$) that remained at a significance level after adjustment for confounders ($p = 0.04$). In the crude model of the AI, individuals in the lowest tertile had higher LDL that lost their significance level after adjustment for confounders. Table 3 compares dietary macronutrients and some micronutrients across different AIP, TI, and AI tertiles. As anticipated, there was a rise in nearly all of the food components in different tertiles. The comparison of food groups' intake across different tertiles of AIP, TI, and AI is shown in Table 4. There were no significant differences in terms of food groups across tertils of AIP, AI, and TI.

Discussion

As far as we know, this study was the first to investigate the connection between dietary and plasma indices and cardiometabolic risk factors in Iranian obesity patients. The research findings comprehensively investigate the relationship among anthropometric variables, metabolic parameters, and lipid-related indicators. Our results show interesting findings that provide insight into possible associations between these indices and cardiovascular risk factors.

This study observed a positive relationship between higher AIP tertiles and cardiovascular risk factors, aligning with several prior studies [29–31]. The demographic and anthropometric features of the study participants provide valuable insights into the distribution of AIP among different groups. A significant gender gap was found, with a significantly higher percentage of men in the upper tertiles of AIP. This finding emphasizes the potential role of gender in influencing atherogenic lipid profiles, suggesting that men may be more predisposed to adverse lipid profiles associated with cardiovascular risk. Similarly, the observed gender disparity in our study, with a higher percentage of men in the upper AIP tertiles, is consistent with research by J. Kim et al., suggesting a potential gender-specific influence on lipid profiles and cardiovascular risk [32]. Mechanistically, sex hormones play a crucial role in lipid homeostasis, and variations in hormonal profiles between men and women may contribute to the observed differences [33, 34].

Further investigation into the hormonal regulation of lipoprotein metabolism could provide a more comprehensive understanding. Moreover, our analysis of anthropometric measures indicated that individuals in higher AIP tertiles exhibited a higher WHR in both crude and adjusted models. The persistence of this association after adjusting for confounders emphasizes the independent contribution of AIP to central obesity, a vital risk factor for cardiovascular diseases [35]. This result aligns with prior studies. A cross-sectional study conducted by Wang et al. indicated that WHR has good performance for identifying moderate and high risk of AIP in familial hypercholesterolemia patients [36]. Also, Anandkumar M H et al. concluded that all anthropometric measures of obesity showed a significant correlation with AIP; however, WC showed the strongest correlation, followed by WHR and lastly [37]. Adipose tissue, particularly visceral fat, is known to release pro-inflammatory cytokines, influencing lipid metabolism. The inflammatory pathways activated by visceral adiposity may contribute to insulin resistance [38, 39], providing a potential mechanism for the observed association between AIP and WHR.

The analysis of biochemical variables across different tertiles of AIP, TI, and AI revealed strong associations that emphasize the potential role of these indices as markers of cardiovascular health. In the case of AIP, participants in higher tertiles exhibited elevated SBP, DBP, TC, TG, and glucose concentrations. These associations remained significant even after adjusting for confounders, highlighting the strength of the observed

Table 1 General characteristics and anthropometric measurements of study participants across different tertiles of AIP, TI, and AI

Variables	AIP					TI					AI				
	1st tertile)*n*=213)	**2nd tertile (*n*=212)**	**3rd tertile (*n*=220)**	***P**	****P**	**1st tertile)*n*=213)**	**2nd tertile (*n*=212)**	**3rd tertile (*n*=220)**	***P**	****P**	**1st tertile)*n*=213)**	**2nd tertile (*n*=212)**	**3rd tertile (*n*=220)**	***P**	****P**
Age (year)	39.77 (9.24)	40.19 (9.86)	40.34 (9.60)	0.81		39.10 (9.17)	38.37 (7.714)	38.00 (8.26)	0.60		38.67 (8.88)	38.30 (7.85)	38.80 (8.45)	0.90	
Education (≤ 12 y)	170 (79.9)	180 (84.9)	190 (83.8)	0.61***		192 (81.3)	186 (77.4)	151 (79.8)	0.44***		199 (80.7)	195 (77.3)	154 (73.7)	0.68***	
Marital status (% Single)	39 (18.3)	48 (22.6)	40 (18.2)	0.42***		18 (16.1)	28 (24.3)	21 (18.6)	0.33***		17 (15.6)	17 (15.5)	29 (26.4)	0.11***	
Gender (%Male)	84 (39.4)	104 (49.1)	124 (48.4)	**< 0.001*** **		52 (46.4)	56 (48.7)	55 (48.7)	0.92***		47 (43.1)	60 (54.5)	50 (45.5)	0.20***	
Weight (kg)	91.69 (13.55)	93.09 (15.09)	92.22 (13.92)	0.59	0.17	94.71 (12.63)	93.91 (13.04)	96.12 (14.49)	0.45	0.61	92.66 (12.26)	97.42 (14.10)	93.96 (13.10)	**0.02**	0.12
Height (cm)	164.17 (10.35)	166.16 (11.38)	166.83 (14.80)	0.06	0.87	165.90 (10.00)	165.47 (9.41)	165.69 (9.80)	0.94	0.88	165.20 (10.13)	166.75 (9.93)	164.79 (9.17)	0.28	0.90
BMI (kg/m^2)	33.73 (4.42)	33.28 (5.81)	33.30 (5.40)	0.60	0.05	34.27 (6.22)	34.05 (5.02)	35.30 (4.22)	0.16	0.55	34.28 (5.15)	34.75 (5.13)	34.70 (4.23)	0.73	0.85
WC (cm)	106.10 (9.64)	106.45 (9.88)	107.15 (9.89)	0.53	0.60	107.73 (9.44)	107.25 (10.53)	109.15 (9.88)	0.32	0.45	106.73 (9.58)	110.00 (10.16)	106.71 (9.59)	**0.02**	0.42
HC (cm)	117.08 (10.58)	115.16 (10.82)	115.33 (12.90)	0.17	0.29	114.44 (8.03)	115.12 (9.57)	116.20 (9.91)	0.35	0.25	114.38 (9.11)	116.09 (8.97)	115.19 (9.47)	0.39	0.71
WHR	0.91 (0.07)	0.93 (0.09)	0.93 (0.08)	**< 0.001**	**< 0.001**	0.94 (0.06)	0.93 (0.06)	0.94 (0.05)	0.54	0.45	0.93 (0.05)	0.94 (0.05)	0.92 (0.06)	0.05	0.19
FM (kg)	34.49 (8.06)	35.68 (10.56)	36.05 (10.96)	0.31	0.97	34.30 (8.36)	34.55 (9.08)	34.72 (9.91)	0.94	0.21	34.71 (8.29)	34.27 (8.32)	34.44 (10.59)	0.93	0.48
FFM (kg)	56.70 (12.99)	59.22 (14.18)	60.14 (12.28)	**0.04**	0.74	59.40 (14.31)	58.82 (13.31)	62.56 (12.34)	0.08	0.30	58.48 (13.07)	62.32 (13.80)	60.06 (11.85)	0.09	0.58
BMR (kj)	7435.30 (1687.44)	8004.64 (1435.89)	7730.61 (1622.06)	**0.02**	0.47	7682.91 (1512.02)	7483.89 (1666.06)	7909.33 (1630.76)	0.14	0.30	7452.10 (1601.90)	7942.1 6 (1637.84)	7604.56 (1559.10)	0.07	0.72

BMI, Body mass index; BMR, Basal Metabolic Rate; FM, Fat Mass; FFM, Fat-Free Mass; LC-CFA, long chain combined fatty acids; PUFA, polyunsaturated fatty acid; SES, SFA, saturated fatty acid; WC, Waist Circumference; WHR, waist-to-hip ratio. All data are mean (± SD) except marital status and gender, presented as the number and percent of single and males in each group. * P values derived from One-Way ANOVA. ** P values derived from ANCOVA after adjustment for confounders (age, gender, BMI, physical activity, and energy intake). *** P values derived from chi-squared test

Table 2 Biochemical parameters of study participants across different tertiles of AIP, TI, and AI

Variables	AIP					TI					AI				
	1st tertile (*n* = 213)	2nd tertile (*n* = 212)	3rd tertile (*n* = 220)	*P*	**P*	1st tertile (*n* = 213)	2nd tertile (*n* = 212)	3rd tertile (*n* = 220)	*P*	**P*	1st tertile (*n* = 213)	2nd tertile (*n* = 212)	3rd tertile (*n* = 220)	*P*	**P*
SBP (mmHg)	116.79 (15.49)	122.30 (15.87)	123.33 (16.89)	**< 0.001**	**< 0.001**	115.35 (14.88)	114.22 (13.02)	118.24 (18.88)	0.14	0.20	115.23 (13.72)	116.23 (14.56)	116.91 (18.56)	0.73	0.80
DBP (mmHg)	78.17 (11.50)	79.35 (11.55)	81.66 (12.30)	**0.02**	0.12	76.25 (11.46)	75.26 (10.96)	77.63 (12.93)	0.31	0.43	76.44 (11.44)	77.20 (10.96)	75.72 (12.56)	0.64	0.59
TC (mg/dL)	184.46 (43.43)	189.12 (38.82)	200.03 (41.96)	**< 0.001**	**< 0.001**	199.00 (45.78)	195.24 (44.26)	188.92 (45.66)	0.24	0.40	203.03 (46.65)	188.39 (43.64)	191.49 (44.04)	0.04	0.06
TG (mg/dL)	71.33 (25.64)	125.36 (27.54)	220.29 (94.01)	**< 0.001**	**< 0.001**	131.60 (74.94)	113.87 (67.43)	118.40 (60.96)	0.12	0.11	124.37 (71.52)	120.06 (74.02)	119.21 (59.89)	0.83	0.72
HDL-C (mg/dL)	50.87 (11.40)	44.11 (8.68)	35.94 (7.76)	**< 0.001**	**< 0.001**	45.54 (11.40)	45.02 (12.36)	44.96 (10.31)	0.91	0.91	44.87 (11.82)	46.10 (11.04)	44.93 (11.29)	0.32	0.28
LDL-C (mg/dL)	120.15 (42.22)	122.57 (32.33)	125.64 (38.06)	0.31	0.34	127.68 (39.03)	127.60 (43.28)	120.58 (42.61)	0.34	0.57	133.48 (42.41)	118.64 (40.17)	124.29 (42.08)	**0.03**	**0.05**
Glucose (mg/dL)	92.55 (15.26)	94.44 (22.02)	96.14 (23.93)	**< 0.001**	**< 0.001**	96.95 (22.51)	100.99 (22.16)	106.28 (33.69)	**0.03**	**0.04**	99.72 (20.82)	104.18 (33.36)	101.06 (25.84)	0.46	0.46
Insulin (μIU/mL)	15.96 (15.81)	16.70 (24.29)	16.83 (14.96)	0.88	0.97	19.23 (29.04)	15.41 (9.88)	17.82 (18.53)	0.37	0.40	15.58 (9.16)	19.45 (29.27)	17.48 (19.09)	0.39	0.41

SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; TC, Total Cholesterol; TG, Triglyceride; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; QUICKI, Quantitative Insulin sensitivity Check Index, SFA, saturated fatty acid; PUFA, polyunsaturated fatty acid; LC-CFA, long chain combined fatty acids. *P values derived from One-Way ANOVA. ** P values derived from ANCOVA after adjustment for confounders (age, gender, BMI, physical activity, and energy intake)

relationships. However, the significance of DBP diminished in the adjusted model, suggesting that additional factors may influence some of the observed associations. Consistent with our results, studies by Moussavi Javardi et al., Wu et al., and Su Bo et al. reported a significant association between elevated AIP and increased SBP, DBP, TC, TG, and glucose levels [40–42]. Mechanistically, AIP reflects the balance between atherogenic and anti-atherogenic lipoproteins [43]. Elevated AIP often correlates with increased LDL, TG, and decreased HDL, contributing to a pro-atherogenic lipid profile [44]. Studies have implicated inflammatory pathways and insulin resistance as potential mechanistic links, emphasizing the predictive value of AIP in identifying individuals at risk of cardiovascular events [45, 46].

Another study finding demonstrated that a higher tertile of TI was associated with higher glucose concentrations. The observed association, in concordance with the research conducted by Takato et al., is that C14:0 reduces body weight and insulin-responsive glucose levels to alleviate hyperglycemia. The ability of high-fat dairy products to protect against diabetes is attributed to this fatty acid. One possible option for treating and preventing type 2 diabetes mellitus and associated conditions is C14:0 [47]. In contrast, Pu al. indicate that in skeletal muscle cells, C16:0 activates Akt and ERK1/2 to increase glucose absorption sharply, and a clinical trial conducted by Louheranta al. shows that a high-stearic acid diet does not impair glucose tolerance and insulin sensitivity in healthy women [48]. On the other hand, an animal study showed that the high-fat diet with a high C18:0/C16:0 ratio induced more severe glucose and lipid metabolic disorders and inflammation [49].

Possible mechanisms are as follows: Saturated fatty acids, particularly C16:0 and C18:0, have been implicated in impairing insulin sensitivity [49]. These fatty acids can induce cellular stress and activate inflammatory pathways, reducing insulin responsiveness in target tissues such as muscle and adipose tissue [50]. Excessive levels of some saturated fatty acids might cause inflammatory reactions [51]. Disturbances in glucose metabolism and insulin resistance are closely associated with inflammation. Pro-inflammatory pathways may activate and disrupt the insulin signaling cycle, hindering cells' ability to absorb glucose [52].

The other outcome of this study, the association between the AI and LDL levels in the crude model, which loses significance after adjusting for confounders, differs from specific previous research on the connection between adverse lipid profiles and AI Kazemi et al. show that age, body mass index, sex, as well as CRI and AI had affirmative correlation with TC, LDL-C, TAG, SBP, and DBP [53]. The differences could result from differences in the sample size, genetic variables, or lifestyle impacts

Table 3 Dietary intake of participants across different tertiles of AIP, TI, and AI

Variables	AIP					TI					AI				
	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P	**P	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P	**P	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P	**P
Protein (g/day)	89.38 (34.38)	91.57 (35.36)	95.52 (34.76)	0.18		93.87 (36.62)	101.58 (33.28)	98.49 (33.65)	0.24		95.83 (33.37)	98.40 (33.90)	99.69 (36.18)	0.70	
Fat (g/day)	101.48 (45.97)	97.46 (42.62)	96.18 (45.18)	0.44		102.92 (46.53)	111.83 (44.04)	111.49 (49.93)	0.27		102.16 (45.26)	111.50 (42.41)	111.55 (50.94)	0.22	
CHO (g/day)	605.59 (287.79)	416.14 (165.03)	424.28 (169.82)	0.41		422.62 (162.56)	826.93 (393.92)	451.18 (171.34)	0.33		427.51 (160.21)	459.15 (174.21)	823.93 (399.24)	0.37	
Total Fiber (g/day)	63.63 (36.53)	63.21 (42.07)	59.18 (36.17)	0.42	0.10	68.94 (43.51)	69.92 (37.87)	77.36 (51.98)	0.30	0.84	68.35 (41.73)	75.74 (47.40)	71.49 (45.01)	0.47	0.70
SFA (g/day)	28.15 (11.82)	28.54 (12.07)	29.63 (17.20)	0.52	0.24	28.92 (12.20)	31.13 (12.38)	32.08 (18.52)	0.25	0.12	29.24 (12.68)	30.23 (11.42)	32.65 (18.84)	0.21	0.15
MUFA (g/day)	34.35 (17.04)	32.21 (14.87)	31.86 (15.94)	0.22	0.10	34.36 (16.97)	37.07 (16.48)	37.39 (18.48)	0.35	0.23	33.53 (16.29)	36.82 (15.80)	38.14 (19.38)	0.12	0.15
PUFA(g/day)	24.71 (14.68)	21.82 (11.83)	20.36 (10.79)	< 0.001	<0.001	24.78 (14.50)	26.44 (13.79)	25.38 (12.36)	0.64	0.80	24.01 (13.26)	26.61 (13.82)	25.66 (13.14)	0.35	0.59
Cholesterol (mg/day)	282.27 (205.06)	287.78 (219.62)	279.85 (147.10)	0.91	0.76	315.06 (256.35)	344.39 (268.50)	293.19 (148.51)	0.24	0.28	322.48 (263.65)	319.38 (166.05)	293.45 (156.45)	0.50	0.42
Sodium (mg/day)	4565.05 (2050.24)	4594.29 (2358.21)	4376.87 (1966.39)	0.52	0.20	4792.71 (2285.87)	4970.87 (2173.87)	5025.35 (2268.61)	0.71	0.64	4792.07 (2217.12)	5074.42 (2311.64)	4892.45 (2183.11)	0.63	0.84
Iron (mg/day)	22.13 (11.38)	21.82 (9.50)	22.66 (11.22)	0.71	0.62	22.77 (9.55)	25.20 (13.10)	25.19 (13.64)	0.22	0.14	23.10 (9.60)	25.28 (13.49)	24.66 (13.28)	0.40	0.68
Magnesium (mg/day)	484.01 (239.21)	481.33 (187.61)	517.25 (221.60)	0.16	0.01	503.00 (207.44)	548.97 (205.25)	530.17 (205.12)	0.24	0.17	521.59 (212.61)	531.77 (194.39)	528.24 (208.01)	0.93	0.82
Zinc (mg/day)	14.20 (14.86)	13.33 (5.46)	14.15 (6.05)	0.58	0.79	13.76 (5.55)	15.35 (5.62)	14.35 (5.33)	< 0.001	0.06	14.41 (5.47)	14.42 (5.34)	14.65 (5.70)	0.93	0.62
Phosphorus (mg/day)	1573.87 (604.62)	1629.86 (620.16)	1728.51 (699.57)	0.04	< 0.001	1655.39 (640.37)	1810.10 (613.56)	1759.63 (635.07)	0.17	0.09	1721.78 (630.80)	1756.89 (633.22)	1748.30 (635.08)	0.91	0.89
Calcium (mg/day)	1123.72 (505.82)	1162.77 (514.41)	1201.03 (630.05	0.35	0.04	1185.34 (49.90)	498.59 (46.90)	566.17 (53.02)	0.12	0.03	1237.22 (533.47)	1304.49 (557.26)	1263.99 (514.45)	0.64	0.86
Potassium (mg/day)	4152.24 (2021.78)	4122.28 (1722.05)	4416.76 (2158.73)	0.24	0.07	4245.25 (1705.07)	4629.96 (1908.72)	4597.31 (2017.93)	0.23	0.11	4364.94 (1845.75)	4558.55 (1837.29)	4546.81 (1974.80)	0.69	0.97
Copper (mg/day)	2.30 (1.20)	2.21 (1.03)	2.34 (1.10)	0.48	0.61	2.36 (1.14)	2.57 (1.19)	2.54 (1.25)	0.35	0.29	2.50 (1.31)	2.52 (1.18)	2.45 (1.10)	0.91	0.59
Manganese (mg/day)	8.30 (3.63)	8.24 (3.63)	8.73 (3.70)	0.31	0.20	8.68 (3.77)	9.18 (3.66)	9.03 (4.00)	0.59	0.64	8.98 (4.13)	9.11 (3.57)	8.77 (3.69)	0.79	0.51
Selenium (mg/day)	136.54 (51.09)	143.91 (65.93)	150.53 (59.18)	0.05	0.51	147.25 (58.88)	156.09 (57.27)	150.55 (60.57)	0.52	0.65	151.84 (59.02)	153.55 (58.48)	148.71 (58.99)	0.82	0.53
Fluorine (mg/day)	3771.75 (2937.32)	3278.82 (2419.96)	3189.08 (2835.62)	0.06	0.07	3901.75 (2898.24)	3691.54 (2420.26)	4198.84 (4064.95)	0.48	0.35	3958.46 (3735.48)	4012.31 (2674.01)	3784.85 (3144.34)	0.86	0.77
Chromium (mg/day)	8.72 (125.20)	0.14 (0.11)	0.16 (0.12)	0.37	0.36	0.13 (0.10)	0.14 (0.10)	0.13 (0.10)	0.34	0.41	0.14 (0.11)	0.13 (0.09)	0.13 (0.10)	0.77	0.63

Table 3 (continued)

Variables	AIP					TI					AI				
	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P*	**P*	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P*	**P*	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P*	**P*
Vitamin C (mg/day)	183.88 (150.62)	183.66 (144.30)	213.92 (181.34)	0.08	0.05	171.73 (97.49)	192.23 (141.39)	208.64 (144.21)	0.10	0.02	181.63 (113.29)	196.78 (152.10)	193.17 (123.22)	0.67	0.92
VitaminB1 (mg/day)	2.46 (1.05)	2.48 (1.13)	2.50 (1.04)	0.91	0.40	2.56 (1.19)	2.74 (1.15)	2.76 (1.23)	0.37	0.21	2.59 (1.12)	2.82 (1.31)	2.65 (1.12)	0.34	0.41
VitaminB2 (mg/day)	2.29 (0.97)	2.35 (1.02)	2.38 (1.08)	0.67	0.13	2.47 (1.11)	2.66 (0.94)	2.65 (1.07)	0.29	0.19	2.55 (1.04)	2.70 (1.12)	2.53 (0.96)	0.45	0.40
VitaminB3 (mg/day)	26.8 (9.88)	27.48 (11.49)	28.79 (10.88)	0.16	< 0.001	28.18 (11.54)	29.72 (10.72)	30.16 (12.25)	0.39	0.17	28.38 (10.74)	30.38 (12.15)	29.35 (11.49)	0.43	0.68
VitaminB6 (mg/day)	2.06 (0.90)	2.07 (0.79)	2.22 (1.01)	0.12	0.01	2.09 (0.77)	2.35 (1.00)	2.31 (1.01)	0.08	0.02	2.11 (0.79)	2.32 (1.01)	2.29 (1.00)	0.22	0.37
VitaminB9 (µg/day)	693.14 (289.45)	675.40 (297.71)	674.20 (271.83)	0.75	0.58	721.58 (320.78)	751.57 (314.27)	775.53 (342.26)	0.45	0.15	727.41 (327.62)	773.53 (333.74)	741.51 (313.04)	0.56	0.71
VitaminB12 (µg/day)	5.07 (6.08)	4.74 (4.27)	5.27 (6.06)	0.61	0.70	5.40 (6.05)	6.65 (7.48)	5.94 (7.27)	0.40	0.44	6.60 (8.96)	5.85 (6.38)	5.57 (5.28)	0.53	0.45
VitaminB5 (mg/day)	6.07 (2.36)	6.15 (2.42)	6.53 (2.71)	0.09	< 0.001	6.22 (2.21)	7.05 (2.68)	6.64 (2.39)	0.04	0.01	6.47 (2.33)	6.81 (2.45)	6.68 (2.62)	0.59	0.90
VitaminB8 (mg/day)	35.53 (16.83)	36.74 (16.85)	39.48 (19.82)	0.06	< 0.001	37.43 (15.24)	40.86 (17.91)	40.54 (19.46)	0.27	0.21	39.24 (17.83)	39.94 (16.87)	39.68 (18.62)	0.95	0.89
Vitamin A (RAE/day)	824.367 (701.08)	784.44 (525.31)	878.06 (683.98)	0.32	0.37	891.43 (673.56)	990.75 (770.88)	988.04 (827.66)	0.53	0.57	985.78 (909.58)	979.80 (743.30)	907.20 (624.03)	0.70	0.57
Vitamin D (µg/day)	1.92 (1.53)	1.95 (1.60)	2.02 (1.51)	0.81	0.76	2.25 (1.77)	2.44 (1.89)	2.28 (1.61)	0.68	0.77	2.30 (1.69)	2.42 (1.98)	2.29 (1.61)	0.82	0.87
Vitamin K (µg/day)	185.36 (155.17)	224.39 (153.78)	336.66 (323.78)	0.45	0.60	259.11 (244.10)	284.54 (235.41)	311.15 (302.72)	0.32	0.25	259.22 (253.16)	290.85 (288.95)	290.27 (211.10)	0.26	0.77
Vitamin E (mg/day)	17.43 (9.99)	15.11 (7.87)	14.83 (7.27)	< 0.001	< 0.001	16.96 (9.55)	18.59 (10.73)	18.24 (9.88)	0.44	0.47	16.58 (9.36)	18.00 (9.67)	17.77 (9.84)	0.57	0.44

CHO, carbohydrate, SFA, saturated fatty acids, MUFA, mono-unsaturated fatty acids; PUFA, polyunsaturated fatty acids. All data are mean (± SD). * P values derived from One-Way ANOVA. ** P values derived from ANCOVA after adjustment for confounder (energy intake)

Table 4 Food group intake of study participants across different tertiles of AIP, TI, and AI

Variables	AIP					TI					AI				
	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P*	**P*	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P*	**P*	T1 (*n* = 213)	T2 (*n* = 212)	T3 (*n* = 220)	*P*	**P*
Fruits (g/d)	507.59 (460.47)	504.55 (419.21)	599.53 (603.25)	0.08	0.09	477.22 (292.61)	547.69 (453.85)	559.63 (414.43)	0.23	0.21	499.24 (327.52)	550.56 (458.00)	547.96 (397.44)	0.56	0.77
Vegetables (g/d)	306.79 (257.49)	281.49 (182.81)	319.43 (265.19)	0.24	0.36	287.34 (176.63)	341.99 (309.17)	355.80 (347.15)	0.16	0.13	314.55 (292.42)	330.49 (255.91)	330.78 (320.68)	0.89	0.99
MFP (g/d)	61.56 (37.22)	63.02 (46.76)	68.24 (46.49)	0.25	0.24	62.63 (45.90)	70.16 (49.23)	63.74 (38.42)	0.39	0.47	61.57 (39.16)	64.84 (42.14)	70.61 (52.70)	0.32	0.37
Dairy (g/d)	268.07 (208.25)	292.33 (220.13)	315.98 (272.26)	0.11	0.05	292.02 (222.70)	322.90 (220.37)	311.94 (214.44)	0.56	0.64	311.50 (225.22)	316.44 (239.17)	303.46 (195.85)	0.90	0.85
Grains (g/d)	538.42 (364.54)	546.07 (374.80)	536.16 (234.69)	0.94	0.83	561.22 (317.99)	601.03 (467.84)	587.99 (386.92)	0.74	0.78	607.06 (517.12)	578.36 (257.03)	538.52 (234.49)	0.36	0.26
Nuts(g/day)	16.51 (40.95)	14.41 (22.72)	22.63 (119.12)	0.49	0.51	64.68 (63.34)	72.92 (72.68)	67.07 (58.23)	0.46	0.47	64.39 (66.23)	61.54 (56.79)	77.25 (69.82)	0.15	0.31
Beans(g/day)	59.79 (64.58)	51.14 (51.39)	48.52 (50.42)	0.09	0.08	30.90 (159.43)	19.85 (23.94)	16.11 (19.57)	0.61	0.74	34.09 (162.35)	16.52 (20.49)	17.08 (21.28)	0.30	0.11
Fiber (g/day)	63.63 (36.53)	63.21 (42.07)	59.18 (36.17)	0.42	0.10	68.94 (43.51)	69.92 (37.87)	77.36 (51.98)	0.30	0.84	68.35 (41.73)	75.74 (47.40)	71.49 (45.01)	0.47	0.70

MFP, meat, fish, and poultry; All data are mean (± SD). * P values derived from One-Way ANOVA. ** P values derived from ANCOVA after adjustment for confounder (energy intake)

that needed to be fully considered in this study, leading to more research.

Certain limitations should be considered when interpreting our findings. Firstly, our study was cross-sectional, so we cannot establish a causal relationship between dietary and plasma indices and cardiometabolic risk factors. We need further research with a prospective design to truly understand the direction of the association between dietary and plasma indices and cardiometabolic risk factors. Secondly, even though we used a validated FFQ to assess dietary and fatty acid intakes, the closed-ended format of the questionnaires may have increased the chances of misclassification [54]. Nevertheless, any misclassifications would likely have a neutral effect on the odds ratios. Lastly, despite our efforts to control various confounding factors in our study, we must partially rule out the potential influence of residual confounders.

In summary, this research investigated the relationship between dietary and plasma indices and cardiometabolic risk factors in the population of Iran. Our study provides comprehensive insights into the associations between atherogenic indices and various demographic, anthropometric, and biochemical variables. The observed gender differences, the independent association of AIP with central obesity, and the links between AIP, TI, and biochemical markers underscore the complexity of cardiovascular risk factors. More investigation is necessary to confirm the potential efficacy of these indices as indicators of cardiovascular outcomes and clarify the underlying mechanisms.

Conclusion

Our findings suggest that dietary and plasma indices, particularly atherogenic indices like AIP and TI, could be effective indicators of cardiometabolic health in the Iranian population. By identifying associations with central obesity, lipid profiles, and other metabolic risk factors, this study highlights the potential of these indices for early detection and risk stratification in clinical settings. Implementing routine assessment of these indices may aid healthcare professionals in developing targeted prevention and intervention strategies for cardiovascular diseases, especially in populations with similar dietary patterns and lifestyle factors. However, further research is essential to confirm these associations and establish these indices as reliable predictive tools for cardiovascular outcomes.

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Author contributions
All authors approved the final version of the article. MAF contributed to study design, supervision, statistical analysis, and manuscript writing. RM was involved in revision and English language revision, she also wrote the first draft of manuscript and was involved in data collection and statistics. Both authors performed the statistical analysis and were involved in hypothesis generation.

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Data availability
The datasets generated and analyzed during the current study are not publicly available due to privacy and ethical considerations but can be available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
Ethics approval for this study was obtained from the Ethics Committee of Tabriz University of Medical Sciences. The full approval details, including the reference number (IR.TBZMED.REC.1403.836), were reviewed and confirmed by the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants before their inclusion in the study. All subjects provided written informed consent before participation in the study. For illiterate participants, their legal guardians provided written informed consent, and the participants themselves either provided verbal consent, documented in the presence of a witness or confirmed their consent via a thumbprint. All methods were carried out by the ethical principles outlined in the Declaration of Helsinki's guidelines and regulations.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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