BMC Endocrine Disorders

RESEARCH

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Metabolic syndrome in association with novel dietary index, metabolic parameters, nesfatin-1 and omentin-1

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Abstract

Background Metabolic syndrome is a prevalent and common health problem and numerous studies have revealed the role of diet and lifestyle change in prevention of metabolic syndrome. However, the novel dietary index, cardioprotective index (CPI) and its association with metabolic syndrome is not investigated yet. In the current study, we evaluated the association between metabolic syndrome and its components, CPI, Nesfatin-1 and Omentin-1in a cross-sectional study.

Methods Three hundred forty eight overweight and obese individuals with metabolic syndrome were recruited. Subjects underwent anthropometric and laboratory assays including metabolic markers, Nesfatin-1 and Omentin-1 with commercial kits.

Results Those at the first tertile of CPI had lower high density lipoprotein concentrations (HDL) and higher low density lipoprotein concentrations (LDL), triglyceride (TG), systolic and diastolic blood pressures (SBP, DBP) levels compared with those at the highest tertiles ($P < 0.05$). After adjustment for the confounding effects of age, sex, body mass index, physical activity and total calorie intake, LDL lost its significance across CPI tertiles. Moreover, serum total cholesterol, insulin and insulin resistance were not significant across CPI tertiles neither in crude nor in adjusted models ($P > 0.05$). Additionally, being at the third tertile of CPI was accompanied with significantly higher Nesfatin-1 and Omentin-1 levels compared with lowest tertiels ($P < 0.05$) in crude and confounder – adjusted models.

Conclusions: To our findings, CPI was in positive relationship with metabolic parameters, blood pressure, Nesfatin-1 and Omentin-1 levels in metabolic syndrome. Further future studies will help to elaborate the causality.

Clinical trial number Not applicable.

Keywords Cardio-protective index, CPI, Metabolic syndrome, Nesfatin-1, Omentin-1

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Introduction

Metabolic syndrome represents a complex constellation of interconnected metabolic abnormalities, including central obesity, insulin resistance, dyslipidemia, and hypertension, collectively predisposing individuals to an elevated risk of cardiovascular diseases (CVDs) and type 2 diabetes mellitus (T_2DM) [1, 2]. As a multifactorial disorder of global significance, the prevalence of metabolic syndrome has surged in recent decades, paralleling the worldwide epidemic of obesity and sedentary lifestyles [1, 3]. Consequently, there is an urgent imperative to identify robust biomarkers and predictive indices capable of elucidating the intricate pathophysiology underlying metabolic syndrome and its associated cardio-metabolic complications.

Diet is an important modifiable risk factor of chronic non-communicable disease (NCDs) including metabolic syndrome and dietary modifications has been consistently known as strong preventive strategies against metabolic syndrome [4]; for the prevention and management of metabolic syndrome, it is recommended to increase the daily consumption of fiber-rich foods, fish, dairy products and nuts and to increase the intake of unprocessed cereals, legumes, and fruit. Replacing saturated fatty acids with monounsaturated and polyunsaturated fatty acids and limiting the consumption of free sugars are highly recommended [5].

In this context, the cardio-protective index (CPI) has emerged as a promising tool for risk stratification and prognostication of metabolic disorders. Unlike conventional risk assessment algorithms that predominantly rely on individual risk factors, the CPI integrates multiple dietary parameters to provide a comprehensive evaluation of cardiovascular health and metabolic status. First suggested by Ponce X et al. [6], CPI is based on seven dietary guidelines of world health organization (WHO) for the prevention of cardiovascular diseases [7]; considering a recommended intake of dietary protein ≥ 10% of total dietary energy intake, total fat ≤ 30% total dietary energy intake, saturated fatty acids (SFAs) ≤ 10% total dietary energy intake, polyunsaturated fatty acids (PUFAs) < 10% total dietary energy intake, cholesterol < 300 mg/d, fiber ≥ 20 g/d, and a fruit and vegetable intake ≥ 400 g per day.

The significance of the cardio-protective index lies not only in its ability to quantify cardiovascular risk but also in its potential to delineate distinct metabolic phenotypes and predict the development of metabolic complications among individuals with cardio-metabolic risk factors [8]. Recent studies have underscored the utility of the cardio-protective diet as a potent predictor of adverse cardiovascular events, including myocardial infarction, stroke, and heart failure, independent of traditional risk factors [9, 10]. Moreover, its role in identifying high-risk metabolic phenotypes characterized by visceral adiposity, insulin resistance, and dyslipidemia highlights its clinical relevance in risk stratification and therapeutic decision-making [11].

Furthermore, emerging evidence suggests a potential link between the dietary behaviors and novel adipokines, Nesfatin-1 and Omentin-1 in metabolic syndrome, which have garnered attention for their pleiotropic effects on energy homeostasis, insulin sensitivity, and cardiovascular function [12, 13]. Studies show that high fat diet reduces Nesfatin-1 levels [14]; Nesfatin-1 suppresses food intake and consequently, improves glucose metabolism, through the suppression of food intake and body weight gain [15].

Nesfatin-1, derived from nucleobindin-2 (NUCB2), has garnered considerable attention for its diverse physiological roles in metabolic regulation, energy homeostasis, and cardiovascular function [12]. Initially identified as an anorexigenic peptide localized within the hypothalamus, Nesfatin-1 exerts pleiotropic effects on appetite suppression, food intake modulation, and body weight regulation through central and peripheral mechanisms [12].

Beyond its role in energy balance, emerging evidence suggests that Nesfatin-1 plays a pivotal role in the pathophysiology of metabolic syndrome and its associated cardiovascular complications [12, 16, 17]. Nesfatin-1 expression has been shown to be dysregulated in obesity, insulin resistance, and dyslipidemia, implicating its involvement in the development and progression of metabolic disorders [18, 19]. Notably, Nesfatin-1 exhibits anti-inflammatory properties and may exert protective effects against endothelial dysfunction, oxidative stress, and atherosclerosis, thus contributing to its cardio-protective potential [20].

Interestingly, the relationship between Nesfatin-1 and the cardio-protective diet remains relatively unexplored, presenting a compelling avenue for further investigation. Very limited number of studies have revealed its association with Mediterranean diet [21, 22] and no study is available to evaluate its association with the CPI or a cardio-protective diet.

Nesfatin-1 and Omentin-1 highly correlate each-other in regulation of metabolic pathways [23]; Omentin-1, also known as intelectin, is a fascinating protein; primarily produced by visceral adipose tissue, Omentin-1 exhibits anti-inflammatory properties and plays a crucial role in metabolic regulation [13]. Its involvement in insulin sensitivity and glucose metabolism has sparked interest in its potential as a therapeutic target for metabolic disorders such as metabolic syndrome, diabetes, obesity and cardiovascular disease [24]. Omentin-1 has been found to have implications in metabolic regulation, insulin sensitivity, and glucose metabolism, all of which are central to the development and progression of metabolic syndrome

[13, 24]. Studies have shown that Omentin-1 levels are often altered in individuals with metabolic syndrome, with lower levels observed in those who are obese, insulin resistant, or have type 2 diabetes [25]. Additionally, Omentin-1's anti-inflammatory properties may play a role in mitigating the chronic low-grade inflammation characteristic of metabolic syndrome [26]. It is suggested that circulating adiponectin mediates the association between Omentin-1 and cardio-metabolic health [27].

Understanding the interplay between these adipokines and the cardio-protective index may offer novel insights into the pathogenesis of metabolic syndrome and inform the development of targeted pharmaco-therapeutic agents aimed at mitigating cardio-metabolic complications. To our literature review, only one study evaluated the compliance to CPI ingredients in general population [6] and the role of CPI in metabolic syndrome and its association with metabolic risk factors is not studied yet. In the current research, we elucidate the clinical utility of the cardio-protective index as a potent predictor of metabolic phenotypes, high blood pressure, Nesfatin-1, and Omentin-1 among patients diagnosed with metabolic syndrome. Therefore, our primary outcome was to measure CPI among subjects with metabolic syndrome and to investigate the association between CPI and components of metabolic syndrome including glycemic markers, blood pressure, serum lipids, Nesfatin-1 and Omentin-1 in patients with metabolic syndrome.

Subjects and methods

Participants and recruitment

In the current cross-sectional study, subjects were included if they had overweight or obesity (BMI≥25 kg/m2), and were diagnosed with metabolic syndrome according to diagnostic criteria of National cholesterol Education Adult Treatment Panel III (NCEP-ATP III) revised form for Iranians, aged between 18 and 65 years old and had willingness to participate in the research. Accordingly, metabolic syndrome was defined as if there were ≥3 of the following conditions: (1) waist circumference (WC)≥90 cm for both genders; (2) Elevated triglyceride level≥150 mg/dl; (3) Reduced HDL-C<40 mg/dL in men; < 50 mg/dL in women; (4) Systolic blood pressure≥130 mmHg or diastolic blood pressure≥85 mmHg; (5) Elevated plasma glucose (FPG)≥100 mg/dl [28]. The cut-off points for WC was based on previous studies that suggested a WC≥90 cm as the more suitable WC for Iranians for identification of metabolic syndrome [29, 30]. Exclusion criteria were having cardiovascular disorders, cancer, liver and kidney disorders, or taking any medication that could affect the stud results. Eligible patients were recruited consecutively from the outpatient endocrinology clinics. Research personnel explained the study objectives, procedures, and potential risks and benefits to potential participants. Informed consent was obtained from all participants prior to enrollment. Study flowchart is provided in Fig. 1.

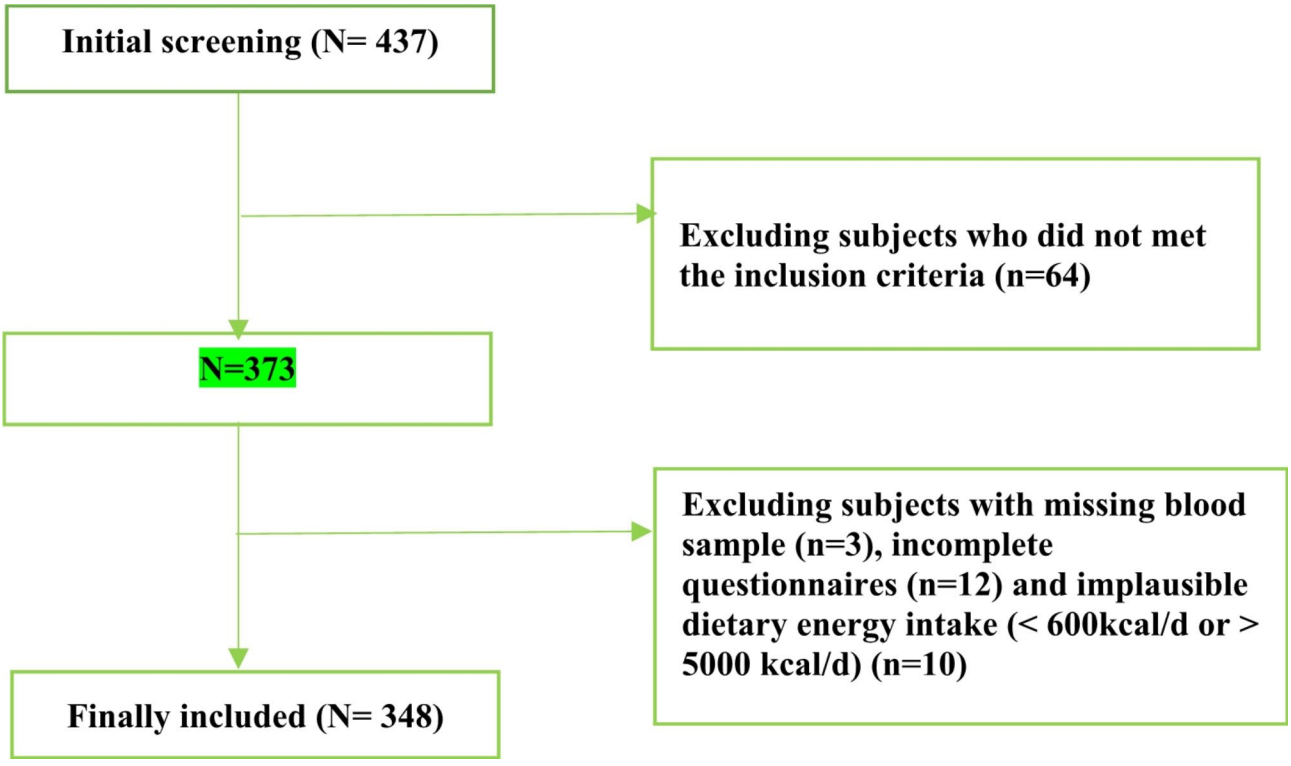

Fig. 1 Study flowchart

Sample size calculation was done based on the formula of $n= \frac{Z^2 P (1-P)}{E^2}$, from previous study [6] by Ponce X et al., where n = calculated sample size, Z = Z-score of 1.96 for 95% confidence level, P = estimated prevalence of metabolic syndrome and E = desired margin of error (5%). The calculated sample size was 313 subjects. Considering a 10% drop-out rate, 348 subjects were enrolled.

Data collection

a. ***Anthropometric measurements***.

Weight was assessed using a calibrated scale with a precision of 0.1 kg. Participants were asked to stand barefoot on the scale platform while remaining motionless. Height was measured using an anthropometer, ensuring participants stand upright against a wall with heels, buttocks, shoulders, and head touching the wall. The anthropometer was gently lowered onto the participant's head, and the measurement was recorded to the nearest 0.1 cm. Waist Circumference (WC) was measured using a non-stretchable tape measure. Participants were stand relaxed with feet together, and the circumference was taken at the midpoint between the lower rib margin and the iliac crest. Measurements were recorded to the nearest 0.1 cm. Hip circumference (HC) was measured at the widest part of the buttocks. Participants stand with feet together, and the tape measure was placed horizontally around the hips. Measurements were recorded to the nearest 0.1 cm. Body mass index (BMI) and waist to hip ratio (WHR) were also calculated.

b. ***Dietary assessment and calculation of cardioprotective index***.

A validated food frequency questionnaire (FFQ) was used for dietary assessment. Participants reported the frequency of their dietary intake for the previous year and the reported amounts were then analyzed with a dietary analyzer tool. CPI was calculated based on seven dietary guidelines of world health organization (WHO) for the prevention of cardiovascular diseases [7] including recommended intake of dietary protein ≥ 10% of total dietary energy intake, total fat ≤ 30% total dietary energy intake, SFA ≤ 10% total dietary energy intake, PUFA < 10% total dietary energy intake, cholesterol < 300 mg/d, fiber ≥ 20 g/d, and a fruit and vegetable intake ≥ 400 g/d. To create the index, individuals were awarded 1 point for adhering to any of the specified dietary recommendations and 0 points if they did not comply. The index ranged from 0 to 7 points, with higher scores indicating better dietary quality in terms of its cardioprotective benefits [6]. An illustration of details for CPI calculation is provided in Supp. Figure 1.

c. ***Laboratory measurements***.

Blood samples were collected following an overnight fasting period. Standard laboratory assays using an autoanalyzer (Alpha Classic E analyzer) were employed to measure serum lipids, including triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C). Commercial kits (AccuBind, Insulin, USA, Monobind Inc.) were utilized to measure serum insulin levels. Blood pressure readings were obtained using a standard mercury sphygmomanometer. Circulating Nesfatin-1 and Omentin-1 were analyzed using a human ELISA kit (Bioassay Technology Laboratory, Shanghai Korean Biotech, China), according to the manufacturer's protocol, with the intra-and inter-assay coefficient of variations (CVs) of 8 and 10%, respectively.

d. ***Data analysis***.

Using SPSS (version 21) for data analysis, 348 individuals were included in the analysis. Continuous and discrete variables were reported as mean and standard deviation or frequencies and percentage.

Normality of data distribution was checked using Kolmogorov–Smirnov test and because of normal distribution, the parametric statistical tests were used; one-way Analysis of Variance (ANOVA) was used to analyze the differences in variables (demographic and anthropometric variables) between tertiles of CPI. The chi-squared test was used to compare discrete variables. In order to examine the association between CPI and biochemical risk factors, general linear model (GLM) with adjustment for confounders was used. The Fig. 2 was represented using online Capilot image designer. The CPI categorization into tertiles was performed according to power of 80% and β = 0.2. The robustness of our findings was tested by performing sensitivity analyses including re-analyzing the data using different cut-off points for CPI and adjusting for potential confounders such as age, gender, BMI, physical activity and energy intake. We also performed a complete case analysis to compare with results from our primary model, which used multiple imputation for missing data. Variance inflation factor (VIF), was used to assess multicollinearity. All predictors had VIF values below 5, indicating that multicollinearity was not a concern in our model.

Results

The study participants' general demographic and anthropometric characteristics across tertiles of CPI are summarized in Table 1. As shown, participants' mean age differed significantly across CPI tertiles ($p < 0.001$). Specifically, individuals in the lowest CPI tertile (T3) had

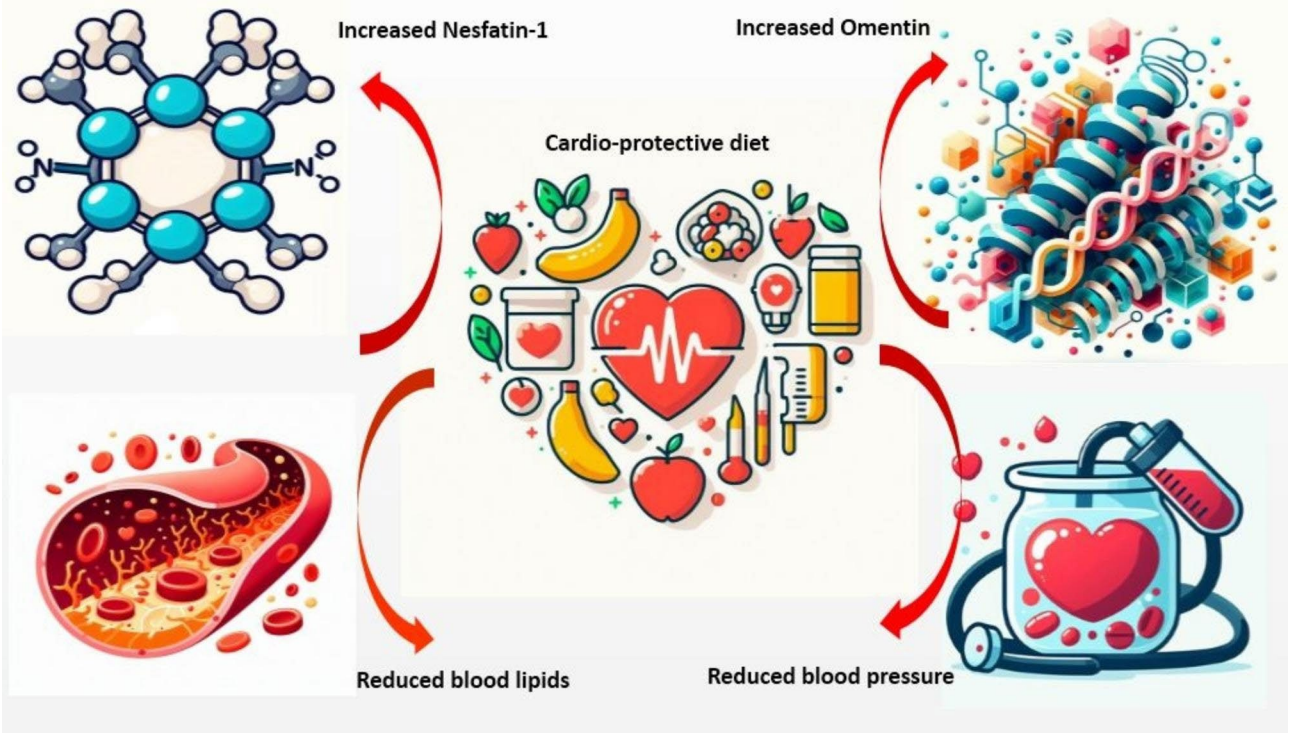

Fig. 2 An illustrative summary of study findings

Table 1 General demographic and anthropometric variables of study participants across CPI tertiles

Variables (continuous)	All participants		Tertiles of CPI						*P** Value
			T_1		T_2		T_3		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Age (y)	40.78	9.23	45.70	9.54	38.30	8.94	42.03	9.08	**< 0.001**
BMI (kg/m^2)	32.62	4.80	32.78	5.99	33.34	4.49	32.18	4.90	0.100
WC (cm)	106.78	9.62	112.90	9.44	107.18	9.85	106.25	9.41	0.086
WHR	0.93	0.07	0.96	0.08	0.93	0.08	0.93	0.07	0.535
Variables (discrete)	Number	Percent	Number	Percent	Number	Percent	Number	Percent	Number
Gender (n[%] Male)	145	41.8	1	10.0	54	42.9	90	42.7	0.118
Marital status (% Single)	46	13.3	1	10.0	24	19.0	21	10.0	**< 0.001**
Education (n [%] ≤ 12 years)	173	92.4	1	100	78	91.9	94	93	0.835
Family size (n [%] > 4)	25	13.2	0	0	14	16.3	11	10.9	0.359
Physically active (n [%] moderate & high)	98	52.2	0	0	48	55.8	50	49.5	0.584

CPI, cardioprotective index; SD, standard deviation; BMI, body mass index; WC, Waist circumference; WHR, waist-to-hip ratio; continuous and discrete data are presented as mean (±SD) and number and percent respectively. P* values derived from one-way ANOVA for continuous variables (age, BMI, WC, WHR) and chi-squared test for discrete variables (gender, marital status, education, family size and physical activity)

significantly higher age compared with lowest tertiles ($P<0.001$). No significant difference was observed in terms of other demographic or anthropometric variables. Table 2 shows the frequency of participants with adherence to CPI ingredients (e.g. achieved+1 score for each item). The results show that there was a statistically significant difference between components of cardioprotective index according to its tertiles except for fiber score. As expected, a rising trend was observed between CPI tertiles for the frequency of individuals who adhered to the recommendations toward protein, fat, SFA, PUFA, cholesterol, and fruits and vegetable consumption of CPI ingredients ($P<0.05$). The comparison of biochemical variables across CPI tertiles are represented in Table 3. As shown in this Table, those at the first tertile of CPI had significantly higher SBP, DBP, LDL and TG and higher HDL compared with second and third tertile in crude model. In the Tukey's post hoc analysis, these differences were observed between first tertile with other tertiles. After adjustment for the confounders of age, sex, BMI, physical activity and total calorie intake, LDL lost its significance across CPI tertiles. Moreover, serum TC,

Table 2 The frequency of study participants with (+1) score for each components of cardio-protective index by CPI tertiles

Variable	Tertiles of CPI						*P* Value
	T_1		T_2		T_3		
	Number	Percent	Number	Percent	Number	Percent	
Protein (+1) score	1	10	116	92.1	208	98.1	**0.019**
Fat (+1) score	0	0	9	7.1	185	87.3	**<0.001**
SFA (+1) score	0	0	53	42.1	203	95.8	**<0.001**
PUFA (+1) score	0	0	95	75.4	212	100	**<0.001**
Cholesterol (+1) score	0	0	52	41.3	162	76.4	**<0.001**
Fiber (+1) score	2	20	122	96.8	211	99.5	0.359
Fruit and vegetable (+1) score	3	30	107	84.9	188	88.7	**<0.001**

CPI, cardio-protective index; SFA, saturated fatty acid, PUFA, polyunsaturated fatty acid, P* values derived from chi-squared test

Table 3 The comparison of biochemical variables of study participants across CPI tertiles

Variable	All participants		Tertiles of CPI						*P* Value	*P** values
			T_1		T_2		T_3			
	Mean	SD	Mean		Mean	SD	Mean	SD		
FBS (mg/dl)	92.66	19.18	88.50	4.83	93.23	25.74	91.32	14.27	0.154	0.098
Insulin (mIU/l)	16.17	13.66	15.60	6.32	15.09	8.00	16.94	16.66	0.580	0.075
HOMA-IR	3.76	3.26	3.35	1.35	3.66	2.44	3.85	3.78	0.874	0.075
SBP (mmHg)	122.99	16.35	134.00	19.55	119.41	16.99	124.61	15.38	**0.002**	**<0.001**
DBP (mmHg)	81.78	11.69	86.00	8.75	79.39	13.13	83.00	10.65	**0.012**	**0.002**
LDL (mg/dl)	123.68	31.83	127.04	25.26	117.20	29.70	117.41	32.82	**0.016**	0.176
HDL (mg/dl)	43.32	9.52	39.70	11.92	44.88	9.44	42.57	9.35	**0.046**	**0.001**
TG (mg/dl)	152.55	94.13	224.40	91.20	136.57	99.52	158.71	88.91	**0.005**	**0.010**
TC (mg/dl)	191.45	36.64	184.90	29.95	186.91	32.47	194.49	39.02	0.158	0.097
Nesfatin-1 (ng/ml)	3.5	1.12	2.5	0.23	3.29	0.88	3.71	1.26	**0.029**	**0.040**
Omentin-1 (ng/ml)	6.20	1.41	3.33	1.00	6.13	1.296	6.54	1.21	**<0.001**	**<0.001**

CPI, cardioprotective index; SD, standard deviation; FBS, fasting blood sugar; HOMA-IR, homeostatic model assessment for insulin resistance; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL, low density lipoprotein cholesterol; HDL, high density lipoprotein cholesterol; TG, triglyceride; TC, total cholesterol; P values derived from unadjusted ANOVA. P* values derived from general linear model (GLM) after adjustment for confounders (age, gender, BMI, PA and energy intake)

insulin and HOMA-IR were not significant across CPI tertiles neither in crude not in adjusted models ($P>0.05$). Also, significantly higher Nesfatin-1 and Omentin-1 levels were observed in the highest versus lowest CPI tertiles ($P<0.05$). All of these differences were significant either for crude or confounder-adjusted models.

Discussion

In the current study, we revealed a decreased level of blood pressure, LDL, TG and increased concentrations of HDL across tertiles of cardio-protective index. Also, serum Nesfatin-1 and Omentin-1 were increased toward CPI tertiels among patients with metabolic syndrome (Fig. 2).

CPI ingredients reveal its beneficial effects toward cardio-metabolic health; numerous studies have reported the metabolic benefits of increased fruits and vegetables intake in metabolic syndrome; in the recent meta-analysis of more than 25 observational studies, increased fruits and vegetable intakes was associated with lower risk of metabolic syndrome [31, 32]. Similarly, reduced saturated fatty acids [33, 34]. Some studies reported that a diet high in fruits and vegetables and low in saturated fatty acids increases the Nesfatin-1 concentrations [21, 22, 35]. Also, in other study, serum Nesfatin-1 levels was only correlated with dietary fiber intake among patients with metabolic syndrome ($r=-0.355$, $p<0.050$) [36]. Another study revealed that obese individuals are faced with decreased cerebrospinal fluid/plasma ratio of the novel satiety molecule, Nesfatin-1/NUCB-2, in and that there is a Nesfatin-1/NUCB-2 resistance among them [37].

Similar reports are available in case of Omentin-1's cardio-metabolic health; high intake of fruits and vegetable intake was positively associated with circulating Omentin-1 concentrations [38, 39] and dietary interventions of high fruits and vegetable intake led to increased its circulating level [40, 41]. It is known that a long-term administration of a diet with a lower fat content and a lower SFA intake, increases Omentin-1 plasma concentration, possibly via improved insulin resistance and reduced inflammation [42]. Omentin-1 is an adipokine predominantly is produced by visceral adipose tissue with reduced expression in obesity, insulin resistance and diabetes. Lower plasma Omentin-1 levels contribute to cardiovascular

diseases [13]. Omentin-1 has anti-inflammatory effects via reducing C - reactive protein (CRP) levels and it exerts antiatherogenic and anti-cardiovascular disease properties [13, 24]. It is known as a novel biomarker of metabolic risk factors and as a promising therapeutic target against obesity and related disorders. Recent study found that both subcutaneous adipose tissue derived and circulating levels of omentin-1 were significantly lower in subjects with metabolic syndrome compared to controls after adjustment for age, BMI, or WC [43].

In the current study, higher CPI was accompanied with lower TG and higher HDL while difference in TC and LDL were non-significant in confounder-adjusted model. Previous studies also recommended high intake of fruit and vegetables for cardiovascular health. However, there have been persistent beliefs that fruits having high concentrations of fructose elevate the level of TG in blood unlike vegetables; in the previous meta-analysis, high intake of fruits but not vegetables inversely associated with hypertriglyceridemia [44]. In another meta-analysis, fruits and vegetable consumption had small effects on increasing serum HDL among overweight and obese individuals [45]. Lowering SFA and increasing PUFA intake as recommended by CPI ingredients, are also associated with increase in HDL and reduction in TG concentrations [46, 47].

considering a recommended intake of dietary protein ≥ 10% of total dietary energy intake, total fat ≤ 30% total dietary energy intake, saturated fatty acids (SFAs) ≤ 10% total dietary energy intake, polyunsaturated fatty acids (PUFAs) < 10% total dietary energy intake, cholesterol < 300 mg/d, fiber ≥ 20 g/d, and a fruit and vegetable intake ≥ 400 g per day.

The current study has some limitations; the cross-sectional design of the study limits the causality inference; also, dietary data were collected using a self-reported questionnaires that stem for recall bias. Although the FFQ was a validated questionnaire with acceptable sensibility.

In conclusion, in the current study, high CPI scores were associated with favorable metabolic profile and higher Nesfatin-1 and Omentin-1 in subjects with metabolic syndrome; these results underscores the importance of assessing the cardio-protective potential of a usual diet as a potential predictor of cardiovascular disease and implementing preventive measures to optimize cardiovascular health in this population. Longitudinal studies are needed to elucidate the causal relationships between CPI and cardio-metabolic risk. Also, it is suggested to measure inflammatory parameters like C-reactive protein (CRP) to better identify the underlying mechanisms and also to adjust for their possible confounding effects. Additionally, research exploring the effectiveness of targeted lifestyle interventions and pharmacological therapies in reducing the risk of cardiovascular disease in patients with metabolic syndrome is warranted.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12902-024-01791-2.

Supplementary Material 1

Acknowledgements

We are thankful from all of the study's participants.

Author contributions

HBK, HE and AK were involved in data collection and subjects' recruitment. OQA, MSM and HBK were involved in hypothesis generation and statistics. AK was involved in data collection, data analysis and supervision of the project. RKAA and MJW were involved in statistical approaches and drafting the paper. AG was also involved in data collection and revision of the paper. OQA was also involved in funding acquisition. All of the authors contributed in writing the draft of manuscript and agreed to its submission.

Funding

Research division of Alnoor University for their financial support.

Data availability

The datasets of the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Written consent was obtained from all of the participants of the study. The protocol of the study is approved by ethics committee of Alnoor University (143-P912).

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 10 September 2024 / Accepted: 21 November 2024
Published online: 27 November 2024

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