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Gender-specific association between a novel atherogenic index of plasma, metabolic parameters and inflammation among obese adults

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Abstract

Background Previous studies have demonstrated the association between lipoprotein combined index (LCI), as a novel atherogenic index with cardiovascular disease, fatty liver, diabetes and numerous other health problems; however, its association with metabolic syndrome risk and its components has not been investigated before. The current study was aimed to investigate the association between LCI metabolic and inflammatory risk factors among obese men and women.

Methods In the current cross-sectional study, the association between LCI, anthropometric parameters and metabolic risk factors including serum lipids, glycemic markers, insulin resistance and C - reactive protein (CRP) concentrations were measured. LCI was calculated as (total cholesterol [TC] × triglyceride [TG] × low density lipoprotein cholesterol [LDL]) / (high density lipoprotein cholesterol [HDL]).

Results Highest quartiles of LCI was accompanied with higher waist to hip ratio ($P=0.017$). Also, higher systolic and diastolic blood pressure, higher serum lipids and lower high density lipoprotein concentrations were observed in higher quartiles of LCI HDL ($P<0.05$). Among men and women, higher LCI was also associated with higher CRP and lower HDL in men ($P<0.05$); while among women, higher CRP, TG, TC and lower HDL was observed in highest versus lowest quartiles of LCI ($P<0.05$). Among anthropometric and biochemical variables, TG has the highest power for identification of metabolic syndrome with area under curve (AUC) of 0.82 and Youden index of 0.58 while LCI was in the second place after TG in prediction of metabolic syndrome (e.g. AUC of 0.80 and Youden index of 0.47).

Conclusion LCI was in direct association with lipid parameters and inflammation among obese men and women. Although predictive power of LCI for metabolic syndrome was acceptable, but it came in the second place after TG for men and women. Further studies are warranted to make a better conclusion.

Clinical trial number Not applicable.

Keywords Lipoprotein combined index, Metabolic syndrome, Obesity, Cardiovascular risk factors

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Introduction

The increasing prevalence of metabolic syndrome —a constellation of metabolic abnormalities that includes central obesity, insulin resistance, dyslipidemia, and hypertension—has become a significant public health concern worldwide [1]. Metabolic syndrome is not merely a cluster of risk factors but a precursor to more severe health conditions, particularly cardiovascular diseases (CVDs) and type 2 diabetes mellitus, both of which contribute substantially to global morbidity and mortality [2]. As such, the early identification of individuals at risk for metabolic syndrome is critical to preventing these associated complications.

In recent years, the Lipoprotein Combined Index (LCI) has gained attention as a promising biomarker in the prediction health problems related to metabolic syndrome including cardiovascular disease, coronary artery disease and myocardial infarction [3–6]. Traditional lipid measurements, such as low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides, have long been used to assess cardiovascular risk; numerous studies have revealed that reduction in LDL-C significantly reduces the incidence rate of CVD-related events; however, even by achieving the recommended level of LDL-C, the residual CVD risk remains at 50%, confirming the need for new CVD predictors [7]. These parameters individually may not fully capture the complex lipid alterations associated with metabolic syndrome. Compared with individual lipid biomarkers, the comprehensive lipid indexes, such as LCI are considered to be better predictors of cardiovascular disease [8]. The LCI, by integrating multiple lipoprotein parameters into a single composite score, offers a more holistic view of lipid metabolism and its disturbances [5].

Research has increasingly demonstrated the potential of LCI to predict CVD-related events more effectively than conventional lipid measures. For instance, in one study, among all of the traditional and non-traditional CVD predictors, LCI was demonstrated with highest specificity for prediction of coronary artery bypass graft surgery (CABG) with a value of 65.8% (AUC = 0.634, $p < 0.001$) [3]. In one other study, LCI was significantly higher among postmenopausal women with CAD than in control group and in those with Gensini Score of greater than 38.75 revealing the potential of LCI in prediction of coronary artery events [9]. Also, several studies have shown that higher LCI values are significantly associated with an increased risk of developing components of metabolic syndrome including low HDL-cholesterol even after adjusting for traditional cardiovascular risk factors [10]. These findings suggest that LCI could serve as an early warning signal, allowing for timely interventions aimed at reducing the burden of metabolic syndrome and its complications. Therefore, LCI's relevance extends beyond its predictive capability for metabolic syndrome. Although, numerous evidence indicates a strong correlation between LCI and cardiovascular events and risk factors, however, its predictive ability in metabolic syndrome is not studied yet. LCI, combines the effects of total cholesterol, TG, LDL and HDL [2]. It is well-known that TG and HDL have opposite effects on oxidative stress, inflammation, formation of extracellular matrix, and changes in vascular smooth muscle from the contractile to the synthetic form, and the LCI, summarizes all of these influences altogether [11]. This is particularly noteworthy given the role of systemic inflammation in the pathogenesis of both metabolic syndrome and cardiovascular diseases.

This article for the first time, aims to delve deeper into the association between LCI and anthropometric and biochemical biomarkers including serum lipids, glycemic markers, blood pressure and CRP concentrations among obese individuals and to determine its predictive power for metabolic syndrome among obese individuals. Our primary hypothesis was that there is a relationship between LCI with anthropometric and biochemical variables and our second hypothesis was that LCI has an acceptable power in prediction of metabolic syndrome.

Methods and materials

Sampling method

The study utilized a stratified random sampling method to select subjects from the general adult population. The target population included adults aged 18 to 60 years old, residing in Anshan, with no restrictions on gender, ethnicity, or socioeconomic status. To ensure representation across key demographic factors, the population was stratified based on age groups (e.g., 18–29, 30–44, 45–60 years old), gender, and socio-economic status. Within each stratum, participants were randomly selected to achieve proportional representation to minimize selection bias. Inclusion criteria were adults aged 18 to 60 years old, and those who provided informed consent and those with body mass index (BMI) greater than 30 kg/m^2. Exclusion criteria were individuals with severe cognitive impairments that preclude informed consent, those with any previous history of CVD, cancers were also excluded. A total of 647 subjects were included in the study, aligning with the predetermined sample size. Subjects were enrolled though flyers, posters and online advertising. Also, community outreaches were used to obese individuals' recruitment. The sample size was determined using a power analysis to ensure that the study was adequately powered to detect a statistically significant effect, assuming a 5% level of significance ($\alpha = 0.05$) and a power of 80% ($\beta = 0.20$). The expected effect size based on previous research [8]. The formula used for sample size calculation

was: $\frac{z^2 \times P \times (1-P)}{d^2}$, where: n = required sample size, Z = Z value (e.g., 1.96 for a 95% confidence level), p = estimated proportion of the population with the characteristic of interest (if unknown, 0.5 was used to maximize the sample size), d = margin of error (precision). Given these parameters and the assumptions, the calculated sample size was 550. To account for potential non-responses or dropouts, an additional 15% was added, leading to the final sample size of 647 subjects.

Laboratory analysis

Blood samples were collected from all subjects after an overnight fast of at least 12 h. The fasting blood sugar (FBS) and lipid profile, including total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and triglycerides, were measured using enzymatic colorimetric method with an with Abbott Diagnostics C8000i autoanalyzer (Abbott Laboratories, Abbott Park, IL, USA). Serum insulin was measured by commercial kits (AccuBind, Insulin, USA, Monobind Inc.). Serum high sensitivity C-reactive protein (hs-CRP) was measured by enzymatic immunoassay turbidimetric assay (Roche Cobas 6000, Penzberg, Germany). Homeostatic model assessment for insulin resistance (HOMA-IR) was calculated as follows: $\frac{\text{fasting insulin}\left(\mu\ \frac{\text{IU}}{\text{L}}\right) \times\ \text{fasting glucose}\left(\frac{\text{nmol}}{\text{L}}\right)}{22.5}$ [12] and LCI was calculated as $\frac{\text{TC} \times \text{TG} \times \text{LDL}}{\text{HDL}}$ [3]. Metabolic syndrome was defined according to the National Cholesterol Education Program Adult Treatment Panel III criteria (NCEP-ATP III) as follows: [1] WC of 90 cm for men and 80 cm for women; [2] TG level of 150 mg/dl 3) HDL-C level of 40 mg/dl in men and 50 mg/dl in women; [4] High blood pressure of 130/85 mmHg; [5] Impaired fasting blood glucose level of 144 mg/dl [13].

Blood pressure measurement

Blood pressure was measured using a mercury sphygmomanometer (Jiangsu, China) in a standardized manner. Subjects, after avoiding caffeine, smoking, and physical activity for 30 min, were seated with their arm at heart level. After a 5-minute rest, three readings were taken at 1-minute intervals, with the average of the last two measurement were recorded [14, 15].

Anthropometric and dietary measurements

Participants were instructed to wear minimal clothing (such as lightweight attire and undergarments) to ensure precise measurements and to remove shoes, socks, jewelry, and any items that might affect accuracy. Height and weight were measured with HM1000-SZ (HeMei Tech Corp., China) with the precision of 0.1 cm and 0.1 kg respectively while participants standing barefoot, heels together, back against the wall, and head aligned in the Frankfort horizontal plane. Waist circumference (WC) was measured at the midpoint between the lower rib cage and the iliac crest, while hip circumference (HC) was recorded at the widest point around the buttocks. The waist-to-hip ratio (WHR) was calculated by dividing WC by HC. Body composition was assessed using a BIA device (Inbody 770 Co., Seoul, Korea) following the manufacturer's instructions, with subjects standing barefoot on the scale, ensuring even contact on the electrode plates and removal of shoes, socks, and heavy clothing or accessories. A validated food frequency questionnaire (FFQ) [16] was employed for dietary assessments. Participants were asked about the amount and frequency of consumption of each food item. The reported quantities and frequencies were then converted into grams and days, respectively. Physical activity status was assessed using a validated physical activity questionnaire [17].

Statistical analysis

Data analysis was performed using SPSS (version 23). Normality of the data was assessed using the Kolmogorov-Smirnov test, and because the data were normally distributed, the following analyses were conducted: continuous and discrete variables were reported as means and standard deviations or as frequencies and percentages. LCI was categorized into quartiles based on the power of 80% and $\beta = 0.2$. One-way Analysis of Variance (ANOVA) was used to assess differences in demographic and dietary characteristics across the quartiles of LCI. The chi-squared test was employed to compare discrete variables. To examine associations between LCI and biochemical risk factors, multiple logistic regression analyses were conducted in three models: (I) unadjusted, (II) adjusted for age and sex, and (III) further adjusted for BMI, and physical activity and dietary energy intake; (IV) further adjusted for WHR and red meat intake. The Receiver Operating Characteristic (ROC) curve analysis was used to assess the diagnostic performance of LCI and other metabolic parameters. The ROC curve plots sensitivity against 1-specificity across various thresholds, with the Area Under the Curve (AUC) summarizing the test's overall ability to distinguish between positive and negative cases. The optimal cutoff was determined by maximizing the Youden Index. Confidence intervals for the AUC were calculated, and significance testing was conducted to confirm the test's discriminatory power. Comparisons of ROC curves were made using statistical tests if multiple tests were evaluated. According to generally accepted classifications of ROC AUC, ROC AUC of 0.7 to 0.8 is considered acceptable, 0.8 to 0.9 is considered excellent, and more than 0.9 is considered outstanding [18].

Variance inflation factor (VIF), was for multicollinearity analysis. All independent variables had VIF values

Table 1 Demographic and anthropometric variables across different LCI quartiles

Variable	All participants (*n* = 647)		Quartils of LCI								*P** Value
			Q1 (*n* = 161)		Q2 (*n* = 162)		Q3 (*n* = 162)		Q4 (*n* = 162)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Age (y)	40.08	9.55	37.86	9.15	40.33	9.88	40.95	9.26	41.17	9.62	**0.006**
Gender (% Male)†	312	48.2	60	37.3	82	50.6	76	46.9	94	58	**0.001**
Education (12 years ≤) †	495	76.50	121	75.15	130	80.24	127	78.39	112	69.13	0.352
Occupation (% employed) †	433	66.92	102	63.35	111	68.51	111	68.51	109	67.28	0.311
Marital status (% Single) †	360	55.64	81	50.31	92	56.79	107	66.04	82	50.61	0.695
BMI (kg/m^2)	33.43	5.23	33.76	4.69	33.65	4.79	33.61	4.60	32.72	6.57	0.250
WC (cm)	106.55	9.79	105.35	9.36	107.05	10.39	106.95	9.51	106.85	9.85	0.350
WHR	0.92	0.08	0.90	0.07	0.92	0.091	0.93	0.10	0.93	0.07	**0.017**
HC	115.88	11.45	116.75	10.22	116.09	11.22	115.31	11.75	115.30	12.64	0.647
FM (%)	35.28	9.78	34.75	8.24	35.12	10.28	35.59	9.92	35.90	11.03	0.811
FFM (%)	58.53	13.24	56.74	13.33	59.21	12.19	60.16	13.53	58.33	14.01	0.191
Physically active (% moderate & high)	241	37.24	49	30.43	78	48.14	66	40.74	55	33.95	0.977

LCI, Lipoprotein combine index; BMI, Body mass index; WC, Waist circumference; WHR, Waist to hip ratio; HC, Hip circumference; FM, Fat mass; FFM, Fat free mass. *P-values for continuous variables are obtained from one-way-analysis of variance (ANOVA) and *Tuky's* post hoc test revealing the significant difference between 3rd and 4th quartiles with 1st and 2nd. P-values for discrete variables are derived from chi-squared test. †, Data are presented as number and percent

Table 2 Dietary intake of macronutrients and food groups across different LCI quartiles

Variable	All participants (*n* = 647)		Quartils of LCI								*P** Value
			Q1 (*n* = 161)		Q2 (*n* = 162)		Q3 (*n* = 162)		Q4 (*n* = 162)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Energy (kcal/d)	2915.87	1050.09	2949.04	996.05	2949.32	1124.69	2792.90	1004.66	2973.82	1097.88	0.391
Carbohydrate (%)	77.13	276.29	84.19	360.02	56.39	9.61	86.81	358.03	81.43	218.37	0.741
Protein (%)	16.12	55.22	19.17	90.06	12.89	2.80	12.93	2.29	19.62	64.39	0.523
Fat (%)	38.95	150.09	50.42	258.20	30.47	7.78	30.21	7.65	44.97	153.67	0.523
Grains (g/d)	540.88	330.07	576.06	396.93	487.29	237.17	543.20	284.11	557.96	374.56	0.089
Legumes (g/d)	61.28	63.23	59.08	46.00	74.17	82.06	55.57	61.75	56.07	55.05	0.084
Red meat (g/d)	20.50	21.86	21.74	18.94	23.46	27.96	27.77	21.79	27.94	16.67	**0.025**
Fish (g/d)	9.0891	13.25353	8.8016	13.79912	8.8803	11.34357	7.1500	10.41020	115,858	197.47476	0.471
Low fat daily (g/d)	226.80	199.17	19.11	189.51	232.72	217.98	126.85	143.29	259.57	197.47	0.470
High fat daily(g/d)	104.44	139.50	98.08	123.84	108.53	170.59	84.81	110.61	143.29	11.43	0.520
Fruits (g/d)	571.04	521.82	495.35	433.28	587.80	526.19	598.52	479.61	685.48	621.95	0.089
Vegetables (g/d)	360.31	257.62	337.70	215.35	368.44	291.40	346.04	223.63	389.62	289.81	0.271
Nuts (g/d)	18.63	74.22	13.96	15.54	18.25	48.27	13.69	19.15	28.82	139.22	0.230

LCI, Lipoprotein combine index; *P-values for continuous variables are obtained from ANCOVA analysis after dietary energy adjustment and *Tuky's* post hoc test revealing the significant difference between 1st quartile and other quartiles

below 5, therefore, multicollinearity was not a concern in our model [19].

Results

General characteristics of study participants are shown in Table 1. Totally, 647 adult population were enrolled in the current study. Those at the higher quartiles of LCI, were older and were likely to be male participants ($p < 0.01$). There was no significant difference in other demographic variables. Among anthropometric parameters, WHR was significantly higher in 3rd and 4th quartiles of LCI than in 1st and 2nd quartiles ($P = 0.017$). In comparison of dietary energy, macronutrients and food groups between LCI quartiles (Table 2), those at the highest quartiles of LCI had significantly higher red meat consumption ($P = 0.025$). The comparison of biochemical variables across different LCI quartiles (Table 3) shows that there was higher values of serum LDL, TG, TC, FBS, CRP and SBP and lower HDL concentrations in the highest versus lowest quartiles of LCI ($P < 0.05$). Multinomial logistic regression for the association between LCI and anthropometric variables and biochemical parameters among study participants are presented in Tables 4 and 5. As shown in these tables, in forth model of adjustment, WHR was significantly higher in forth quartile and second quartile versus reference. While for other anthropometric variables in the fully adjusted model, the significance was lost. Also, In multinomial logistic regression analysis of the association between LCI and biochemical risk factors (Table 5), those at the second,

Table 3 The comparison of biochemical variables across different LCI quartiles

Variable	All participants (*n* = 647)		Quartiles of LCI								*P** Value
			Q1 (*n* = 161)		Q2 (*n* = 162)		Q3 (*n* = 162)		Q4 (*n* = 162)		
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
HOMA-IR	3.84	3.32	3.47	2.73	3.88	3.25	4.06	3.99	3.99	3.29	0.448
SBP (mmHg)	120.78	20.32	115.36	14.61	120.80	20.32	122.09	13.39	124.50	14.79	**< 0.001**
DBP (mmHg)	80.10	11.85	76.92	11.13	80.16	13.18	81.35	10.53	81.82	11.85	0.05
CRP (mg/dl)	4.51	1.39	3.61	1.19	4.25	0.95	4.80	1.24	5.38	1.47	**< 0.001**
LDL (mg/dl)	122.84	37.72	96.71	31.90	113.32	26.06	131.79	33.78	149.36	36.28	**< 0.001**
HDL (mg/dl)	43.61	11.24	50.37	12.00	43.78	9.20	42.56	9.48	37.76	10.37	**< 0.001**
TG (mg/dl)	139.46	85.67	71.45	34.91	112.06	36.17	140.13	44.20	233.79	103.17	**< 0.001**
TC (mg/dl)	191.30	41.86	159.01	32.95	178.59	26.88	199.63	34.72	227.77	37.55	**< 0.001**
FBS (mg/dl)	99.69	21.35	91.11	16.67	95.11	21.34	98.45	32.58	99.69	21.35	**< 0.001**
Insulin (mIU/l)	16.45	18.60	16.34	17.55	15.93	12.06	18.42	29.49	15.17	9.75	0.546

LCI, Lipoprotein combine index; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; CRP, C-reactive Protein; LDL-C, Low Density Lipoprotein Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; TG, Triglyceride; TC, Total Cholesterol; FBS, Fasting blood sugar; *P-values for continuous variables are obtained from analysis of variance (ANOVA) and *Tuky's* post hoc test revealing the significant difference between 4st quartile and other quartiles

Table 4 The association between LCI and anthropometric parameters

Anthropometric variable		Q1 (*n* = 161)	Quartils of LCI					
			Q2 (*n* = 162)		Q3 (*n* = 162)		Q4 (*n* = 162)	
			OR (CI)	*P*-value	OR	*P*-value	OR	*P*-value
Weight (kg)	Model-I	**1**	0.964 (0.932–0.966)	**0.029**	0.971(0.936–1.007)	0.109	0.964 (0.931–0.998)	**0.039**
	Model-II	**REF**	0.964(0.931–0.998)	**0.039**	0.972(0.935–1.011)	0.159	0.964(0.930-1.000)	0.052
	Model-III		0.957(0.899–1.019)	0.170	0.953(0.888–1.022)	0.176	0.971(0.916–1.029)	0.319
Height (cm)	Model-I	**1**	0.985(0.0.922-996)	**0.030**	0.977(0.932–1.024)	0.334	0.95(0.914–0.998)	**0.010**
	Model-II	**REF**	0.954(0.912–0.998)	**0.040**	0.986(0.929–1.046)	0.645	0.947(0.904–0.991)	**0.019**
	Model-III		0.943(0.867–1.026)	0.171	1.031(0.937–1.135)	0.527	0.991(0.913–1.077)	0.838
BMI (kg/m^2)	Model-I	**1**	0.874(0.771–0.99)	**0.035**	0.939(0.812–1.085)	0.394	0.87(0764-0.991)	**0.036**
	Model-II	**REF**	0.858 (0.753–0.977)	**0.021**	0.938(0.803–1.097)	0.423	0.853(0.746–0.976)	**0.021**
	Model-III		0.878(0.707–1.09)	0.238	1.099(0.836–1.443)	0.499	0.947(0.806–1.113)	0.508
WC (cm)	Model-I	**1**	1.057(0.852–1.311)	0.614	0.873(0.71–1.073)	0.198	1.044(0.848–1.286)	0.686
	Model-II	**REF**	1.033(0.831–1.284	0.767	0.863(0.701–1.063)	0.166	1.022(0.828–1.261)	0.840
	Model-III		0.821(0.270–2.501)	0.729	1.806(0.621–5.252)	0.278	0.95(0.628–1.436)	0.806
HC (cm)	Model-I	**1**	0.981(0.808–1.192)	0.848	1.154(0.9581.39)	0.133	1.001(0.828–1.209)	0.993
	Model-II	**REF**	0.989(0.813–1.202)	0.908	1.156(0.958–1.394)	0.130	1.007(0.833–1.219)	0.94
	Model-III		1.231(0.435–3.489)	0.695	0.581(0.213–1.586)	0.29	1.032(0.701–1.518)	0.875
WHR	Model-I	**1**	0.921(2.468–3.438)	0.995	0.924 (2.469–3.468)	0.094	0.935 (2.469–3.329)	0.697
	Model-II	**REF**	0.920(2.476–3.429)	0.995	0.927 (2.498–3.324)	0.094	0.978 (2.234–3.143)	0.697
	Model-III		1.064 (1.018–1.112)	**0.006**	1.058 (1.008–1.110)	**0.022**	1.057(1.008–1.108)	**0.021**
FM (%)	Model-I	**1**	1.077(1.010–1.149)	**0.024**	1.053(0.982–1.129)	0.147	1.083(1.011–1.160)	**0.023**
	Model-II	**REF**	1.096(1.022–1.175)	**0.010**	1.053(0.976–1.137)	0.184	1.101(1.023–1.184)	**0.010**
	Model-III		1.091(0.983–1.212)	0.102	0.97(0.848–1.109)	0.655	1.039(0.947–1.140)	0.422
FFM (%)	Model-I	**1**	1.062(1.017–1.109)	**0.006**	1.055(1.007–1.105)	0.240	1.054(1.006–1.103)	**0.026**
	Model-II	**REF**	1.064(1.018–1.112)	**0.006**	1.058(1.008–1.110)	0.220	1.057(1.008–1.108)	**0.021**
	Model-III		1.053(0.996–1.113)	0.071	1.004(0.939–1.074)	0.901	1.038(0.981–1.098)	0.199

LCI, Lipoprotein combine index; BMI, Body mass index; WC, Waist circumference; HC, Hip circumference; WHR, Waist to hip ratio; FM, Fat mass; FFM, Fat free mass. Model I: Crude, Model II: Adjusted for age and sex, Model III: Adjusted for age, sex, physical activity and dietary energy intake. Model –IV, Further adjusted for red meat intake

third and fourth quartile of LCI were more likely to have significantly higher serum CRP, TG and TC and lower serum HDL levels compared with those at the first quartile in all of four models (e.g. model 1, crude; model 2, adjusted for age and sex; and model 3, further adjusted for BMI, physical activity and dietary energy intake and model 4 further adjusted for WHR and red meat intake). In terms of serum LDL levels, those at the second quartile in models 2 and 3 were more likely to have higher LDL concentrations while those at the fourth quartile in

Table 5 The association between LCI and metabolic risk factors

Biochemical variable		Q1 (*n* = 161)	Quartils of LCI					
			Q2 (*n* = 162)		Q3 (*n* = 162)		Q4 (*n* = 162)	
			OR (CI)	*P*-value	OR	*P*-value	OR	*P*-value
HOMA-IR	Model-I	**1**	1.038 (0.951–1.132)	0.403	1.026 (0.931–1.132)	0.604	0.964 (0.856–1.086)	0.547
	Model-II	**REF**	1.039 (0.949–1.138)	0.409	1.028 (0.929–1.137)	0.599	0.957 (0.847–1.082)	0.485
	Model-III		1.035 (0.946 – 0.133)	0.452	1.021 (0.922–1.053)	0.648	0.945 (0.833–1.072)	0.379
	Model-IV		1.024 (0.362–2.89)	0.966	0.522 (0.333–1.018)	0.876	0.499 (0.257–1.09)	0.987
SBP (mmHg)	Model-I	**1**	0.990 (0.941–1.041)	0.700	0.997 (0.931–1069)	0.943	0.986 (0.897–1.083)	0.763
	Model-II	**REF**	0.954 (0.897–1.015)	0.138	0.957 (0.881–1039)	0.291	0.925 (0.826–1.036)	0.179
	Model-III		0.954 (0.896–1015)	0.133	0.953 (0.873–1040)	0.281	0.900 (0.775–1.045)	0.166
	Model-IV		0.937 (0.872–1.007)	0.078	0.936 (0.852–1.028)	0.167	0.908 (0.801–1.031)	0.136
DBP (mmHg)	Model-I	**1**	1.030 (0.952–1.115)	0.462	1.012 (0.911–1.125)	0.817	1.004 (0.881–1.145)	0.950
	Model-II	**REF**	1.055 (0.967–1.151)	0.228	1.037 (0.924–1.163)	0.542	1.008 (0.870–1.167)	0.916
	Model-III		1.060 (0.969–1.160)	0.203	1.032 (0.913–1.169)	0.615	1.049 (0.863–1.275)	0.630
	Model-IV		1.067 (0.970–1.172)	0.181	1.059 (0.933–1.202)	0.377	1.062 (0.903–1.247)	0.468
CRP (mg/dl)	Model-I	**1**	1.170 (1.099–1.320)	**< 0.001**	1.294 (1.202–1.478)	**< 0.001**	1.401 (1.299–1.507)	**< 0.001**
	Model-II	**REF**	1.162 (1.088–1.312)	**< 0.001**	1.321 (1.233–1.440)	**< 0.001**	1.454 (1.317–1.670)	**< 0.001**
	Model-III		1.168 (1.102–1.314)	**< 0.001**	1.365 (1.234–1.457)	**< 0.001**	1.409 (1.360–1.760)	**< 0.001**
	Model-IV		1.125 (1.103–1.423)	**< 0.001**	1.423 (1.011–1.236)	**< 0.001**	1.300 (1.030–1.506)	**< 0.001**
LDL (mg/dl)	Model-I	**1**	1.022 (0.962–1.086)	0.476	1.097 (0.999–1.205)	0.053	1.147 (1.012–1.299)	**0.032**
	Model-II	**REF**	1.019 (0.956–1.86)	0.570	1.122 (1.013–1.244)	**0.028**	1.159 (1.014–1.324)	**0.031**
	Model-III		1.015 (0.952–1.082)	0.684	1.145 (1.014–1.292)	**0.028**	0.988 (0.848–1.150)	0.874
	Model-IV		1.059 (0.868–1.292)	0.573	1.192 (0.955–1.488)	0.119	1.072 (0.845–1.360)	0.569
HDL (mg/dl)	Model-I	**1**	0.711 (0.623–0.826)	**< 0.001**	0.486 (0.399–0.592)	**< 0.001**	0.333 (0.226–0.410)	**< 0.001**
	Model-II	**REF**	0.719 (0.626–0.826)	**< 0.001**	0.478 (0.385–0.593)	**< 0.001**	0.305 (0.226–0.410)	**< 0.001**
	Model-III		0.716 (0.623–0.823)	**< 0.001**	0.468 (0.371–0.589)	**< 0.001**	0.189 (0.110–0.324)	**< 0.001**
	Model-IV		0.726 (0.574–0.918)	**0.007**	0.479 (0.355–0.645)	**< 0.001**	0.235 (0.147–0.376)	**< 0.001**
TG (mg/dl)	Model-I	**1**	1.156 (1.099–1.216)	**< 0.001**	1.292 (1.212–1.378)	**< 0.001**	1.400 (1.302–1.507)	**< 0.001**
	Model-II	**REF**	1.162 (1.099–1.229)	**< 0.001**	1.322 (1.228–1.424)	**< 0.001**	1.433 (1.327–1.569)	**< 0.001**
	Model-III		1.165 (1.101–1.233)	**< 0.001**	1.344 (1.243–1.454)	**< 0.001**	1.501 (1.360–1.656)	**< 0.001**
	Model-IV		1.190 (1.098–1.289)	**< 0.001**	1.367 (1.240–1.507)	**< 0.001**	1.481 (1.333–1.645)	**< 0.001**
TC (mg/dl)	Model-I	**1**	1.026 (1.017–1.035)	**< 0.001**	1.047 (1.036–1.057)	**< 0.001**	1.068 (1.056–1.080)	**< 0.001**
	Model-II	**REF**	1.026 (1.017–1.036)	**< 0.001**	1.047 (1.036–1.057)	**< 0.001**	1.071 (1.058–1.083)	**< 0.001**
	Model-III		1.027 (1.017–1.036)	**< 0.001**	1.048 (1.037–1.059)	**< 0.001**	1.074 (0.833–1.072)	**< 0.001**
	Model-IV		1.124 (0.925–1.366)	0.238	1.216 (0.980–1.508)	0.076	1.690 (1.282–2.226)	**< 0.001**
FBS (mg/dl)	Model-I	**1**	1.006 (0.993–1.020)	0.341	1.013 (0.999–1.027)	0.069	1.018 (1.003–1.034)	**0.017**
	Model-II	**REF**	1.005 (0.992–1.019)	0.447	1.012 (0.998–1.026)	0.103	1.017 (1.001–1.032)	**0.035**
	Model-III		1.006 (0.992–1.020)	0.391	1.012 (0.998–1.026)	0.102	1.018 (1.002–1.034)	**0.024**
	Model-IV		0.961 (0.906–1.020)	0.193	0.989 (0.950–1.031)	0.607	0.995 (0.943–1.049)	0.844
Insulin (mIU/l)	Model-I	**1**	0.984 (0.931–1.040)	0.559	0.981 (0.925–1.040)	0.518	0.968 (0.882–1.062)	0.493
	Model-II	**REF**	0.977 (0.928–1.029)	0.375	0.973 (0.921–1.027)	0.321	0.960 (0.887–1.039)	0.313
	Model-III		0.911 (0.927–1.030)	0.383	0.972 (0.919–1.028)	0.319	0.972 (0.902–1.047)	0.453
	Model-IV		0.986 (0.763–1.288)	0.786	1.107 (1.030–1.190)	**0.006**	1.114 (1.008–1.231)	**0.036**

LCI, Lipoprotein combine index; HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; CRP, C-reactive Protein; LDL-C, Low Density Lipoprotein Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; TG, Triglyceride; TC, Total Cholesterol; FBS, Fxasting blood sugar; The multivariate multinomial logistic regression was used for estimation of ORs and confidence interval (CI). Model I: crude, Model II: adjusted for age and sex, Model III: adjusted for age, BMI, sex, physical activity and dietary energy intake.Model –IV, further adjusted for WHR and red meat intake

models 1 and 2 had significantly higher LDL levels compared with those at the first quartile ($P < 0.01$). Also, those at the fourth quartile of LCI were more likely to had significantly higher serum FBS compared with first quartile in all three models; while this values were non-significant for second and third quartiles.

Multinomial logistic regression for the association between LCI, anthropometric and metabolic parameters separately for men and women are represented in supplementary Tables 1 to 4. In men, there was no association between anthropometric parameters and LCI; while among women, those at the fourth, third and second quartile of LCI were more likely to have higher fat mass

and fat free mass in all of four model ($P<0.05$). Also, men in the fourth quartile of LCI were more likely to have higher CRP levels compared with first quartile and men in the fourth and third quartile had higher TG and lower HDL values (Sup. Table 3), while among women, higher CRP in fourth quartile and higher TG, TC and lower HDL in the fourth, third and second quartile versus first quartile was observed ($P<0.05$).

The ROC curves constructed to compare the predictive values of LCI and anthropometric and metabolic risk factors for identifying metabolic syndrome in entire population are shown in Fig. 1. The AUC for LCI was 0.80 which was significantly higher than AUC of all of anthropometric variables. While among biochemical risk factors, AUC for LCI was higher than FBS, TC and LDL in identifying metabolic syndrome ($P<0.001$) and it was lower than TG (0.82). The Youden index, sensitivities, specificities, true positive and false positive rate and optimal cut-points for the anthropometric and metabolic risk factors alongside with LCI in prediction of metabolic syndrome shown are shown in Tables 6 and 7. The Youden index for LCI was 47.74 with sensitivity, specificity, true positive and false positive rates of and NPV of 62.6 (55.9–69.0), 85.1 (80.3–89.2), 77.7 and 73.4 respectively.

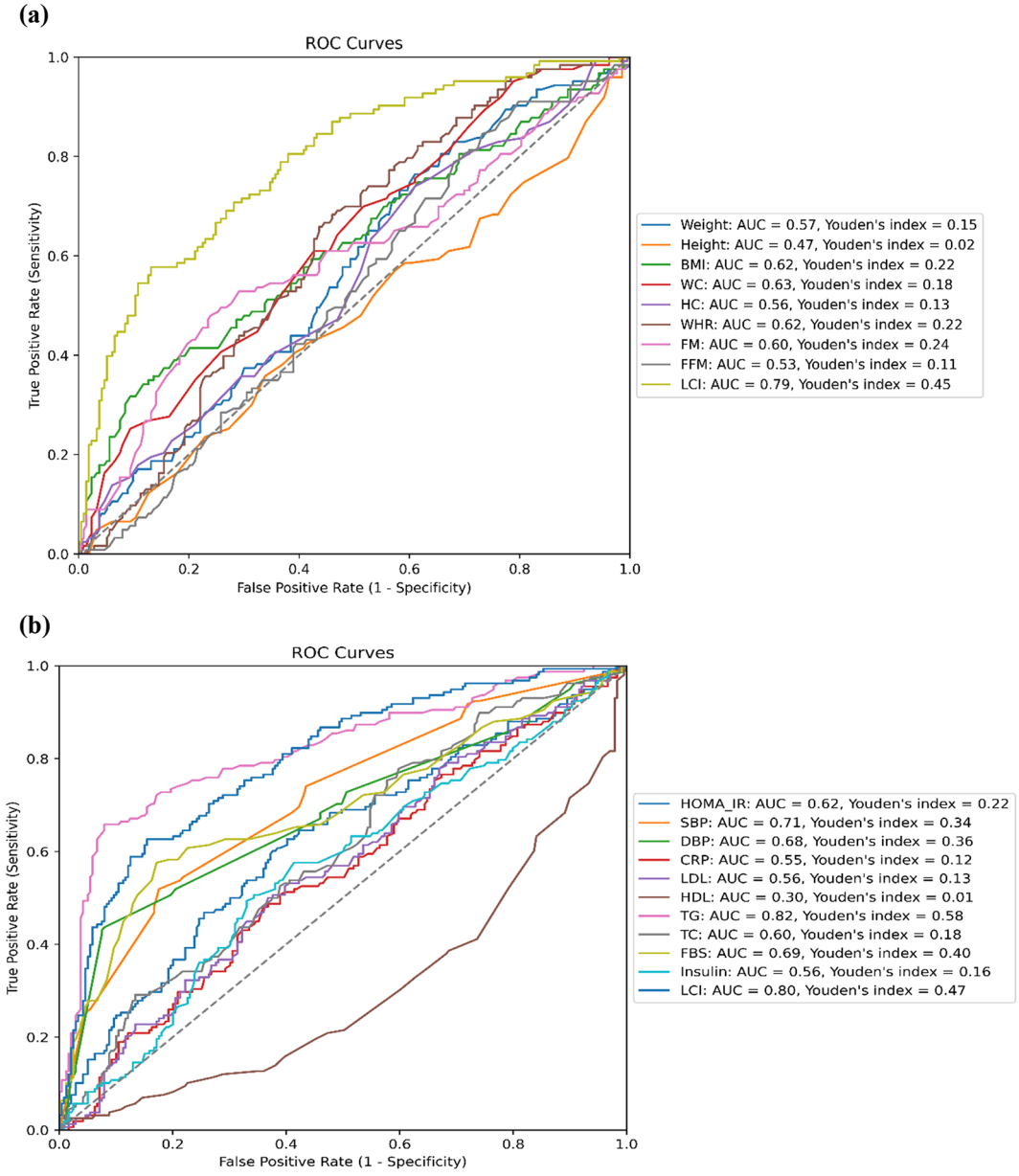

Fig. 1 Receiver operation characteristic (ROC) curve of (**a**) anthropometric parameters and LCI (**b**) biochemical variables and LCI for prediction of metabolic syndrome

Table 6 Receiver operating characteristic curves (ROC) analysis of LCI and anthropometric parameters for metabolic syndrome

Area under the curve											
Variables	**AUC**	**SE**	***P* value**	**95% CI**		**Youden's index**	**Optimal cut-points**	**Sensitivity**	**Specificity**	**True positive rate**	**False positive rate**
				Lower bound	**Upper bound**						
Weight (kg)	0.57	0.032	**0.031**	0.508	0.633	0.15	88.90	0.76	0.39	0.76	0.61
Height (cm)	0.47	0.033	**0.448**	0.410	0.541	0.02	171	0.36	0.66	0.36	0.34
BMI (kg/m^2)	0.62	0.033	**< 0.001**	0.557	0.685	0.22	37.78	0.34	0.88	0.34	0.12
WC (cm)	0.63	0.031	**< 0.001**	0.570	0.891	0.18	107	0.70	0.48	0.70	0.52
HC (cm)	0.65	0.033	**0.102**	0.490	0.617	0.13	112	0.74	0.39	0.74	0.61
WHR	0.62	0.030	**< 0.001**	0.561	0.681	0.22	0.94	0.68	0.54	0.68	0.46
FM (%)	0.60	0.034	0.003	0.531	0.663	0.24	28.20	0.48	0.76	0.48	0.24
FFM (%)	0.53	0.032	**0.401**	0.465	0.590	0.11	49.80	0.85	0.27	0.85	0.73
LCI	0.79	0.026	**< 0.001**	0.557	0.685	0.45	22.66	0.58	0.87	0.58	0.13

LCI, Lipoprotein combine index; BMI, Body mass index; WC, Waist circumference; HC, Hip circumference; WHR, Waist to hip ratio; FM, Fat mass; FFM, Fat free mass; AUC, Area under the curve; SE, Standard error; CI, Confidence interval; ROC, Receiver operating characteristic

Table 7 Receiver operating characteristic curves (ROC) analysis of LCI and metabolic risk factors for metabolic syndrome

Area under the curve											
Variables	**AUC**	**SE**	***P* value**	**95% CI**		**Youden's index**	**Optimal cut-points**	**Sensitivity**	**Specificity**	**True positive rate**	**False positive rate**
				Lower bound	**Upper bound**						
HOMA-IR	0.62	0.029	**< 0.001**	0.565	0.679	0.22	3.22	0.63	0.60	0.63	0.40
SBP (mmHg)	0.71	0.027	**< 0.001**	0.660	0.764	0.34	127	0.52	0.82	0.52	0.18
DBP (mmHg)	0.68	0.029	**< 0.001**	0.625	0.738	0.36	87	0.44	0.92	0.44	0.08
CRP (mg/dl)	0.55	0.030	**0.071**	0.496	0.611	0.12	4.78	0.48	0.64	0.48	0.36
LDL (mg/dl)	0.56	0.029	**0.031**	0.506	0.621	0.13	128	0.50	0.63	0.50	0.37
HDL (mg/dl)	0.30	0.027	**< 0.001**	0.248	0.354	0.01	74	0.03	0.98	0.03	0.02
TG (mg/dl)	0.82	0.023	< 0.001	0.775	0.865	0.58	151	0.66	0.92	0.66	0.08
TC (mg/dl)	0.60	0.029	**0.001**	0.546	0.658	0.18	171	0.78	0.40	0.78	0.60
FBS (mg/dl)	0.69	0.029	**< 0.001**	0.633	0.745	0.40	101	0.58	0.83	0.58	0.17
Insulin (mIU/l)	0.56	0.030	**0.039**	0.503	0.619	0.16	15.40	0.51	0.66	0.51	0.34
LCI	0.80	0.023	**< 0.001**	0.751	0.841	0.47	22.66	0.63	0.85	0.63	0.15

HOMA-IR, Homeostatic Model Assessment for Insulin Resistance; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure; CRP, C-reactive Protein; LDL-C, Low Density Lipoprotein Cholesterol; HDL-C, High Density Lipoprotein Cholesterol; TG, Triglyceride; TC, Total Cholesterol; FBS, Fasting blood sugar; LCI, Lipoprotein combine index; AUC, Area under the curve; SE, Standard error; CI, Confidence interval; ROC, Receiver operating characteristic

In analysis of predictive power of the LCI in comparison of anthropometric variables, biochemical parameters and blood pressure separately for males and females (Fig. 2 and Supplementary Tables 5–7) the same results were observed; among anthropometric variables, LCI had the highest AUC of all of the anthropometric variables both in men and women. Among biochemical variables, LCI was in the second level of importance in prediction of metabolic syndrome after TG among men and women.

Discussion

To the best of our knowledge, there is no information about the LCI index and its association with metabolic syndrome at the present. In the present study, we found that the LCI was in strong association with unfavorable lipid profile, serum glucose and CRP in a community based sample of obese individuals. In addition, we also found that the LCI index had an acceptable power in identification of metabolic syndrome in obese individuals after TG.

It is well-known that dyslipidemia is in close association with inflammation and is a central driving factor for its development and progress; numerous studies have shown that elevated levels of TG, TC, LDL-C, and a reduced HDL-C are associated with inflammation and increased serum CRP values [20–22]. Increased pro-inflammatory cytokines of interleukin-6, tumor necrosis factor-α in hyperlipidemic male subjects [20], interactive effects of increased CRP and dyslipidemia on cardiovascular diseases after 12-years follow-up [23] and the role of increased CRP levels in progression of dyslipidemia in patients with diabetes [24] has been reported before. CRP is a mediating factor in development of atherosclerosis via disturbed lipid metabolism and it is possibly via

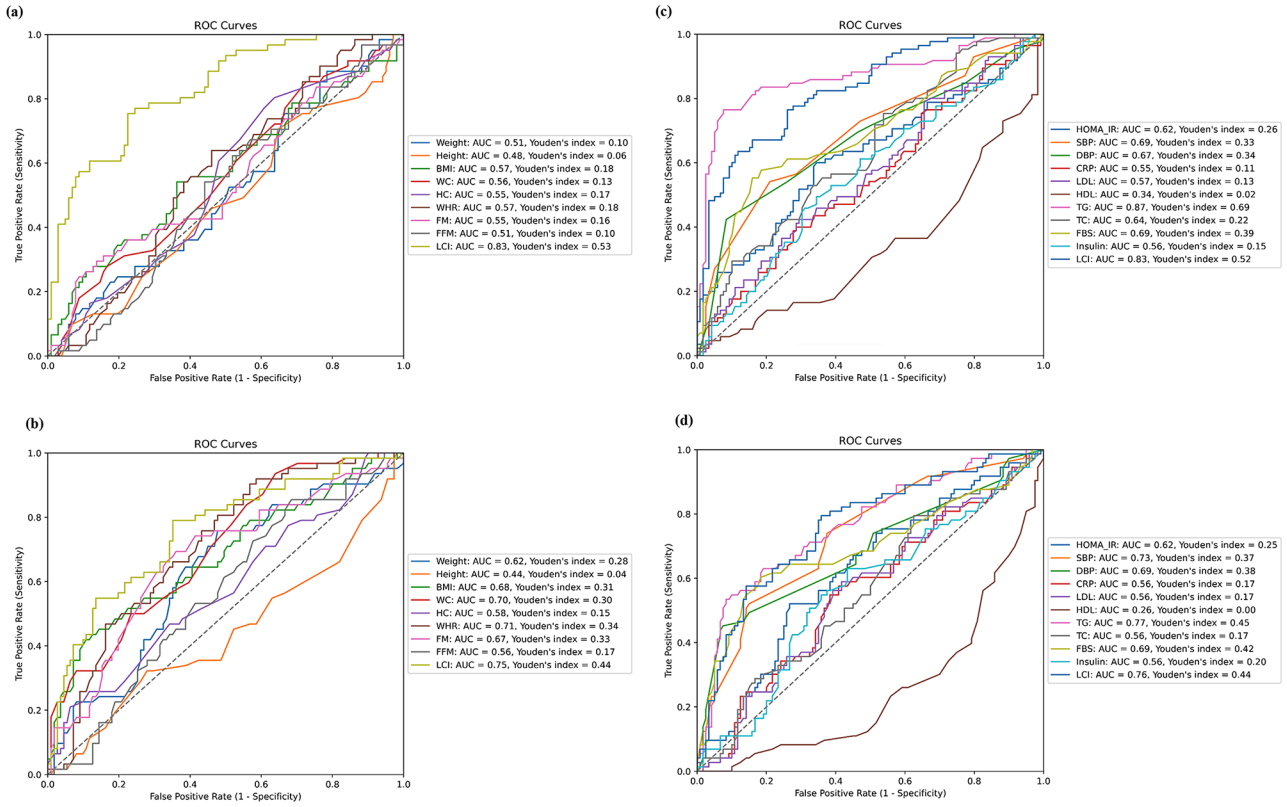

Fig. 2 Receiver operation characteristic (ROC) curve of (**a**) anthropometric parameters and LCI among men (**b**) anthropometric parameters and LCI among women, (**c**) biochemical variables and LCI for prediction of metabolic syndrome among men (**d**) biochemical variables and LCI for prediction of metabolic syndrome among women

the central role of oxidative stress [25, 26]; importantly, an individual lipid biomarker cannot fully reflect the body's lipid metabolism status, and utilizing a combination of various lipid parameters, like LCI, could provide a more comprehensive assessment of lipid status, thereby improving the accuracy of CVD identification in different disease statues [3, 8, 10, 11].

Also, the positive association between LCI and serum FBS in our study, is in consistent with previous studies [27, 28] and elevated triglyceride concentrations but not cholesterol has the same effect of elevated both triglyceride and cholesterol together to increase serum glucose values among 438 diabetic and no-diabetic subject [29]. In our study, those at the highest quartile of LCI had higher energy-adjusted intakes of red meat and fruits. There are numerous evidence about the positive association between red meat consumption and dyslipidemia; in a prospective cohort study of 20,407 Korean adults, being at the highest quintile of dietary red meat made people to have 34% and 10% greater risk of hypercholesterolemia in both men and women, and further, a 58% and 17% greater risk of increased LDL-C and dyslipidemia, in men, compared to the lowest consumption group [30]. In another study among Henan cohort adults, "meat" (high intakes of red meat) dietary pattern was positively related to the risk of dyslipidemia (OR: 1.10; 95% CI: 1.05–1.16, $p < 0.05$) [31]. In our study, the association between LCI and metabolic or inflammatory parameters were more pronounced among men than in women; among men, LCI was is positive association with CRP and a negative association with HDL ($P < 0.05$); while among women, higher CRP, TG, TC and lower HDL was observed in highest versus lowest quartiles of LCI ($P < 0.05$). In a similar study, LCI was in stronger association with incidence of non-alcoholic fatty liver disease among women compared with men [32]. The transport of fat in the blood is approximately twice as fast in women as men. Disease states such as obesity and diabetes are associated with greater lipoprotein abnormalities in women compared with men. A greater increment in cardiovascular disease risk in women is linked to these abnormalities. A greater change in triglyceride level and a lesser change in low-density lipoprotein are observed in women than men with high-carbohydrate or high-fat feeding [33]. In the study by Ji et al. [34] the prevalence of diabetes was associated with HDL-c and TG in women and LDL-c/HDL-c, TG/HDL-c, and TC/HDL-c ratios were associated with the diabetes prevalence only in women. These differences are probably due to the sex hormones

and their interaction with blood lipids in women [35]. Further studies will help to understand the underlying mechanisms.

In the current study, the predictive power of LCI was lower than TG but higher than all other biochemical and anthropometric parameters. TG achieved the greatest AUC for metabolic syndrome. TG is an integral part in metabolic syndrome definition and in some of the previous studies, TG or its related index, TyG achieved the highest AUC for prediction of metabolic syndrome [36]. However, the association of TyG index which is estimated as $\ln\frac{TG\times FBS}{2}$, is considered before and it considers low number of biomarkers compared with LCI (e.g. is $\frac{TC\times TG\times LDL}{HDL}$) [36]. Also, it is common sometimes for these indices to achieve lower AUC for prediction of disease risk compared with the classical lipid biomarkers. For example, in the study by Wang et al. [37], fasting blood sugar had the highest AUC compared with TyG index or TyG-BMI index in prediction of diabetes because it is an inherent biomarker for diabetes diagnosis (same as TG for metabolic syndrome). Or in the other study by Yang H et al. [38], TyG index has a non-significant difference in AUC compared with triglyceride in prediction of metabolic syndrome among women with PCOS. In other study also, HbA1C (that is a diagnostic biomarker for diabetes) achieved higher AUC compared with HbA1C/HDL index in prediction of diabetes mellitus [39]. Similar other studies are also available in literature.

The LCI was initially suggested by Wu T et al. [40], as one of the atherogenic indices for predicting acute coronary syndrome. However, the literature offers no detailed explanation regarding the rationale behind its equation. The LCI was formulated to incorporate multiple lipid components, providing an overall representation of dyslipidemia rather than focusing on individual parameters. Total cholesterol (TC) is included in the numerator as a general indicator of the lipid profile, enhancing the emphasis on lipid imbalances when combined with LDL-C and HDL-C. Despite its unconventional mathematical structure, the LCI's clinical significance lies in its ability to underscore the imbalance between atherogenic and anti-atherogenic lipoproteins, aligning with the metabolic disruptions commonly observed in metabolic syndrome (MetS).

Numerous studies have highlighted the LCI's role in predicting coronary artery disease, cardiovascular conditions, non-alcoholic fatty liver disease (NAFLD), and diabetes. For example, higher LCI levels have been observed in patients undergoing coronary artery bypass graft (CABG) compared to controls [3], as well as in cases of arterial stiffness [41], and CAD [9, 42]. Research has also established its association with metabolic-associated fatty liver disease [43], increased odds of coronary artery risk [42], acute coronary syndrome [44], severity of CAD [45], NAFLD [32] and diabetes [46] and its predictive capability for atherosclerotic diseases [11]. The LCI is intended not as a replacement but as a complementary index, integrating lipid parameters to provide a holistic view of lipid metabolism disturbances.

In conclusion, in the current study, LCI was in positive association with CRP, lipid profile and FBS among obese individuals. Although LCI was able to identify metabolic syndrome with an acceptable AUC, however, its power for prediction of metabolic syndrome was lower than TG. Because of the very low number of studies, further studies are needed to make better conclusion. Also, longitudinal studies, are needed to identify the predictive power of LCI to better elucidate its utility in routine practice in detection or management of metabolic syndrome.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12902-024-01813-z.

Supplementary Material 1

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Author contributions
Both authors were involved in data collection and subjects' recruitment. They also were involved in hypothesis generation and statistics. KZ was involved in data analysis and supervision of the project. He also performed the statistical approaches, both authors also wrote the first draft of the paper.

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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. All methods in the current research were performed in accordance with the declaration of Helsinki's guidelines and regulations. The protocol of the current study has been approved by the ethics committee of Anshan Normal University.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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