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# The association between low carbohydrate dietary score (LCDS) and cardiovascular risk factors: results from the Shiraz Heart Study (SHS)

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## Abstract

**Background** Cardiovascular disease (CVD) is a major cause of death and recent studies have highlighted the potential role of dietary carbohydrate indices in cardiovascular health. Given the controversial results in this field, the aim of this study was to investigate the association between low carbohydrate dietary score (LCDS) and CVD risk factors in a population of Iranian adults.

**Methods** This cross-sectional study was conducted within the framework of the Shiraz Heart Study (SHS) including 1982 adults. The validated 168 items food frequency questionnaire (FFQ) was used to assess participants' dietary intakes. To investigate the association between LCDS and cardiometabolic risk factors, logistic regression, was conducted.

**Results** During 5 years of follow-up, a total of 1982 adults, with a mean age of  $53.07 \pm 8.38$  years, were included to the analysis. The adjusted model based on known confounding factors (age, sex, smoking, physical activity, energy intake and body mass index) revealed a significant decrease in a body shape index (ABSI) (OR=0.70, 95% CI= (0.50 to 0.98),  $P=0.038$ ) comparing highest LCDS tertile vs. the reference. In contrast, risk of hypertension (HTN), body mass index (BMI), waist-hip ratio (WHR), triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-c), high-density lipoprotein cholesterol (HDL-c), atherogenic index of plasma (AIP), and fasting blood sugar (FBS) were not significantly associated with LCDS.

**Conclusion** Current findings suggest that adherence to a low carbohydrate diet reduces ABSI, a main indicator of central obesity.

**Keywords** Cardiovascular disease, Low carbohydrate diet, Nutrition, Obesity, Cohort study

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## Introduction

Cardiovascular disease (CVD) is the leading cause of death worldwide, with 422.7 and 17.9 million annual cases and deaths, respectively [1]. CVDs are responsible for one-third of global mortality and one-half of non-communicable diseases (NCDs) [2, 3], while more than three-quarters of CVD mortality was seen in low-to middle-income countries due to poor health facilities and low public awareness [4]. Iran has one of the highest rates of CVD incidence in Asia and around the world, with over 9,000 cases per 100,000 individuals [5]. Dietary choices have a significant impact on the development of *obesity*, dyslipidemia, hyperglycemia, insulin resistance (IR) and diabetes mellitus (DM), which are the major risk factors for CVD [4, 6–9].

It is crucial to comprehend the association between carbohydrate intake and the prevalence of *overweight*, *obesity* and risk factors for CVD, as dietary carbohydrates are acknowledge as the primary energy source [10, 11]. A low-carbohydrate diet (LCD) is defined as a dietary pattern where carbohydrates make up less than 45% of daily energy [11]. Nowadays, carbohydrate-restricted diets have become a popular *weight loss strategy*. It remains unclear whether LCD can improve biomarkers of cardiovascular health or diseases [11]. However, recent studies have shown that individuals who adhere to a LCD experience greater reductions *in weight*, total cholesterol (TC) and triglyceride (TG), and increased high density lipoprotein cholesterol (HDL-c) levels, compared to the counterparts on a low-fat diet (LFD) [12–14]. On the other hand, clinical studies contradict the benefits of a LCD and its relationship with metabolic diseases and CVDs [11, 15, 16]. In a cross-sectional study, the results showed that LCD was not associated with CVD risk factors in a group of Iranian women [11]. In addition, some studies have reported that LCD increased LDL-c, fasting glucose, TG levels, and blood pressure (BP) [17–19]. Therefore, given the inconsistent findings in the field, the aim of this study was to investigate the relationship between LCD scores and CVD risk factors in Iranian adults.

## Methods

### Study design and population

The present cross-sectional study was conducted within the framework of the Shiraz Heart Study (SHS). SHS is a prospective cardiovascular cohort, which planned to be performed in a middle-aged population in Shiraz, Iran, to investigate the CVD risk factors. The present study used the data gathered after 5 years of follow-up.

The details of participant allocation in SHS have been documented elsewhere [20]. Briefly, by allocating 25 clinics and 400 participants for each, the whole sample size of SHS reached 10,000 people, but only 1982 of these participants were eligible to enter the current study.

Free-CVD participants who were aged  $\geq 30$  years at baseline were included if they had lived in the urban areas of Shiraz for at least one year from the date of entering the study and had no plans of migration in the next few years from baseline assessments. Individuals who resided far from the study centers, had mental or physical disabilities, or had a history of coronary artery bypass grafting, percutaneous coronary intervention, positive angiographic reports, invalid dietary data, and a clinical evidence related to CVD outcomes were excluded.

Participants of the study signed informed consent documents. The protocol of the current study was approved by the Ethics Committee of Shiraz University of Medical Sciences (ID: IR.SUMS.SCHEANUT.REC.1401.030).

### Data collections and measurements

Dietary intakes and other baseline data, including sociodemographic characteristics (age, gender, smoking, education, physical activity, number of children, dietary intakes), anthropometric indices (body weight, height, body mass index (BMI), waist and hip circumference, waist-hip-ratio (WHR)), laboratory data (such as fasting blood sugar (FBS), blood lipid profile (LDL-c, HDL-c, triglyceride (TG), total cholesterol (TC))), and health status (medications in use, family history of diseases) were collected. The body weight of participants was measured (in the A.M.) using a calibrated digital scale (Sahand, Iran) with an accuracy of 100 g when participants were minimally clothed without shoes. In addition, the participants' body height was assessed using a stadiometer with an accuracy of 0.1 cm, while standing next to the wall without shoes, and the shoulders, hips, and heels were tangential to the wall, and the head was facing forward. Then, BMI was calculated by dividing the body weight (kg) by the square of the height ( $m^2$ ). The waist circumference (WC) was measured considering the midpoint between the last rib and the iliac crest, and the hip circumference, the largest circumference of the hip was measured by an inflexible meter with an accuracy of 0.1 cm. WHR was obtained by dividing the waist circumference by the hip circumference. A body shape index (ABSI) was calculated through the following formula [21]:

$$\frac{wc(m)}{\left(BMI\left(\frac{kg}{m^2}\right)\right)^{\frac{2}{3}} \times (height(m))^{\frac{1}{2}}}$$

Also, the atherogenic index of plasma (AIP) was calculated from the combination of triglyceride and HDL-c indices with the following equation:  $AIP = \log(TG/HDL-c)$ .

Before measuring the blood pressure, the participants were asked to rest for 10 min, then their blood pressure

was measured from both arms consecutively using a digital device (OMRON, Germany); and repeated twice with an interval of 10 min. At the end, the mean of three measurements was recorded as the final blood pressure.

Blood samples were collected after 12 h of fasting and analyzed in the laboratory of Shiraz heart center. FBS, TC, and TG were measured by an enzymatic method with an automatic analyzer using kits (Byrex, Iran). HDL-c was determined enzymatically after precipitation of other lipoproteins with dextran magnesium-chloride-sulfate. Friedewald's equation was used to calculate LDL-c in subjects with TG < 400 mg/dL, however, direct measurement of LDL-c via the turbidity method was performed for subjects with TG ≥ 400 mg/dL.

The International Physical Activity Questionnaire (IPAQ) was used to assess the participants' physical activities over a week. Questionnaire scores were reported as continuous chains (by METs index) [22].

The validated 168-item food frequency questionnaire (FFQ) was used to assess the participants' dietary intakes [23]. The amount and frequency of food consumption were evaluated on a daily, weekly, monthly, or yearly basis via an interview with experienced nutritionists at the health center of Shiraz. The amount of each food was converted to grams per day using household standards. The United States Department of Agriculture (USDA) was executed to compute daily energy and

dietary components. The Iranian food composition table was used for traditional foods, which were not reported by USDA [24].

#### LCD score

Seven criteria were considered to calculate the LCDS: the intake amounts of dietary carbohydrate (% total energy intake), fiber (gr/1000 kcal), monounsaturated fatty acids (MUFAs) (% total energy intake), the ratio of omega-3 to omega-6 (n3/n6 PUFA), refined carbohydrate (% total energy intake), vegetable protein (% of total energy intake), and glycemic load (GL) and for each criterion, a score between 0 and 10 was defined.

The participants who had the highest intake in terms of fiber, MUFAs, n3/n6 PUFA, and vegetable proteins scored 10, and those who had the lowest intake scored 0. Regarding the intake of total carbohydrates, refined carbohydrates and GL, participants with the highest intakes scored 0 and those with the lowest intakes scored 10. Then the scores of each item were collected to create a total score that ranged from 0 (lowest consumption of fat and protein and highest consumption of carbohydrates) to 70 (highest consumption of protein and fat and lowest consumption of carbohydrates) [25].

#### Statistical methods

Data were analyzed using statistical software SPSS version 26 (SPSS Inc., Chicago, IL, USA). Quantitative data were reported as mean (standard deviation (SD)) and qualitative data were reported as frequency (%). The normality of the variables was determined by the Kolmogorov-Smirnov test. To report the amount of daily energy intake and dietary components across tertiles of LCDS and related correlations, we used analysis of variance (ANOVA). To investigate the relationship between LCDS and CVD risk factors, binary logistic regression in a crude and adjusted model, was used. In the adjusted model, the effects of age, gender, smoking, physical activity, daily energy intake, and BMI were controlled, except for BMI and ABSI, which were not adjusted for BMI. Moreover, dietary intakes were adjusted based on 1000 kcal daily energy intake. *P*-values less than 0.05 were considered significant.

## Results

### Baseline characteristics

Participants were predominantly female (52.9%), and mean age (SD) was 53.07 (8.38). The baseline characteristics of participants are shown in Table 1. Regarding dietary intakes, compared to the participants in the lowest tertile of LCDS, those in the third tertile had a higher intake of energy, protein, total fat, cholesterol, fiber, dairy products, fruits, and vegetables (*P*=0.001). However, lower intakes of total carbohydrates, whole grains,

**Table 1** Baseline characteristics of the study population

Characteristic	Total* (n = 1982)
Age (year)	53.0 ± 8.3
Female (%)	1045 (52.9%)
Current smoker (%)	446 (22.6%)
Physical activity (Low/moderate/high)	534 (50.3%) 263 (24.8%) 264 (24.9%)
SBP (mmHg)	123 ± 16
DBP (mmHg)	80 ± 10
Weight (kg)	74.4 ± 13.4
BMI (Kg/m <sup>2</sup> )	27.8 ± 4.6
WC (cm)	98.4 ± 11.1
Hip Circumference (cm)	104 ± 8.7
WHR (cm <sup>2</sup> )	0.9 ± 0.6
ABSI	0.08 ± 0.0
TG (mg/dl)	147 ± 72.7
TC (mg/dl)	189 ± 40.6
LDL-c (mg/dl)	108 ± 28.0
HDL-c (mg/dl)	47.8 ± 10.5
AIP	0.4 ± 0.2
FBS (mg/dl)	104 ± 28.8

\*Data are expressed as mean ± SD for continuous or frequency (percent) for categorical variables; Abbreviations: (HTN: Hypertension), (BMI: Body mass index), (WHR: Waist-hip-ratio), (ABSI: A body shape index), (TG: Triglyceride), (TC: Total cholesterol), (LDL-c: Low-density-lipoprotein cholesterol), (HDL-c: High-density-lipoprotein cholesterol), (AIP: Atherogenic index of plasma), (FBS: Fasting blood sugar)

sweets, and refined carbohydrates were seen in the higher tertiles of LCDS ( $P=0.001$ ) (Table 2).

#### Association of LCDS with CVD risk factors

The association of LCDS with the CVD risk factors is shown in Table 3. Higher LCDS was associated with higher BMI and WHR in the crude model in the participants of the third tertile in comparison with the reference ones. However, after adjusting for confounders (age, gender, smoking, physical activity, daily energy intake and BMI, except for BMI and ABSI, which were not adjusted for BMI), the significant associations were attenuated. The adjusted model, based on known confounders, revealed a significant reduction in ABSI (OR=0.70, 95% CI= [0.50 to 0.98]) following higher LCDS vs the reference. In contrast, HTN (OR=0.94, 95% CI=[0.68 to 1.31]), BMI (OR=1.01, 95% CI=[0.71 to 1.43]), WHR (OR=0.84, 95% CI=[0.50 to 1.40]), TG (OR=0.82, 95% CI=[0.59 to 1.15]), TC (OR=0.99, 95% CI=[0.71 to 1.38]), LDL-c (OR=0.88, 95% CI=[0.59 to 1.20]), HDL-c (OR=0.92, 95% CI=[0.67 to 1.27]), AIP (OR=1.16, 95% CI=[0.83 to 1.62]) and FBS (OR=0.87, 95% CI=[0.52 to 1.45]) were not significantly associated with LCDS when comparing the 3rd tertile vs 1st one.

#### Discussion

In the present cross-sectional study, we found an inverse association between LCDS and ABSI independent from known confounders (age, gender, smoking, physical activity, and daily energy intake). In contrast, the HTN, FBS, AIP, lipid profiles, BMI, WC, and WHR were not found to be associated with higher LCDS, which is in accordance with Jafari-Maram et al., who reported a lack of association between low carbohydrate diet and cardiovascular risk factors [11].

Mirmiran and colleagues [25] found an inverse association between metabolic syndrome components and higher LCDS during 3.6 years of follow-up, which was divergent from the results of the current study regarding the association between cardiovascular risk factors and LCDS, which could be due to the difference in the study design. In a review assessing RCTs, the authors concluded that low carbohydrate diets may improve cardiovascular risk factors, accelerate weight loss, and even recommended a low carbohydrate diet as an appropriate strategy for weight management and cardiovascular protection through improving the carbohydrate quality and quantity [26]. Moreover, in a meta-analysis evaluating the effects of low carbohydrate diets in RCTs, Dong et al. found that these diets could possibly improve cardiovascular risk factors in study durations of shorter than 6 months, while for confirming the beneficial effects in the longer-term, further studies are warranted [27]. One of the main reasons for such a different findings could pertain to the fact that the aforementioned reviews assessed the results of the RCTs that could better demonstrate the causal relationships compared to the cross-sectional studies.

Previous studies including cohort and RCTs have shown controversial results regarding the relationship between body weight and waist circumference with low carbohydrate diet [26, 28–32]. In the present study, low carbohydrate diet was associated with lower ABSI. ABSI emphasizes on the distribution of fat, especially the abdominal fat, by considering WC. A higher BMI could either reveal a higher amount of lean mass or fat mass and unlikely to be a good marker of central obesity [21]. ABSI included both WC and BMI, that could be considered as a marker of fat distribution and fat accumulation [21, 33]. Even it was asserted that ABSI could be a

**Table 2** Baseline dietary intakes of the participants across tertiles of low carbohydrate diet score (LCDS)

Dietary intakes*	Total (n = 1982)	Low carbohydrate diet score (LCDS)			P <sup>#</sup>
		T1 (n = 661)	T2 (n = 661)	T3 (n = 660)	
Energy (kcal)	2838 ± 1065	2640 ± 1047	2809 ± 971	3064 ± 1123	0.001
Carbohydrate (g/d)	159 ± 16.9	169 ± 12.5	158 ± 15.8	149 ± 15.7	0.001
Protein (g/d)	34.6 ± 5.9	31.3 ± 4.2	34.5 ± 5.0	38.0 ± 6.3	0.001
Fat (g/d)	28.2 ± 7.5	24.5 ± 5.5	28.7 ± 7.6	31.3 ± 7.7	0.001
Dietary fibers (g/d)	16.1 ± 4.8	14.0 ± 3.3	16.0 ± 4.4	18.3 ± 5.4	0.001
Cholesterols (g/d)	76.8 ± 32.8	68.6 ± 30.1	77.6 ± 29.7	84.3 ± 36.1	0.001
Whole grains (g/d)	37.4 ± 33.5	38.1 ± 38.3	40.5 ± 34.6	33.9 ± 26.3	0.001
Fruits (g/d)	214 ± 109	198 ± 106	221 ± 110	223 ± 108	0.001
Vegetables (g/d)	190 ± 93.7	166 ± 76.9	189 ± 87.9	215 ± 107	0.001
Refined grains (g/d)	111 ± 52.7	149 ± 54.2	105 ± 40.0	77.7 ± 32.8	0.001
Dairy (g/d)	98.7 ± 70.6	83.5 ± 63.2	100 ± 68.7	112.7 ± 76.3	0.001
Sweets (g/d)	46.2 ± 44.0	57.2 ± 53.2	43.8 ± 39.4	37.2 ± 34.6	0.001

\* Total dietary intakes were adjusted based on 1000 Kcal intake of daily energy; data are expressed as mean ± SD;

# P-values less than 0.05 are considered significant and one-way ANOVA was used for the analysis

**Table 3** Odds ratio of cardiometabolic risk factors according to LCDS tertile

Models	T1 (n=661)	T2 (n=661)	T3 (n=660)	P <sup>#</sup>
<b>HTN (mmHg)</b>				
Crude	1.00	0.97 (0.77–1.21)	0.87 (0.70–1.09) 0.23	0.23
Adjusted*	1.00	0.91 (0.66–1.26)	0.94 (0.68–1.31) 0.57	0.57
<b>BMI (kg/m<sup>2</sup>)</b>				
Crude	1.00	1.34 (1.05–1.71)	1.48 (1.16–1.89) <0.001	<0.001
Adjusted	1.00	1.18 (0.84–1.68)	1.01 (0.71–1.43) 0.94	0.94
<b>WHR</b>				
Crude	1.00	<b>1.50 (1.17–1.94)</b>	<b>1.78 (1.38–2.31) &lt;0.001</b>	<0.001
Adjusted	1.00	0.81 (0.50–1.30)	0.84 (0.50–1.40) 0.51	0.51
<b>ABSI</b>				
Crude	1.00	0.88 (0.71–1.11)	0.82 (0.66–1.03) 0.09	0.09
Adjusted	1.00	0.82 (0.59–1.13)	<b>0.70 (0.50–0.98) 0.03</b>	<b>0.03</b>
<b>TG (mg/dl)</b>				
Crude	1.00	0.97 (0.77–1.21)	0.94 (0.75–1.18) 0.64	0.64
Adjusted	1.00	0.89 (0.64–1.23)	0.82 (0.59–1.15) 0.26	0.26
<b>TC (mg/dl)</b>				
Crude	1.00	1.05 (0.84–1.32)	1.02 (0.81–1.27) 0.86	0.86
Adjusted	1.00	1.08 (0.78–1.49)	0.99 (0.71–1.38) 0.99	0.99
<b>LDL-c (mg/dl)</b>				
Crude	1.00	1.04 (0.80–1.37)	1.10 (0.85–1.44) 0.44	0.44
Adjusted	1.00	0.95 (0.65–1.39)	0.88 (0.59–1.30) 0.52	0.52
<b>HDL-c (mg/dl)</b>				
Crude	1.00	0.99 (0.79–1.24)	0.98 (0.79–1.22) 0.91	0.91
Adjusted	1.00	1.20 (0.88–1.65)	0.92 (0.67–1.27) 0.63	0.63
<b>AIP</b>				
Crude	1.00	1.11 (0.88–1.39)	1.23 (0.98–1.54) 0.06	0.06
Adjusted	1.00	0.97 (0.70–1.35)	1.16 (0.83–1.62) 0.36	0.36
<b>FBS (mg/dl)</b>				
Crude	1.00	0.93 (0.64–1.34)	0.81 (0.57–1.15) 0.23	0.23
Adjusted	1.00	1.02 (0.60–1.71)	0.87 (0.52–1.45) 0.59	0.59

Abbreviations: (HTN: Hypertension), (BMI: Body mass index), (WHR: Waist-hip-ratio), (ABSI: A body shape index), (TG: Triglyceride), (TC: Total cholesterol), (LDL-c: Low-density-lipoprotein cholesterol), (HDL-c: High-density-lipoprotein cholesterol), (AIP: Atherogenic index of plasma), (FBS: Fasting blood sugar)

\*Adjusted for of age, gender, smoking, physical activity, daily energy intakes and BMI (except for BMI and ABSI that are not adjusted for BMI)

#  $P < 0.05$  was considered statistically significant (Obtained from logistic regression)

Results are defined as odds ratio (OR) and 95% confidence interval (CI)

better predictor of diabetes and all-cause mortality than BMI in some populations [33, 34], a meta-analysis concluded that it underperformed BMI and WC in predicting chronic diseases [35]. A recent study shed light on the clinical performance of ABSI in identifying the presence of metabolic syndrome and arterial stiffness [36].

It was previously reported that low carbohydrate diets could decrease the odds of central obesity, especially through decreasing fat mass [26, 37]. Moreover, it has been asserted that low carbohydrate diets could decrease waist circumference, to an even greater extent than low-fat diets [38–40]. Hence, the inverse association between LCDS and ABSI could likely confirm the protective effects of low carbohydrate diets on decreasing the risk of central obesity and high ABSI. Possibly, this could indirectly show the protective effects of low carbohydrate diets against central obesity and metabolic syndrome due to the correlations between ABSI and obesity [41] as it

was mentioned previously. However, this issue needs further investigation to achieve more definite results.

Further, in the current study, those in the higher tertiles of LCDS had higher dietary intakes of fruits, vegetables, fiber, and dairy products, and lower intakes of total carbohydrates, sweets, and refined carbohydrates, which can, possibly, beneficially affect WC and ABSI. This can partially justify the inverse association between ABSI and LCDS. Similar dietary intakes including lower consumption of total and refined carbohydrate and whole grains and higher intakes of dairy products was also reported in those with higher LCDS in a study by Jafari-maram and colleagues in an Iranian population [11]. Shirani et al. also reported lower intakes of total and refined carbohydrates in those with LCDS [15]. Hence, a lower carbohydrate intake was accompanied by higher dietary fiber, dairy products and lower refined sugar in the present study, which could be associated with improvements in ABSI.



This finding was also confirmed by the studies reporting the decreasing effects of fiber [42] and dairy products [43] on WC.

There are several limitations to the interpretation of our findings as well. Of note, the cross-sectional design did not allow for the establishment of a causal relationship, only associations between variables. Although we attempted to control for potential confounders, residual confounding could remain because of unmeasured confounders. As the Iranian food composition table was incomplete, we mostly used the USDA nutrient databank for the dietary analyses. In addition, due to the differences in the food culture and dietary habits, it will be difficult to generalize our findings to other societies. However, the main strength of our study lies in the relatively large sample size of SHS. In addition, we used a validated FFQ for Iranian and measured variables in regard to the standard methods. Enrolled participants were free from CVD at baseline, which decreases the risk of diet modification in response to disease.

## Conclusion

Adherence to a low carbohydrate diet was not associated with main CVD risk factors in an Iranian population. So far, a low carbohydrate diet may be able to decrease ABSI, as an indicator of central obesity that includes WC as a main component in its calculation. A low carbohydrate diet in this study was accompanied by higher dietary intakes of fruits, vegetables, fiber, and dairy products, and lower intakes of total carbohydrates, sweets, and refined carbohydrates that could improve ABSI. Further investigations in different populations and with various study designs are needed to better confirm these results.

## Abbreviations

CVD	Cardiovascular disease
LCDS	low carbohydrate dietary score
SHS	Shiraz Heart Study
FFQ	Food frequency questionnaire
NCD	non-communicable disease
IR	Insulin resistance
DM	diabetes mellitus
HDL-c	high-density lipoprotein cholesterol
LDL-c	low-density lipoprotein cholesterol
TG	triglyceride
TC	total cholesterol
LFD	low fat diet
BP	blood pressure
BMI	body mass index
WHR	waist-hip ratio
FBS	fasting blood sugar
WC	waist circumference
ABSI	a body shape index
AIP	atherogenic index of plasma
IPAQ	International physical activity questionnaire
USDA	the United States Department of Agriculture
MUFA	monounsaturated fatty acid
PUFA	polyunsaturated fatty acid

GL	glycemic load
GI	glycemic index
SD	standard deviation
HTN	hypertension
RCT	randomized controlled trial

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## Author contributions

ZM and ZS conceptualized the study. MJZ, SSM, MS, IRJ, NP and FZ developed the methodology. MJ, ZM, CC and ZS wrote the original draft. MJ, MJZ and MN reviewed and edited the manuscript. ZS and MJZ supervised and administered the project and acquired funding. All of the authors contributed significantly to the work and accepted the final manuscript to be published.

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## Data availability

Data will be presented upon forwarding the request to the corresponding author (zahra\_2043@yahoo.com) and confirmation of the director of SHS (zibaem2@gmail.com).

## Declarations

### Ethics approval and consent to participate

We obtained written informed consent from all participants. Based on the ethical guidelines of the 1975 Declaration of Helsinki, the study protocol was approved by research Ethics Committee of Shiraz University of Medical Sciences, Shiraz, Iran IR.SUMS.SCHEANUT.REC.1401.030).

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

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