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Waterpipe smoking is associated with presence and severity of coronary artery disease: a propensity score-matched study

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Abstract

Background The prevalence of waterpipe smoking (WPS) has been increasing worldwide. This trend is alarming as WPS can negatively impact cardiovascular health. In the present study, we explored the association between WPS and the presence and severity of CAD.

Methods This study was a retrospective analysis of patients who underwent diagnostic coronary angiography at Tehran Heart Center between April 2021 and May 2022. Patients with a previous history of percutaneous coronary intervention and coronary surgery were excluded. Waterpipe smokers were matched with non-smokers based on age, gender, and cigarette smoking using a 1:4 propensity score matching model. Stenosis ≥ 50% in any coronary artery was considered a CAD diagnosis. Gensini score was also calculated to measure the severity of the CAD.

Results We reviewed the medical records of 8699 patients, including 380 waterpipe smokers. After matching, 1520 non-smokers with similar propensity scores to the waterpipe smokers were selected. Waterpipe smokers were more likely to have CAD than non-smokers (OR: 1.29; 95% CI: 1.04–1.60, $P = 0.021$). In addition, WPS increased the natural logarithm of the Gensini score by 1.24 (95% CI: 1.04–1.48, $P = 0.014$) in patients with atherosclerotic coronary disease.

Conclusion WPS may increase the risk of CAD independent of age, gender, and cigarette smoking. In addition, among patients with any degree of atherosclerosis in coronary arteries (GS > 0), WPS may lead to higher average GS, suggesting more severe atherosclerosis.

Highlights

- Waterpipe smokers were more likely to have CAD than non-smokers.
- Waterpipe smoking increased the severity of CAD in patients with atherosclerotic coronary disease.
- The association between CAD and waterpipe smoking was independent of age, gender, and cigarette smoking.

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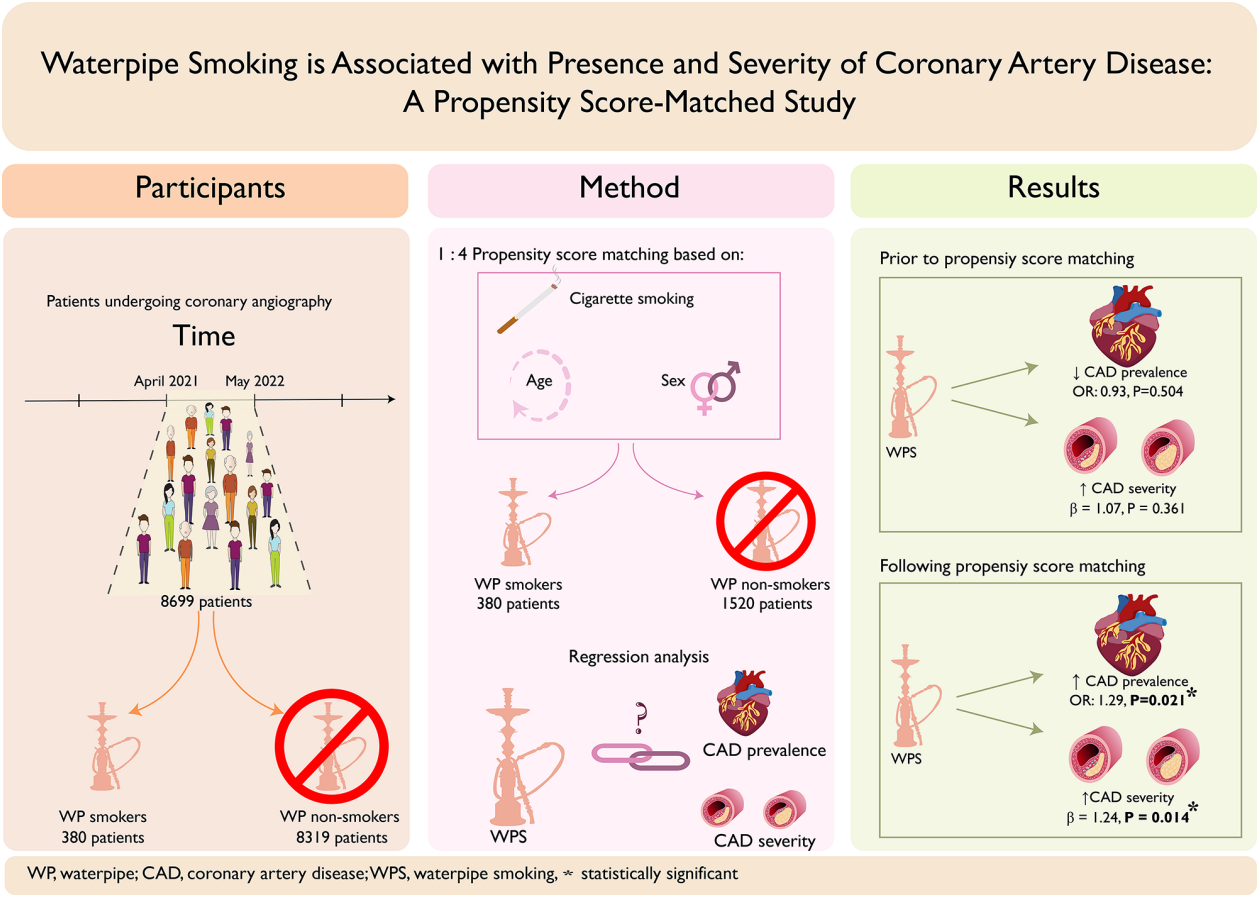
Graphical Abstract



Keywords Coronary artery disease, Hookah, Smoking, Waterpipe, Cardiovascular disease

Introduction

Waterpipe smoking (WPS), a less frequent tobacco smoking method, has become an increasingly popular social phenomenon worldwide in the previous decade [1]. While the prevalence of cigarette smoking (CS) has generally decreased as a result of population-based preventive initiatives, WPS and other forms of tobacco smoking have shown an upward trend over time due to misconceptions that WPS is less hazardous than CS [1, 2]. Contrary to this, WPS poses the same or even greater health risks than CS due to its extended smoking period and similar cardiorespiratory toxicants [3]. In fact, studies have shown that WPS exposes users to much more smoke than CS and has a greater carbon monoxide (CO) content and comparable nicotine level [3].

Several studies have been conducted on the estimation of the total prevalence of WPS in different areas of the world [4–7]. According to a nationwide survey, the prevalence of WPS in Iran was approximately 2.4% between 2006 and 2009 [8]. The reported prevalence rates of WPS among adults in the Middle East range from 5 to 36%, and among adolescents in Europe, it has been reported to be 10.9% [5, 6]. Although WPS has been a prevailing trend in the Eastern Mediterranean and Middle Eastern regions, there has been a recent increase in its popularity among young adults in Western countries as well [5, 9]. Therefore, it is imperative that future studies investigate the long-term effects of WPS on health outcomes, particularly its impact on the cardiovascular system.

Coronary artery disease (CAD) is the primary contributor to cardiovascular mortality in developed countries and is one of the leading causes of the increase in cardiovascular disease (CVD) burden in developing countries [10]. Cigarette smoking has been widely recognized as a well-established cardiovascular risk factor, and due to the similar cardiorespiratory toxicant profiles of WP and cigarettes, the same association would be expected regarding WPS. Although the association between WPS and cardiometabolic diseases has been shown in many studies [11, 12], the association between WPS and CAD was

inconsistent in previous studies. In addition, the exact magnitude of the associated risk with WPS is unknown. A recent meta-analysis suggested that WPS does not significantly increase the CAD risk [13], whereas other studies have shown a high CAD risk among WP smokers [14–17]. Therefore, in this study, we aimed to investigate the association between WPS and the presence and severity of CAD in a large sample of patients undergoing diagnostic coronary angiography.

Materials and methods

Participants

The present study was a retrospective analysis of adult patients who underwent diagnostic coronary angiography at Tehran Heart Center between April 2021 and May 2022. Patients with a previous history of percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) were excluded from the study. The ethics committee of Tehran University of Medical Sciences agreed to the study design (Ethics CODE: IR.TUMS.THC.REC.1400.004). All patients provided written informed consent at the start of the study.

Data collection

At baseline after obtaining written informed consent, trained nurses conducted face-to-face interviews using structured questionnaires to collect data on a large number of variables including (1) demographic variables, (2) past medical history (hypertension, diabetes mellitus, dyslipidemia, chronic kidney disease, congestive heart failure, atrial fibrillation, and arrhythmias), (3) history of previous angiography and premature CAD in first-degree family members (<55 years in men and <65 years in women). Patients were considered cigarette, opium, or alcohol users if they reported current or previous history of use. WPS was similarly defined as the current or previous history of WP use.

Body mass index (BMI) was calculated as weight (kg)/height^2 (m). Laboratory samples, including HDL, LDL, fasting blood glucose (FBS), triglycerides, total cholesterol, and hemoglobin levels, were obtained from blood samples. All data were obtained from the hospital records.

Main research variables

Two types of outcomes were used for the current study. First, CAD was used as a binary outcome to assess the association of WPS and CAD. Patients with stenosis < 50% in all coronary arteries were regarded as not having significant CAD while having stenosis ≥ 50% in any coronary arteries was considered a CAD diagnosis [14, 18]. Second, we used the Gensini score (GS) as a continuous outcome to assess the severity of CAD in WP smokers.

The calculation of GS has been previously described in detail [19]. Briefly, the different segments in each of the three main coronary branches (main left coronary artery/left anterior descending, left circumflex, and right coronary artery) were assigned a multiplying factor based on the functional importance of the myocardium that the arterial segment supplies (e.g., ×5 for the main left and ×0.5 for second diagonal). Each segment was then scored from 0, 1, 2, 4, 8, 16, or 32 based on the severity of the obstruction (no occlusion, 25%, 50%, 75%, 90%, 99%, or complete occlusion, respectively) and the roentgenographic appearance of concentric lesions and eccentric plaques on the angiogram. The obstruction scores in each segment were multiplied by the multiplying factor. Then, all the scores were summed up to determine the GS [19].

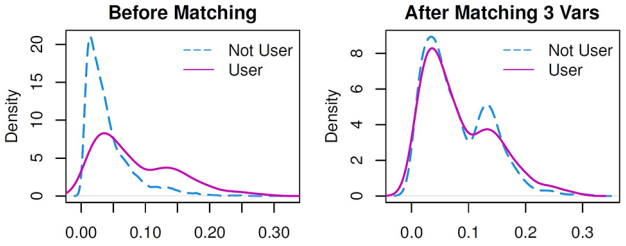


Fig. 1 The coverage plot of the included population before and after propensity score matching based on age, gender, and cigarette smoking

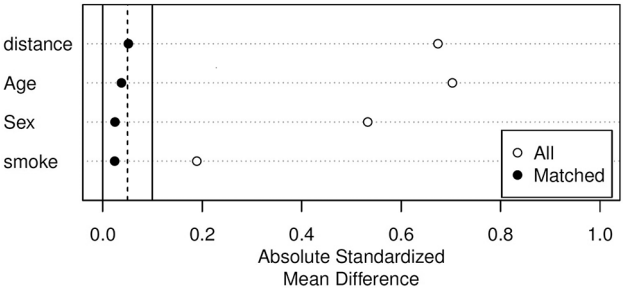


Fig. 2 Covariate balance between waterpipe smokers and non-smokers. The white and black circles represent covariate balance before and propensity score matching, respectively. An absolute standardized mean difference < 0.1 is considered to demonstrate optimal covariate balance

Statistical analysis

The normality of the variables was assessed using histograms. Categorical variables were presented as frequency (percentage) and analyzed using the Chi^2 test. Normally distributed continuous variables were presented as mean ± standard deviation (sd) and analyzed by independent t-test. Continuous variables with skewed distribution were presented as median (interquartile range) and compared using the Mann–Whitney U test. Age, gender, and history of cigarette smoking were used as covariates to generate propensity scores for the population. Propensity score matching (PSM) was then used to match the WP smokers with non-smokers using a 1:4 ratio. The coverage plot and covariate balance of the variables are displayed in Figs. 1 and 2, respectively.

Logistic regression was used to assess the effect of WPS on CAD before and after PSM. To analyze the association between WPS and CAD severity, we excluded patients with GS of 0 from the statistical analysis so that patients with at least some degree of atherosclerosis in their coronary arteries were studied. Due to the skewed distribution of GS, natural logarithm (ln) was used to normalize the GS variable. Linear regression was then employed to analyze the effect of WPS on the GS. Variables with >10% missing were not considered in the analysis. All analysis was performed on R statistical software version 4.1.2 (R Core Team, 2021). We used "MatchIt" and "gtsummary" packages.

Results

Baseline data before and following the PSM

Table 1 presents the baseline characteristics of the participants before PSM. Of the 8699 patients enrolled in the study, 380 were categorized as WP smokers, and others had no history of WPS. Prior to the implementation of PSM, male participants comprised a higher proportion of WP smokers than non-smokers (83.2% vs. 63.2%, $P<0.0001$). Additionally, WP smokers exhibited younger ages (51.9±12.2 vs. 60.5±10.8, $P<0.0001$), higher cigarette consumption (42.1% vs. 32.8%, $P<0.0001$), greater alcohol consumption (49.3% vs. 15.5%, $P<0.0001$), and higher BMI values (28.8±5.1 vs. 29.8±5.2, $P<0.0001$). Regarding medical history, while hypertension (HTN) and chronic kidney disease (CKD) were more prevalent among WP non-smokers, a positive family history of CAD and reporting previous angiography were more prevalent among WP smokers. Concerning the laboratory findings, WP smokers had higher triglyceride and hemoglobin (Hgb) levels.

As shown in Table 2, the implementation of PSM based on age, sex, and CS resulted in the matching of 1520 participants who had no prior history of WPS with 380 participants who were WP users. In this model, WP smokers, compared to non-smokers, still had significantly higher BMI, Hgb, and alcohol consumption. In addition, WP smokers had significantly higher FBS levels compared to non-smokers.

The prevalence and severity of CAD

The results of coronary angiography before PSM revealed that 60.3% (229 out of 380) of WP smokers had been identified as having CAD, compared to 62% (5155 out of 8319) of non-smoker patients. Following the adoption of PSM, it was shown that the prevalence of CAD in WP smokers remained constant, whereas it dropped to 54.1% (822 out of 1520) in non-smokers and demonstrated a significant change ($P<0.05$). Regarding the severity of CAD, before PSM, the average GS for WP smokers and non-smokers was 35.67±41.65 versus 36.62±41.93,

Table 1 Baseline characteristics of the study population before PSM

Characteristics	Waterpipe non-smoker *N* = 8,319	Waterpipe smoker *N* = 380	*P*-value
Age	60.5 ± 10.8	51.9 ± 12.2	**< 0.0001**
Sex (male)	5,259 (63.2%)	316 (83.2%)	**< 0.0001**
BMI	28.8 ± 5.1	29.8 ± 5.2	**< 0.0001**
Medical history			
Hypertension	4,478 (53.8%)	161 (42.4%)	**< 0.0001**
Diabetes mellitus	3,051 (36.7%)	134 (35.3%)	0.587
Hyperlipidemia	5,778 (69.5%)	246 (64.7%)	0.053
Family history of CAD	1,815 (21.8%)	113 (29.7%)	**< 0.0001**
History of previous angiography	6,145 (73.9%)	301 (79.2%)	**0.020**
Atrial fibrillation	365 (4.4%)	10 (2.6%)	0.119
Arrhythmias	495 (6.0%)	14 (3.7%)	0.073
Congestive heart failure	387 (4.7%)	17 (4.5%)	> 0.999
Kidney disease	1,667 (20.1%)	32 (8.4%)	**< 0.0001**
Cigarette smoking	2,726 (32.8%)	160 (42.1%)	**< 0.0001**
Alcohol user	508 (15.5%)	100 (49.3%)	**< 0.0001**
Opium user	1,225 (14.7%)	65 (17.1%)	0.209
Laboratory data			
Fasting blood glucose	105.0 (93.0, 132.0)	105.0 (94.0, 127.0)	0.759
HDL	42.05 ± 11.78	41.74 ± 12.28	0.625
LDL	87.51 ± 31.38	89.66 ± 31.47	0.198
Triglyceride	124.0 (90.0, 172.0)	132.00 (97.0, 196.0)	**< 0.0001**
Total cholesterol	153.44 ± 42.61	157.81 ± 45.53	0.070
Hgb	14.04 ± 1.78	15.00 ± 1.76	**< 0.0001**

BMI: body mass index; CAD: coronary artery disease; PSM: propensity score matching

Table 2 Baseline characteristics of the study population after PSM*

Characteristics	Waterpipe non-smoker *N* = 1520	Waterpipe smoker *N* = 380	*P*-value
Age	52.3 ± 11.8	51.9 ± 12.2	0.509
Sex (male)	1,278 (84.1%)	316 (83.2%)	0.696
BMI	29.0 ± 5.0	29.8 ± 5.2	**0.007**
Medical history			
Hypertension	688 (45.3%)	161 (42.4%)	0.327
Diabetes mellitus	479 (31.5%)	134 (35.3%)	0.177
Hyperlipidemia	1,050 (69.1%)	246 (64.7%)	0.109
Family history of CAD	402 (26.4%)	113 (29.7%)	0.198
History of previous angiography	1,182 (77.8%)	301 (79.2%)	0.580
Atrial fibrillation	57 (3.8%)	10 (2.6%)	0.352
Arrhythmias	74 (4.9%)	14 (3.7%)	0.412
Congestive heart failure	69 (4.5%)	17 (4.5%)	> 0.999
Kidney disease	152 (10.0%)	32 (8.4%)	0.384
Cigarette smoking	622 (40.9%)	160 (42.1%)	0.684
Alcohol user	178 (25.5%)	100 (49.3%)	**< 0.0001**
Opium user	239 (15.7%)	65 (17.1%)	0.531
Laboratory data			
Fasting blood glucose	102.0 (91.0, 123.0)	105.0 (94.0, 127.0)	**0.039**
HDL	40.94 ± 12.20	41.74 ± 12.28	0.264
LDL	89.88 ± 33.73	89.66 ± 31.47	0.903
Triglyceride	133.0 (95.0, 190.0)	132.0 (97.0, 196.0)	0.608
Total cholesterol	155.14 ± 44.64	157.81 ± 45.53	0.309
Hgb	14.70 ± 1.75	15.00 ± 1.76	**0.003**

*Propensity score matching was performed for age, sex, and cigarette smoking variables. BMI: body mass index; CAD: coronary artery disease

Table 3 Different outcome variables compared between waterpipe smokers and non-smokers

Outcomes	Effect measure	LLCI 95%	ULCI 95%	*P* value
Before PSM*				
CAD (OR)	0.93	0.76	1.15	0.504
GS in patients with GS > 0 (ß)	1.07	0.92	1.24	0.361
After PSM				
CAD (OR)	1.29	1.04	1.60	**0.021**
GS in patients with GS > 0 (ß)	1.24	1.04	1.48	**0.014**

*Propensity score matching was performed for age, sex, and cigarette smoking variables. CAD = coronary artery disease; GS = Gensini score; LLCI 95%= lower limit of 95% confidence interval; ß= ß coefficient of ln (GS) variable from linear regression; OR = odds ratio; ULCI 95%= upper limit of 95% confidence interval

respectively, and did not demonstrate a statistically significant difference between the two groups ($P > 0.05$). Following the PSM model, the mean GS for WP smokers was 35.67 ± 41.65 compared to a mean GS of 29.86 ± 38.27 for non-smokers, demonstrating a significant difference.

The association between WPS and the prevalence and severity of CAD

Table 3 displays the OR for the prevalence of CAD and the β regression coefficient for the severity of CAD as measured by the GS score, both before and after using the PSM model. The results of logistic regression analysis prior to PSM revealed that WPS was associated with a lower CAD prevalence compared to non-smoker patients, but this association was not statistically significant (OR: 0.93; 95% CI: 0.76–1.15, $P = 0.504$). In contrast, after implementing the PSM model, there was a statistically significant association between WPS and higher CAD prevalence among WP smokers compared to non-smokers (OR: 1.29; 95% CI: 1.04–1.60, $P = 0.021$). In relation to the severity of CAD, the findings from the linear regression analysis indicate that, prior to PSM, there was no statistically significant association between CAD severity and WPS ($P > 0.05$). Following the introduction of the PSM model, it was observed that there was a significant association between WPS and increased severity of CAD ($\beta = 1.24$ and $P < 0.05$).

Discussion

The present study showed that the risk of CAD diagnosis in patients undergoing elective angiography was 29% higher in WP smokers after adjusting for major confounders. Moreover, we demonstrated that WPS was associated with the severity of CAD, as GS was higher in WP smokers than non-smokers.

Research on the link between WPS and CAD has drawn more attention recently. A case-control investigation by Jabbur et al. demonstrated that the risk of newly diagnosed CAD was 1.9 (95% CI: 1.2–2.8) times higher in WP ever-smokers compared to non-smokers [20]. However, upon accounting for potential confounders, the association persisted but did not reach statistical

significance. Similarly, an adjusted comparison between current WP smokers and non-smokers showed no significant differences in the presence of CAD (OR: 0.7, 95% CI: 0.3–1.9) [20].

A more recent meta-analysis by Morovatdar et al. on 1334 WP smokers showed no significant increase in CAD risk smokers compared to non-smokers (OR: 1.18, 95% CI: 0.98–1.38). However, CAD risk in heavy WP smokers (more than 40 to 50 WP years) was two times higher (OR: 2.00, 95% CI: 1.13–2.87) than in light smokers (less than 40 to 50 waterpipe years) [13].

A study by Sibai et al. identified no associations between current WPS and CAD in elective angiography patients. They defined a variable called "waterpipe years" by multiplying the number of smoked WPs daily by the number of years patients smoked [14]. When patients were analyzed based on this variable, the CAD risk was high in patients with 21 to 40 (OR: 1.66, 95% CI: 0.79–3.50), and 41+ (OR: 2.24, 95% CI: 1.00–4.99) WP years [14].

After accounting for potential confounding factors, our analysis demonstrated a statistically significant link between WPS and CAD. The observed disparity in the findings could be attributed to the relatively small sample size of participants in Jabbour et al.'s study ($n=525$) and the restricted number of studies included in the meta-analysis (three studies). Furthermore, the disparity in the study design is an additional element that may have influenced the outcomes. Finally, total lifetime exposure to WP smoke appears to be a more important risk factor for CAD than smoking status (current or past) [14, 20].

Regarding the severity of CAD, Selim et al. showed that the Duke Jeopardy (DJ) score was, on average, 1.2 points higher in WP smokers than in non-smokers [15]. Sibai et al. demonstrated a similar result as the Duke CAD prognostic index score of patients with a smoking history of 41+WPS was higher than non-smokers by 7.8 [14]. In order to study the effect of WPS on CAD severity we investigated patients with GS>0. Our findings showed that the GS was 1.24 points higher in the WP smokers. According to this finding, WPS may exacerbate CAD in patients with some degree of atherosclerotic plaque buildup in the coronaries.

Although the WP exposure dose is imperative to understand the CAD risk, quantifying the exact exposure amount of the patients is difficult. WP is commonly smoked in groups with a practice called "mouthpiece sharing." The amount of smoke that each person inhales might be different from others. Smoking session durations can also differ, ranging from 20 min to more than one hour, with studies estimating that a 20-minute session roughly equals 25 cigarettes [21, 22]. Moreover, the various kinds of tobacco used in WPs have different nicotine and toxic material profiles [23]. Flavored tobaccos, for instance, have been suggested to emit more Polycyclic aromatic hydrocarbons (PAH), particulate matter, carbon monoxide, aldehydes, and heavy metals compared to non-flavored ones [24, 25].

WPS requires a cumbersome apparatus; unlike cigarettes, it can not readily be consumed everywhere [23]. This can limit the exposure dose of many smokers as they can only smoke at home or in WP cafes; however, the emergence of portable, travel-friendly electronic hookahs (E-hookah) can significantly increase the burden of WS in the near future [26].

The exact mechanism behind the atherosclerotic effects of WS is unknown; however, it can primarily be attributed to particulate matter, PAH, oxidizing agents, and nicotine [27]. PAH, which is found in higher concentrations in WP smoke compared to cigarettes, has been associated with increased systolic and diastolic blood pressure levels [28–30]. In addition, PAH has been linked with increased oxidative stress and inflammation, the pathophysiological mechanisms mainly involved in atherosclerosis [31]. WPS has been shown to cause a reduction in heart rate variability (HRV), an index of the autonomic nervous system function [32]. Reduced HRV, indicating sympathetic dominance, is a well-known risk factor for cardiovascular disease, and it is also associated with an increase in inflammatory and oxidative stress markers, which are common pathophysiological mechanisms in CVDs [33, 34]. Nicotine has been proposed to exert its effect via increasing catecholamine levels. Catecholamines increase blood pressure and heart rate levels, creating an adverse hemodynamic effect contributing to atherosclerosis [27].

Limitations

Along with the large sample size of our study, which is its main strength compared to previous ones, our study has some limitations that should be mentioned. Initially, it should be noted that the assessment of exposure to WP was reliant on subjective measures, and the precise dosage of WP could not be determined, as the utilization of quantitative techniques, such as measuring serum nicotine levels, is not feasible for evaluating long-term effects because it can only show recent exposure. Therefore, until a reliable, objective measurement is developed, self-reporting WPS status, despite its drawbacks, is our only option. Second, our findings cannot establish a dose-response relationship between WPS and CAD due to information gaps regarding the exact dose of WPS, including its frequency, duration, and cessation time. Third, we could not distinguish between occasional and daily WP smokers in our study due to the information gap. Fourth, given the study's design, any causal association should be interpreted cautiously. Finally, our study population included patients who underwent coronary

angiography; thus, our findings may not apply to the general population.

Conclusion

In conclusion, the present study showed a positive association between WPS and the presence and severity of CAD in patients undergoing coronary angiography. The risk of CAD was 29% higher in WP smokers than non-smokers. The GS was also higher in the WP smoker group, suggesting that WPS may cause more severe atherosclerosis. Additional investigation is necessary to comprehensively elucidate the inconsistencies observed in prior research and ascertain the precise underlying mechanisms responsible for this association.

Abbreviations

BMI	Body mass index
CABG	Coronary artery bypass-graft
CAD	Coronary artery disease
CKD	Chronic kidney disease
CS	Cigarette smoking
CO	Carbon monoxide
CVD	Cardiovascular diseases
CI	Confidence interval
DJ	Duke Jeopardy
FBS	Fasting blood glucose
GS	Gensini score GS
HDL	High-density lipoprotein
HTN	Hypertension
Hgb	Hemoglobin
LDL	Low-density lipoprotein
OR	Odds ratio
PAH	Polycyclic aromatic hydrocarbons
PCI	Percutaneous coronary intervention
PSM	Propensity score matching
WP	Waterpipe
WPS	Waterpipe smoking
HRV	Heart rate variability

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Author contributions

F.M and S.N wrote the main manuscript. M.Y and F.M designed the study. Z.K, and A.J did the statistical analysis. S.N, Z.K, and B.A prepared the figures and tables. B.A and A.S revised the manuscript. S.Y helped in conducting the study. A.VF supervised the project.

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Data availability

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The ethics committee of Tehran University of Medical Sciences agreed to the study's design (Ethics CODE: IR.TUMS.THC.REC.1400.004). All patients provided written informed consent at the start of the study.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

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