EURASIAN JOURNAL OF EMERGENCY MEDICINE
Original Article
DOI: 10.4274/eajem.galenos.2024.79026
Eurasian J Emerg Med. 2024;23(3): 185-9

Evaluation of Hookah Smoking-Induced Hemodynamic Changes According to the PVI

Mehmet Ali Özbek[1], Gülşah Çıkrıkçı Işık[2], Şeref Kerem Çorbacıoğlu[2], Yunsur Çevik[2]

[1]Şırnak Public Hospital, Clinic of Emergency Medicine, Şırnak, Turkey
[2]University of Health Sciences Turkey, Ankara Atatürk Sanatoryum Training and Research Hospital, Clinic of Emergency Medicine, Ankara, Turkey

Abstract

Aim: Hookah smoking is increasing all over the world, and it can cause many health problems that are rarely known to be experienced by smokers. The aim of this study was to evaluate the acute impact of hookah smoking on hemodynamic parameters using perfusion index (PI) and pleth variability index (PVI).

Materials and Methods: This prospective observational study was conducted in a hookah cafe with 84 participants. Systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse, oxygen saturation (SpO2), carboxyhemoglobin (COHb) PI, and PVI values of the participants were measured immediately before and 30 min after hookah smoking.

Results: Sixty-three (75%) participants were male, and the median age was 26 [interquartile range (IQR) 22-29]. There were no significant changes in SBP, DBP, pulse, and SpO2 following hookah smoking. There was a 2% (IQR 2-3) increase in COHb level ($p<0.001$) and a 1.4% (IQR 0.6-0.4) decrease in PI value ($p=0.02$) with hookah smoking. No difference was detected in PVI value with hookah smoking ($p=0.3$).

Conclusion: Hookah smoking caused a significant decrease in the PI and an increase in COHb levels. Conversely, PVI did not change with hookah smoking. It should be known that hookah is a harmful, not innocent, tobacco product that can cause many hemodynamic changes.

Keywords: Hookah, carbon monoxide, carboxyhemoglobin, PVI, perfusion index

Introduction

Hookah, a kind of smoked tobacco product, has been used widely, especially in Middle Eastern countries since the 16th century; became very popular among young people all over the world after the 1990s (1). Studies have demonstrated that hookah smokers have little knowledge about the impact of hookah on health, and even many of them have false beliefs that hookah smoking is less harmful than cigarette smoking (2,3). However, unlike cigarettes, using charcoal as a heating source in hookah causes hookah smokers to inhale charcoal combustion products in addition to products originating from the tobacco mixture (4). It was found that a single session of hookah smoking contains many times more carbon monoxide (CO), nicotine, and carcinogenic polycyclic aromatic hydrocarbons (PAH) than the smoke of a single cigarette (4). Exposure to such toxins is associated with numerous disorders, such as cancers, respiratory illnesses, cardiovascular disorders, but there are also acute effects of hookah smoking on vital parameters (5). Increased blood pressure and heart rate and decreased perfusion index (PI) were some of the vital changes observed due to hookah smoking (6,7).

Pleth variability index (PVI) is a non-invasive, dynamic measurement that is calculated by measuring changes in PI during a complete respiratory cycle (8). PVI is affected by physiological factors, such as circulating blood volume, vascular tonus, and intrathoracic pressure changes. Many of the studies in the literature focused on the relationship between PVI and the volume status of patients (8,9). However, some studies have evaluated changes in vascular tonus and hemodynamic parameters with PVI under different clinical conditions in normovolemic patients (10,11).

Corresponding Author: Gülşah Çıkrıkçı Işık MD, University of Health Sciences Turkey, Ankara Atatürk Sanatoryum Training and Research Hospital, Clinic of Emergency Medicine, Ankara, Turkey
Phone: +90 505 587 34 36 **E-mail:** gulsah8676@gmail.com **ORCID ID:** orcid.org/0000-0002-6067-7051

Cite this article as: Özbek MA, Çıkrıkçı Işık G, Çorbacıoğlu ŞK, Çevik Y. Evaluation of Hookah Smoking-Induced Hemodynamic Changes According to the PVI. Eurasian J Emerg Med. 2024;23(3): 185-9.

Received: 27.05.2024
Accepted: 13.09.2024

The aim of this study was to evaluate the acute impact of hookah smoking on hemodynamic parameters using PI and PVI.

Materials and Methods

This was a prospective observational study conducted in a hookah cafe that was operating in accordance with the relevant legal regulations (according to relevant regulations, smoking of tobacco products must be done in open area). This study was conducted in accordance with the Ankara Keçiören Training and Research Hospital Clinical Research Ethics Committee (decision number: 2012-KAEK-15/1785, date: 26.12.2018). Participants were not encouraged to smoke hookah in any way. People who had already come to the relevant cafe to smoke hookah were included in the study. Participation was voluntarily. Written informed consent was obtained from the participants.

Study population: Volunteers aged between 18 and 40 were included in the study. Pregnant and lactating women, persons with known chronic comorbid diseases, and volunteers with a hookah smoking period of less than 30 min were excluded.

Study process: First, the participants rested for 5 min in the sitting position. At the end of the 5th min blood pressure was measured from the left arm (Omron M3 Comfort Hem-7134-E®); simultaneously pulse, oxygen saturation (SO2), carboxyhemoglobin (COHb), PI, and PVI were measured from the 4th digit of the right arm with a non-invasive pulse oximeter (Masimo Root Rainbow Set®). Measured values and demographic variables of the participants were recorded on the study forms.

Participants were advised not to smoke any other tobacco product and not to drink beverages containing alcohol while smoking hookah. Alcohol-free beverages were freely consumed. The researchers did not intervene in the hookah smoking style, inhalation frequency, and inhalation duration of the participants. At the end of 30 min hookah smoking, all the parameters mentioned above were measured again using the same technique.

Sample size estimation: The sample size of the study was calculated using G*Power v3.1.9.7 software. The calculation was performed using Cohen's medium effect size. To determine the differences in PI and PVI among hookah smokers, a medium effect size (d=0.5), 1% type I error, and 95% power two-tailed calculation was performed. The sample size was calculated as 75. To prevent protocol violations, it was decided to include 84 participants in the study.

Statistical Analysis

Statistical analyses of the study were performed using SPSS for Windows 20. The distribution of normality was tested with Kolmogorov-Smirnov test, and continuous variables were defined as median and interquartile range (IQR). Categorical variables were defined as case numbers and percentages. Analyses between categorical variables were tested using the chi-square test, and analyses between dependent continuous variables were tested using the Wilcoxon signed-rank test. P value <0.05 considered statistically significant.

Results

Among the 84 participants, 21 were women, and the median age of all participants was 26 (IQR 22-29). Nearly three-fourths of the participants were cigarette smokers. The demographic data of the participants are summarized in Table 1.

There was no difference in systolic blood pressure, diastolic blood pressure, pulse rate, and SpO2 between before and after hookah smoking. On the other hand, there was a significant increase in COHb levels in the participants after hookah smoking (p<0.001).

There was a significant decrease in the PI after hookah smoking [5.7 (IQR 2.8-7.8) and 4.3 (IQR 2.2-7.4), before and after hookah smoking, respectively, p=0.02]. There was also a decrease in the PVI value of the patient with hookah smoking, but this difference was not statistically significant [27 (IQR 21-32) and 24 (IQR 19-31), before and after hookah smoking respectively, p=0.3]. Changes in the vital parameters with hookah smoking are summarized in Table 2.

Differences among basal (before hookah smoking) COHb levels and PIs of participants with and without cigarette smoking habits were evaluated. The median COHb level was significantly high in cigarette smokers [5 (IQR 3-7) and 2 (0-3.25), cigarette smokers and non-smokers respectively, p<0.001]. Conversely, there were no significant differences among the basal PI, basal PVI, delta (difference between before and after hookah smoking) PI, and delta PVI values of the participants with and without cigarette smoking habits. Data were summarized in Table 3.

Table 1. General characteristics of the participants

Variable	
Gender n (%) Male Female	 63 (75) 21 (25)
Age (year) (median IQR 25-75)	26 (22-29)
Height (cm) (median IQR 25-75)	175 (170-180)
Weight (kg) (median IQR 25-75)	76 (67.25-85)
Cigarette smoking n (%) Yes No	 62 (73.8) 22 (26.2)

IQR: Interquartile range

Table 2. Changes in vital parameters due to hookah smoking			
Parameter	**Before hookah smoking**	**After hookah smoking**	**p value**
Systolic blood pressure (mmHg)	119 (107.75-127.75)	117 (106-126.75)	0.1
Diastolic blood pressure (mmHg)	77.50 (71.25-85.75)	78 (70-86)	0.7
Pulse rate (beat/minute)	87.50 (80.25-97)	90.50 (78.50-98)	0.5
Oxygen saturation (%)	97 (96-97)	97 (96-97)	0.1
Carboxy hemoglobin level (%)	4 (3-6)	6 (5-9)	**<0.001**
Perfusion index (%)	5.7 (2.8-7.8)	4.3 (2.2-7.4)	**0.02**
Pleth variability index (%)	27 (21-32.5)	24 (19-31)	0.3
*All the data were given as median and IQR 25-75, IQR: Interquartile range			

Table 3. Difference among carboxyhemoglobin level and perfusion parameters between participant with and without cigarette smoking habit			
Parameter	**Cigarette smokers**	**Cigarette non-smokers**	**p value**
Carboxy hemoglobin level (%)	5 (3-7)	2 (0-3.25)	**<0.001**
Basal** perfusion index (%)	5.8 (2.8-8.1)	5.4 (2.6-7.1)	0.5
Delta*** perfusion index (%)	27 (20.75-33.25)	26 (21.5-29)	0.6
Basal** pleth variability index (%)	-0.4 (-3.5-1.8)	-1.5 (-2.2--0.2)	0.3
Delta*** pleth variability index (%)	-2.5 (-8-5.2)	-0.5 (-7.2-5.2)	0.4
*All the data given as median and IQR 25-75 **Basal means the value, before starting to hookah smoking ***Delta means the difference between the measurement done before and after hookah smoking			

Discussion

This study, in which the impact of single session hookah smoking on vascular tonus and hemodynamic parameters using PVI was evaluated, had two main results. First, we demonstrated that hookah smoking causes a decrease in the PI. Second, there was a decrease in PVI with hookah smoking, but this was not statistically significant.

Hookah, which may seem like an innocent habit by smokers, is a tobacco product with serious side effects on health in the acute and chronic periods. To the best of our knowledge, the first study on the impact of hookah smoking on vital parameters was conducted in Jordan in 1999 (12). Shafagoj et al. (12) demonstrated increases in heart rate and systolic and diastolic blood pressure with hookah smoking in otherwise healthy 18 hookah smokers. Similar results have been observed in many studies (13-15). Nicotine by increasing sympathetic activity is thought to be the mechanism responsible for hemodynamic changes in hookah smoking (6). Similar to the literature, despite not being statistically significant, there was an increase in the heart rate and blood pressure of the participants after hookah smoking in our study.

Another harmful effect of hookah smoking is increased carboxyhemoglobulin (COHb) levels (16). Yıldırım et al. (16) showed an increase at COHb level from 0 (IQR 0-6) to 22 (IQR 6-44) with 30 min hookah smoking. In another study, researchers compared the end-tidal carbon monoxide (eCO) levels of cigarette smokers (5 cigarettes in 30 minutes), passive cigarette smokers, hookah smokers (for 30 minutes), and passive hookah smokers and showed that the mean increases at eCO levels were 9.4+/4.6 ($p<0.005$), 3.5+/2.5 ($p<0.05$), 57.9+/27.4 ($p<0.005$), and 13.3+/4.6 ($p=0.03$), respectively (17). These studies show us that CO production due to hookah smoking is many times greater than cigarette smoking; and hookah smoking may lead to subclinical CO poisoning in hookah smokers. In a study that analyzed the content of the mainstream smoke of hookah, it was demonstrated that a single hookah smoking session contains 143 mg CO; which was 12.6 mg in average in a single cigarette (18). Likewise, there was an increase in the COHb levels of the participants after hookah smoking in our study. In addition, basal COHb levels were higher in participants who smoked cigarette.

When the impact of hookah smoking on the PI was researched, there was only one study on this subject in the literature; and according to the best of our knowledge, our study is the first to investigate the impact of hookah smoking on PVI. Martinasek et al. (7) demonstrated that the mean PI decreased from 3.1% to 2.7% ($p=0.002$) after visiting a hookah lounge. That was the only study about the effect of hookah on PI; but it had an important limitation. Measurements were performed while entering and

leaving of a hookah lounge, and the time spent in there varied between 32 and 314 min. Our results are similar to those of this study. The reason for the decrease in PI with hookah smoking might be due to the increase in vascular tone. Rezk-Hanna et al. (19) demonstrated that hookah smoking led to a decrease in skin blood flow by 23%, as a consequence of the acute vasoconstrictor effect of nicotine.

PVI is a parameter derived from the PI based on the difference between the minimum and maximum PI values over a sufficient period of time (20). Therefore, in the case of a decrease in the PI, an increase in PVI is predicted. However, despite the decrease in the PI, no significant change in PVI was observed in our study. This finding might be due to other variables that may affect PVI indirectly, such as respiratory variations, arterial and venous distensibility, local skin microcirculation, and sympathetic nervous system activity (21). Hookah smoke contains many different chemicals like tar, nicotine, PAH, and CO, and these chemicals affect these variables in different ways; nicotine causes vasoconstriction on the skin, and CO causes vasodilation (18). Therefore, the cumulative effect of chemicals may have prevented the expected unidirectional change in PVI.

Study Limitations

The most important limitation of this study was that it did not standardize the hookah smoking style (smoke inhalation frequency and duration) of the participants. In addition, according to relevant legal regulations, consumption of tobacco products may only be open air. It is possible that being outdoors reduced the amount of smoke inhaled. Although participants were warned to not smoke any other tobacco product during the study period, we did not know the amount of tobacco products they were exposed to or consumed in the immediate past. However, given the very limited data on this subject, we believe that our study is valuable.

Conclusion

This study demonstrated that hookah smoking caused a significant decrease in the PI and an increase in COHb levels. Conversely, PVI did not change with hookah smoking. It should be known that hookah is a harmful, not innocent, tobacco product that can cause many hemodynamic changes.

Ethics

Ethics Committee Approval: This study was conducted in accordance with the Ankara Keçiören Training and Research Hospital Clinical Research Ethics Committee (decision number: 2012-KAEK-15/1785, date: 26.12.2018).

Informed Consent: Written informed consent was obtained from the participants.

Authorship Contributions

Surgical and Medical Practices: M.A.Ö., Concept: M.A.Ö., Y.Ç., Design: M.A.Ö., Ş.K.Ç., Data Collection or Processing: M.A.Ö., G.Ç.I., Analysis or Interpretation: M.A.Ö., G.Ç.I., Y.Ç., Ş.K.Ç., Literature Search: M.A.Ö., G.Ç.I., Writing: M.A.Ö., G.Ç.I., Y.Ç., Ş.K.Ç.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Lim BL, Lim GH, Seow E. Case of carbon monoxide poisoning after smoking shisha. Int J Emerg Med. 2009;2:121-2.
2. La Fauci G, Weiser G, Steiner IP, Shavit I. Carbon monoxide poisoning in narghile (water pipe) tobacco smokers. CJEM. 2012;14:57-9.
3. Asfar T, Ward KD, Eissenberg T, Maziak W. Comparison of patterns of use, beliefs, and attitudes related to waterpipe between beginning and established smokers. BMC Public Health. 2005;5:19.
4. Al Rashidi M, Shihadeh A, Saliba NA. Volatile aldehydes in the mainstream smoke of the narghile waterpipe. Food Chem Toxicol. 2008;46:3546-9.
5. Akl EA, Gaddam S, Gunukula SK, Honeine R, Jaoude PA, Irani J. The effects of waterpipe tobacco smoking on health outcomes: a systematic review. Int J Epidemiol. 2010;39:834-57.
6. Hakim F, Hellou E, Goldbart A, Katz R, Bentur Y, Bentur L. The acute effects of water-pipe smoking on the cardiorespiratory system. Chest. 2011;139:775-81.
7. Martinasek MP, Calvanese AV, Lipski BK. A Naturalistic Study of Carbon Monoxide, Heart Rate, Oxygen Saturation, and Perfusion Index in Hookah Lounge Patrons. Respir Care. 2021;66:269-74.
8. Demirci OL, Çıkrıkçı Işık G, Çorbacıoğlu ŞK, Çevik Y. Comparing Pleth variability index (PVI) variation induced by passive leg raising and Trendelenburg position in healthy volunteers. Am J Emerg Med. 2020;38:278-81.
9. Emektar E, Dagar S, Uzunosmanoğlu H, Çıkrıkçı Işık G, Çorbacıoğlu ŞK, Çevik Y. The role of perfusion index and plethysmography variability index for predicting dehydration severity in patients with acute gastroenteritis. Hong Kong J Emerg Med. 2022;29:78-83.
10. Tokue A, Saito H, Suzue A, Kadoi Y, Saito S. [Time course changes in perfusion index and pleth variability index during electroconvulsive therapy]. Masui. 2011;60:929-35.
11. Kim YJ, Seo JH, Lee HC, Kim HS. Pleth variability index during preoxygenation could predict anesthesia-induced hypotension: A prospective, observational study. J Clin Anesth. 2023;90:111236.
12. Shafagoj YA, Mohammed FI. Levels of maximum end-expiratory carbon monoxide and certain cardiovascular parameters following hubble-bubble smoking. Saudi Med J. 2002;23:953-8.
13. Shaikh RB, Vijayaraghavan N, Sulaiman AS, Kazi S, Shafi MS. The acute effects of Waterpipe smoking on the cardiovascular and respiratory systems. J Prev Med Hyg. 2008;49:101-7.
14. Kadhum M, Jaffery A, Haq A, Bacon J, Madden B. Measuring the acute cardiovascular effects of shisha smoking: a cross-sectional study. JRSM Open. 2014;5:2054270414531127.

15. Layoun N, Saleh N, Barbour B, Awada S, Rachidi S, Al-Hajje A, et al. Waterpipe effects on pulmonary function and cardiovascular indices: a comparison to cigarette smoking in real life situation. Inhal Toxicol. 2014;26:620-7.
16. Yıldırım F, Çevik Y, Emektar E, Çorbacıoğlu ŞK, Katırcı Y. Evaluating ECG and carboxyhemoglobin changes due to smoking narghile. Inhal Toxicol. 2016;28:546-9.
17. Akhter S, Ali Warraich U, Rizvi N, Idrees N, Zaina F. Comparison of end tidal carbon monoxide (eCO) levels in shisha (water pipe) and cigarette smokers. Tob Induc Dis. 2014;12:10.
18. Shihadeh A, Saleh R. Polycyclic aromatic hydrocarbons, carbon monoxide, "tar", and nicotine in the mainstream smoke aerosol of the narghile water pipe. Food Chem Toxicol. 2005;43:655-61.
19. Rezk-Hanna M, Nelson MD, Rader F, Benowitz NL, Rosenberry R, Chang LC, et al. Peripheral Blood Flow Changes to Cutaneous and Muscular Beds in Response to Acute Hookah Smoking. Am J Cardiol. 2020;125:1725-31.
20. Sato M, Kunisawa T, Kurosawa A, Sasakawa T. Pulse oximetry-derived pleth variability index can predict dexmedetomidine-induced changes in blood pressure in spontaneously breathing patients. J Clin Anesth. 2016;34:318-24.
21. Monnet X, Guérin L, Jozwiak M, Bataille A, Julien F, Richard C, et al. Pleth variability index is a weak predictor of fluid responsiveness in patients receiving norepinephrine. Br J Anaesth. 2013;110:207-13.