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Relationship between platelet indices and lipid status in chronic hookah consumption

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Abstract: Hookah smoking is a growing trend, both in the world and in Bosnia and Herzegovina. The aim of this study was to determine the value of platelet indices in hookah smokers and find out associations with lipid profile. Cross-sectional study included 60 students (30 chronic hookah smokers and 30 non-smokers). The complete blood count (erythrocytes, leukocytes, platelets, hemoglobin, hematocrit, erythrocyte and platelet indices), lipid parameters (total cholesterol, triglycerides, LDL-C, HDL-C) were determined. The platelet count, mean platelet volume and MPV/Platelets ratio were statistically significantly higher in chronic hookah consumers in the student population than in the control group ($p < 0.001$). In addition, platelet count was in significant positive correlation with values of total cholesterol, LDL-C and negative correlation with value of HDL-C, while there was a significant negative correlation between mean platelets volume, and MPV/Platelets ratio with HDL cholesterol levels in chronic hookah smokers ($p < 0.05$). These findings suggest that chronic hookah consumption could be associated with the development of atherosclerotic changes in blood vessels, which could lead to the development of long-term consequences on the cardiovascular system's function.

INTRODUCTION

The hookah, also known as the shisha, nargile, is a tobacco, long, flexible pipe that draws smoke through the water contained in the vessel from which it comes out (Bou Fakhreddine et al.,2014). It is estimated that around 1.1 billion people use hookah, which is the most popular smoking product, along with cigarettes (Badran et al., 2020). The prevalence of hookah smoking among students is extremely high in the Eastern Mediterranean countries (> 30%), followed by over 20% in the Czech Republic, Estonia, Latvia, and Jordan, over 10% in Hungary, Poland, Slovakia, and Ukraine (Jawad et al., 2015). Hookah users are exposed to many of the same toxic compounds as cigarette users, but at dramatically higher levels, leading to more serious negative health effects. Data from previous studies have shown that hookah smoke contains 7 carcinogens, 39 central nervous system depressants, and 31 respiratory irritants. (Elsayed et al., 2016). Hookah smoking leads to

significant acute disorders o of the cardiovascular and respiratory systems, which are characterized by an increase in heart rate by 6-13 beats, systolic pressure by 3-16 mmHg, diastolic pressure 2-14 mmHg, and the number inspiration of 2 per minute (Haddad et al., 2016). There is a lack of research in regards to the connection between hookash smoking and emerging chronic effects on the cardiovascular system.

In that sense, an increasing number of researchers analyze the potential proatherogenic effects of chronic hookah consumption in order to prevent the development of other diseases of the cardiovascular system, such as acute coronary syndrome.

Atherosclerosis is a multifactorial disease of medium and large arteries, in which there is a focal accumulation of deposits composed of lipids, carbohydrates, blood products, fibrous tissue, and calcium on the inner wall of the arteries. Due to the accumulation of deposits, the wall hardens and loses elasticity. (Rafieian-Kopaei et al., 2014). In the development of initial atherosclerotic changes, the most important event is changes in lipid fractions concentrations with proatherogenic lipids' dominance. Dyslipidemia is a disorder of fat metabolism, which results in a disorder of the concentration of certain lipids (hyperlipidemia, hypercholesterolemia, hyperlipoproteinemia) and is considered the main cause of atherosclerosis and associated diseases such as : cardiovascular disease, ischemic cerebrovascular disease, and peripheral vascular disease. The most important risk factors for atherosclerosis are elevated LDL, decreased HDL. (Nelson, 2013). Platelets, oval or round cells formed by the fragmentation of megakaryocytes in the bone marrow, liver, spleen, and lungs, from where they are released into the bloodstream, play a significant role in the development of atherosclerotic plaque. The most important physiological function of platelets is active participation in all phases of hemostasis, both by physical and chemical processes and by the release and activity of special platelet factors. Also, they have a role in the processes of maintaining endothelial integrity, phagocytosis, detoxification of the organism, and transport of substances (Periayah et al., 2017). In conditions of endothelial dysfunction in atherosclerosis, e.g., plaque rupture, there is a reduced synthesis of platelet aggregation inhibitors, which with endothelial damage caused by mechanical, chemical, immune mechanisms lead to platelet interaction with endothelium and subendothelial structures and consequent activation, adherence and platelet aggregation (Schäfer and Bauersachs 2008).

The aim of this work is to assess the hazardous effect of chronic smoking tobacco by hookah on lipid profile and platelet indices, as the main factors in the development of atherosclerotic disorders.

EXPERIMENTAL

Subjects

The cross-sectional study included 60 consecutive participants from September 2019 to May 2020 at the Medical Faculty of the University of Sarajevo. The study included 60 students of the University of Sarajevo divided into two groups: 30 chronic hookah consumers of both sexes, average age 24 years, and 30 healthy participants, average age 25 years, who never consumed hookah. Criteria for inclusion in the study were: respondents who consumed a hookah for at least a year at least 2 times a week, respondents who voluntarily agreed to participate in the study, a properly completed questionnaire, respondents aged 18-30, students of the University of Sarajevo. Exclusion criteria were: participants who did not complete the questionnaire correctly, participants older than 30 years, participants who consumed hookahs for less than a year, participants who smoked cigarettes, participants who had an acute or chronic illness or used therapy or supplements, which may affect the values of the examined parameters. The study was carried out in accordance to Declaration of Helsinki, as revised in 2000. Written informed consent for inclusion in the study was obtained from all participants.

Sample analysis

After taking the medical interview and physical examination, the subjects had their blood taken for laboratory tests by puncturing the cubital vein. Serum was extracted from blood samples after coagulation and centrifugation for 10 minutes at 4000 rpm and stored until the required results were obtained.

Blood samples were taken from all participants by cubital vein puncture for the following laboratory analyzes: complete blood count (erythrocytes, leukocytes, platelets, differential blood count, erythrocyte indices) and lipid profile (total cholesterol, triglycerides, LDL-C, HDL -C).

Platelet count, mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) were determined using an automatic Beckman Coulter STKS Hematology Analyzer. Based on the obtained values, the MPV /platelets ratio (MPV/Plt) is calculated.

Lipid profile

Total cholesterol, HDL-cholesterol (high-density lipoprotein cholesterol), and serum triglycerides were determined by standard enzyme methods on an Olympus 2700 analyzer (Beckman Coulter, USA). The results were read automatically on the instrument.

Principle of estimation total cholesterol.

The recommended method for determining total cholesterol is the photometric method after hydrolysis and cholesterol extraction. Enzymatic determination of cholesterol concentration is specific and sensitive. The reaction principle is as follows: cholesterol esterase catalyzes the hydrolysis of cholesterol esters to free cholesterol and free fatty acid. In the presence of cholesterol oxidase, cholesterol is oxidized to cholest-4-en-3-one to form hydrogen peroxide. Phenol and 4-aminoantipyrine with hydrogen peroxide in the presence of peroxidase give the red colored product quinonymine. The intensity of staining is directly proportional to the concentration of total cholesterol in the sample.

Principle of estimation HDL. The recommended method for determining HDL-cholesterol is

ultracentrifugation and precipitation with heparin and $MnCl_2$ and the determination of cholesterol in the supernatant. The determination of HDL-cholesterol is based on the separation of HDL from lipoproteins containing apolipoprotein B (LDL and VLDL) by ultracentrifuge, electrophoresis, or specific precipitation with polyanions and divalent cations. The concentration of HDL-cholesterol is determined by one of the methods for determining total cholesterol. LDL - (low-density lipoproteins) and VLDL - cholesterol (very low-density lipoproteins) are precipitated by the addition of dextran sulfate solution and serum magnesium chloride. Negatively charged groups on the polyanion, which react with positively charged groups of lipoprotein molecules, are probably significant for the reaction. The present divalent cations accelerate the formation of insoluble LDL- and VLDL-cholesterol complexes. Insoluble complexes sediment by centrifugation due to higher density and HDL-cholesterol remains in the supernatant, determined by one of the methods for determining total cholesterol.

Principle of estimation triglycerides.

The recommended method for determining triglycerides is the photometric method after extraction, saponification, and glycerol oxidation. Methods for determining triglyceride concentration are based on the measurement of released glycerol. Reaction principle: the resulting glycerol-1-phosphate is oxidized to dihydroxyacetone phosphate in the presence of glycerol-1-phosphate dehydrogenase and NAD. The resulting reduced coenzyme, NADH, reduces the color 2- (p-iodophenyl) -3-p-nitrophenyl-5-phenyl tetrazolium chloride (INT) to a red colored formazan whose concentration is measured photometrically.

The LDL-C concentration was determined by the Friedwald formula, [LDL] = [K] – [HDL] – ([TG] / 5) in which the LDL-C concentration was calculated from the concentration of total cholesterol, HDL-C, and triglycerides.

Statistical analysis

Statistical data processing was done using the computer program Excel (Microsoft Office Excel 2010) and SPSS computer program for statistical analysis (SPSS-Statistical Package for Social Sciences) version 13.0. Data were processed by standard statistical methods and presented in the form of tables and graphs. Shapiro-Wilk test was used to assess the normality of continuous variables' distribution.

The mean value (X) and standard deviation (SD) for continuous independent variables that followed the normal distribution were determined, ie the median and interquartile range for independent continuous variables that did not follow the normal distribution. The Student t-test tested the significance of the difference for the continuous independent variables that followed the normal distribution. In contrast, the Mann - Whitney test tested the significance of the difference for the independent continuous variables that did not follow the normal distribution for independent samples. The correlation coefficient (rho) was determined by the Spearman method. Values of $p < 0.05$ are considered statistically significant.

RESULTS

The characteristics of hookah smokers and control subjects are presented in Table 1. The values of hemoglobin, hematocrit, platelets, MCV, MCH, MCHC, PDW, MPV, PCT, and MPV/Platelets ratio were statistically significantly higher in chronic hookah smokers compared to the control group of subjects ($p<0.001$).

Table 1: Laboratory values of blood count parameters in chronic hookah smokers in relation to the control group

Variables	Hookah smokers (n=30)	Control group (n=30)	p
Leukocytes ($x10^9$)	7.01 ± 1.54	7.27 ± 1.92	0.894
Erythrocytes ($x10^{12}$)	5.1(5.02 – 5.36)	5.25(4.47 - 5.74)	0.099
Hemoglobin (g/L)	164.97(160.25 – 176)	154(133 - 173)	<0.001
Hematocrit (%)	46.82(45.52 – 49.6)	45.46(38.3 - 50.3)	0.021
MCV (fL)	92.09(90.75 – 94.22)	85.98(65.3 - 97.2)	<0.001
MCH (pg)	32.76(32.07 – 33.9)	29.23(26.9 - 30.8)	<0.001
MCHC (g/L)	353.03(345 – 360.25)	337.63(313 - 360)	<0.001
Platelets ($x10^9$)	242.50(197.5 – 288.25)	192.6(173.25 – 211.25)	<0.001
RDW-CV (%)	12.31(11.7 – 12.32)	12.4(11.4 - 13.8)	0.097
PDW	13.8(11.7 - 14.9)	13.64(11.7 - 16.9)	0.233
MPV (fL)	9.45(8.5 - 10.2)	9.078(8.0 - 10.2)	<0.001
PCT (%)	0.160(0.124 - 0.244)	0.157(0.118 - 0.246)	0.324
MPV/Plt	0.038 ± 0.003	0.047 ± 0.002	<0.001

*data presented as mean ± standard deviation (X ± SD) and as median with 25-75 percentile interquartile range;
Mean Corpuscular Volume, MCV; Mean Corpuscular Hemoglobin, MCH; Mean Corpuscular Hemoglobin Concentration, MCHC; Red blood cell Distribution Width, RDW-CV; Platelet Distribution Width, PDW; Mean Platelet Volume, MPV; Plateletcrit, PCT; MPV/Plt, MPV/Platelet ratio; probability, p

Relationship between basal values of lipid status in chronic hookah smokers and control group are presented in Table 2. Chronic hookah smokers had statistically significantly lower HDL-cholesterol values than the control group (p <0.001), while other parameters did not show statistical significance.

Table 2: Lipid status in chronic hookah smokers and control group

Variables	Hookah smokers (n=30)	Control group (n=30)	p
Cholesterol (mmol/L)	3.9 (3.58 - 4.62)	4.2 (3.9 - 4.3)	0.307
Triglycerides (mmol/L)	1.14 (0.78 - 1.43)	0.95 (0.76 - 1.15)	0.211
LDL (mmol/L)	2.38 (2.02 - 2.68)	2.31 (2.05 - 2.65)	0.941
HDL (mmol/L)	1.09 (0.9 - 1.25)	1.28 (1.15 - 1.5)	0.001

*data presented as mean ± standard deviation (X ± SD) and as median with 25-75 percentile interquartile range;
Low-density lipoproteins, LDL; High-density lipoproteins, HDL

The correlation between platelet index values and plasma lipid fractions is shown in Table 3. Platelet counts were positively correlated with total cholesterol (p <0.05),, LDL (p <0.05), and were negatively correlated with HDL values. MPV and MPV /platelet values were significantly negatively correlated with HDL values (p <0.001).

Table 3: Correlation of platelet indices and lipid status in chronic hookah smokers

Variables		Plateletes	PDW (%)	MPV(fL)	PCT(%)	MPV / Plt
Cholesterol (mmol/L)	Rho	0.396*	0,089	0.332	0.126	0.145
Triglycerides (mmol/L)	Rho	0.062	0.075	0.031	0.061	0.083
LDL (mmol/L)	Rho	0.433*	0.122	0.397	0.101	0.146
HDL (mmol/L)	Rho	-0.718**	-0.002	-0.668*	-0.092	-0,553*

Platelet Distribution Width, PDW; Mean Platelet Volume, MPV; Plateletcrit, PCT; Low-density lipoproteins, LDL;
High-density lipoproteins, HDL; Rho – Spearman correlation coefficient ; *p<0.05. **p<0.001

DISCUSSION

The growing trend of hookah smoking has slowly and imperceptibly begun to have its effect on the overall health of people who enjoy it. Smoking with the use of water pipes causes a number of adverse effects on the smallest structures in the human body, such as blood cells, to the multiple destructions of organs and organ systems. Data from numerous studies have shown that smoking classic cigarettes has a significant influence on the promotion of the proatherogenic effect in the body. In case if hookah smoking, data, due to the limited number of studies, are still not significant to explain all the mechanisms involved in the pathogenesis and promotion of atherosclerotic changes.

The pathophysiological mechanism by which the products released during hookah consumption induce atherosclerosis development and the subsequent consequences has not yet been sufficiently elucidated. The main candidate is considered to be the activation of oxidative stress reactions, with consequent endothelial dysfunction. Previous research has shown that the possibility of vasodilation of blood vessels is significantly reduced in hookah smokers compared to cigarette smokers and non-smokers, which shows a direct impact of hookah toxins on the development of endothelial dysfunction. Hookah smokers, active or passive, are exposed to higher doses of toxic particles, such as polycyclic aromatic hydrocarbons and benzene aldehydes, which are important triggers for oxidative stress reactions (Al-Amri et al. 2020).

Increased synthesis of reactive oxygen species, through the process of lipid peroxidation, is associated with changes in the concentration of individual lipid fractions, which can be expected in hookah smokers. In addition to the influence of reactive oxygen species, a potential cause of dyslipidemia is nicotine, one of the components in the taste hookah, which leads to the activation of catecholamines, with consequent lipolysis of adipose tissue triglycerides (Andersson K., Arner P., 2001).

Our results showed that HDL-C values were statistically significantly lower in chronic hookah consumers, while other lipid status parameters did not differ significantly. The present study findings are similar to the finding of

Trupti and co-works who found that mean HDL-C was significantly lower among smokers than control. In contrast to our study, other studies have shown an increase in hookah smokers' proatherogenic lipid fractions (Sami Alkubaisy et al., 2020; Shafique et al., 2012), that suggests that the research results are not consistent. The reason can be sought in the length of hookah consumption, which implies the need for further research that would elucidate all the pathophysiological mechanisms in developing lipid disorders in hookah use.

By analyzing platelet parameters, our results showed that the number of platelets, MPV, MPV / Plt ratio were statistically significantly higher in chronic hookah consumers compared to the control group, which may indicate increased thrombotic activity in proatherogenic processes. Previous studies have shown that the metabolites of nicotine (cotinine), acrolein, and aldehyde, from hookah smoke, affect platelet activation and lead to increased prothrombotic expression, consumption, and producing new platelets, which explains the increase in their number in chronic hookah consumers. (Alarabi et al., 2020). Activated platelets exert their function on the endothelium, where they modulate the inflammatory response and participate in the formation of atherosclerotic changes and later thrombotic complications (Badimón et al., 2009).

Platelet size, shown as MPV, reflects platelet activity and appears to be a useful predictive and prognostic biomarker of cardiovascular events. Increased MPV values correlate with diseases based on the atherosclerotic process, such as acute coronary syndrome and cardiovascular disease. Larger platelets are known to be metabolically, enzymatically, and functionally more active and produce more thromboxane A2, leading to potentially increased thrombogenic activity and promotion of atherosclerosis (Khode et al., 2012).

The MPV / Plt ratio is easily measured biomarker, suitable for routine use to demonstrate increased platelet activity in atherosclerosis. Increased MPV/Plt ratio, in contrast to platelet count and MPV alone, is a better predictor of long-term mortality in many diseases, including ischemic cardiovascular diseases and nonalcoholic fatty liver disease (Slavka et al., 2011).

We found that MPV and MPV / Plt ratio values were significantly negatively correlated with HDL values by analyzing the relationship between platelet parameters and lipid status. According to data from the literature, HDL inhibits several procoagulation and prothrombotic processes. Low HDL-cholesterol levels are an independent predictor of acute thrombus formation (van der Stoep et al., 2014). The inhibitory effect of HDL particles on platelet activity depends on apo E's presence in these lipoproteins, which stimulates the synthesis of nitric oxide (NO) in platelets. Recent studies have established the presence of LRP8 receptors on the platelet surface. It is a receptor for apo E protein, The binding of HDL particles via apo E to this receptor stimulates the production of nitric oxide synthase(iNOS). HDL stimulates endothelial production of nitric oxide and prostacyclin, potent inhibitors of platelet activation (Holtzman et al., 2012), which leads us to conclude that subjects from our study, where HDL is statistically significantly lower in chronic hookah consumers, have a higher chance of thrombotic clot formation and higher cardiovascular risk.

CONCLUSION

Recently, hookah consumption has been identified as an important factor in the progression and outcome of several important disorders. In addition to numerous systemic effects, hookah consumption products lead to the initiation of changes in the wall of blood vessels, which, combined with the disturbance of lipid concentration, promotes the accelerated development of atherosclerotic damage. A better understanding of the mechanisms involved in these disorders will provide additional opportunities to prevent the development of long-term effects on other organ systems. The results of the current study indicate the potential involvement of hookah on platelet function and lipid metabolism. To validate our findings, further major prospective population-based studies are still required.

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Summary/Sažetak

Pušenje nargile je rastući trend, kako u svijetu, tako i u Bosni i Hercegovini. Cilj ovog istraživanja bio je utvrditi vrijednost tromobcitnih indeksa kod pušača nargile i utvrditi povezanost sa lipidnim profilom. U presječnu studiju je uključeno 60 studenata (30 hroničnih pušača nargile i 30 nepušača). Određena je kompletna krvna slika (eritrociti, leukociti, trombociti, hemoglobin, hematokrit, indeksi eritrocita i trombocita), lipidni parametri (ukupni holesterol, trigliceridi, LDL-C, HDL-C). Broj trombocita, srednji volumen trombocita i omjer MPV /trombociti bili su statistički značajno veći kod hroničnih potrošača nargile u odnosu na kontrolnu grupu ($p < 0,001$). Pored toga, broj trombocita bio je u značajnoj pozitivnoj korelaciji sa koncentracijama ukupnog holesterola, LDL-C i negativnoj korelaciji sa koncentracijom HDL-C, dok je postojala značajna negativna korelacija između srednjeg volumena trombocita i odnosa MPV / trombocita sa nivoom HDL holesterola kod hroničnih pušača nargile ($p < 0,05$). Ovi rezultati sugeriraju da bi hronična konzumacija nargile mogla biti povezana s razvojem aterosklerotskih promjena u krvnim žilama, što bi moglo dovesti do razvoja dugoročnih posljedica na funkciju kardiovaskularnog sistema.