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The Effects of Smoking after Meals on Superior Mesenteric Artery and Portal Vein Haemodynamics: A Doppler Ultrasonography Study

Yemekten Sonra İçilen Sigaranın Superior Mezenterik Arter ve Portal Ven Hemodinamiğine Etkisi: Doppler Ultrasonografi Çalışması

Orhan BABAOĞLU, Banu ALICIOĞLU, Emen SACİT

Trakya University School of Medicine, Department of Radiology, Edirne, Turkey

Abstract

Objective: To assess changes in mesenteric blood flow caused by smoking after meals in healthy young adults.

Subjects and Methods: A total of 50 participants, 22 smokers and 28 non-smokers, were enrolled in the study. Volunteers were divided into three groups: group A, non-smokers (n=28), group B, smokers and smoked postprandially (n=18), group C, smokers but were not allowed to smoke postprandially (n=16). Twelve people participated in both groups B and C. Doppler sonography was performed when the participants were hungry (baseline) and was repeated 30, 60, and 90 minutes after a standard meal. SMA (superior mesenteric artery) and PV (portal vein) flow parameters were measured.

Results: The peak systolic velocity (PSV) of the SMA were significantly higher both baseline and postprandial in group B (p=0.048). The mean vessel diameters and flow volume group averages did not differ based on smoking (F=1.542, p=0.222; F=2.082, p=0.134). Variations in SMA diameter averages in the three groups were different (F=6.406, p<0.001). The mean maximum velocity of the PV and the peak systolic velocity of the SMA were the lowest in group C (p=0.048 and p=0.026).

Conclusion: Both smoking and breaking the habit of postprandial smoking diminish mesenteric flow. *(Marmara Medical Journal 2011;24:187-91)*

Key Words: Mesenteric Artery, Superior, Portal vein, Physiology, Smoking, Ultrasonography, Doppler

Özet

Amaç: Sağlıklı genç erişkinlerde, yemekten sonra içilen sigaranın mezenterik dolaşımdaki oluşturduğu değişikliklerin araştırılmasıdır.

Yöntem: Çalışmaya 28 sigara içmeyen, 22 sigara içen olmak üzere toplam 50 birey katıldı. Üç gruba ayrıldı. Grup A: Sigara içmeyenler (n=28), Grup B: Sigara kullanan ve yemek sonrası da içmelerine izin verilenler (n=18), Grup C: Sigara kullanmakta olan ve yemek sonrası içmelerine izin verilmeyenler (n=16). 12 olgu hem grup B, hem de grup C'ye dahil edildi. Olgulara açlıkta Doppler sonografi yapıldı ve superior mesenterik arter (SMA) ve portal ven (PV) akım parametreleri ölçüldü. Standart bir yemek sonrasında 30, 60 ve 90. dakikalarda ölçümler tekrar edildi.

Bulgular: Grup B'de SMA'in pik sistolik hız (PSH) ortalamaları hem açlık hem postprandial dönemde anlamlı olarak yüksekti (p=0,048). Gruplar arasında damar çap ortalamaları ve akım volümleri arasında sigara kullanımına göre fark yoktu (F=1,542, p=0,222; F=2,082, p=0,134). SMA'daki çap değişiklikleri her üç grupta farklı idi (F=6,406, p<0,001). PV'in maksimum hız ve SMA'nın PSH ortalaması grup C'de en düşüktü (p=0,048 ve p=0,026).

Sonuç: Sigara içmek de yemek sonrası sigara içmeyi bırakmak da mezenterik dolaşımı azaltmaktadır. *(Marmara Üniversitesi Tıp Fakültesi Dergisi 2011;24:187-91)*

Anahtar Kelimeler: Superior, Mezenterik arter, Portal ven, Fizyoloji, Sigara, Doppler, Ultrasonografi

Correspondence to/İletişim: Banu Alıcıoğlu M.D., Trakya University School of Medicine, Department of Radiology, Trakya, Turkey
E-mail: banu_alici@yahoo.com
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Introduction

The sympathetic and parasympathetic components of the autonomic nervous system, which act in opposition, need to be balanced to maintain homeostasis in the body. The function of the gastrointestinal system is based on parasympathetic and enteric nervous system activation, resulting in increases of mesenteric blood flow, peristalsis, and glandular secretion, which are needed for digestion and absorption of food after eating[1].

Smoking increases systolic and diastolic blood pressure via its acute effect and results in vasoconstriction in the arteries and an increase in the heart rate by stimulating the adrenergic system. This effect occurs at the start of smoking and continues for about 15 minutes after smoking. Nicotine and its products stimulate smooth muscle spasm in the arterial wall by reducing the distensibility of the arteries, which leads to a diminution of the arterial compliance. Smoking also inhibits the synthesis of some substances that cause vasodilation secreted by the endothelium, such as nitrous oxide[2-4]. Smoking impairs vascular hemodynamics in the coronary arteries[3], mesentery[5], eye[6], skin[7], and genitalia[8,9]. The fading impact of smoking on compliance in the arteries affects the small muscular arteries and large elastic arteries. The superior mesenteric artery (SMA) is also expected to be affected by smoking as it is in the medium muscular artery group[4,8,10].

In this respect, when a subject smokes after a meal, the parasympathetic and enteric systems will be activated in order to digest and absorb the food; in addition, nicotine will result in sympathetic stimulation of mesenteric flow. In this prospective study, the hemodynamic changes in the SMA and portal vein (PV) were investigated after meals. The blood flow was quantitatively measured by color Doppler ultrasonography (CDUS) among healthy young adults. CDUS is a noninvasive scanning method used for the determination of the type, velocity, and volume of blood flow in the vessels[2,5,11-14].

Subjects and Methods

The experimental protocol was approved by the Local Ethical Committee. Written informed consent was approved and signed by all the study subjects.

The study, carried out between June and November 2007, involved a total of 50 healthy adults, 25 women and 25 men of the hospital staff. The average age was 25.46±4.46 (median: 25, minimum: 19, maximum: 37) among all subjects (n=50), 25.00±4.20 (median: 24, minimum: 19, maximum: 37) among the women (n=25), and 25.92±4.20 (median: 26, minimum: 19, maximum: 35) among the men (n=25). The subjects had no endocrine, cardiovascular, or metabolic diseases, and they did not regularly use drugs.

The number of smokers was 22, 11 women and 11 men, whereas the number of non-smokers was 28, 14 women and 14 men. The smokers were divided into two groups, which were equivalent to each other in terms of age and gender, and there were 12 people common to both groups. Thus, three different study groups were formed (Table I).

Table I. The study groups

Group A (n=28)	Nonsmoker	
Group B (n=18)	Smoker	smoked after the meal
Group C (n=16)	Smoker	not allowed to smoke after the meal

Each participant's Body Mass Index (BMI) was within normal limits (<25 kg/m^2), 21.88 ± 2.67 in the group of non-smokers (Group A) and 21.52± 2.50 in the smoker groups (Groups B and C). The number of cigarettes that the smokers consumed per day was 16.55±10.17 (median: 20, minimum 5, maximum: 50), the average smoking age was 8.05±2.59 (median: 7.5; minimum: 3, maximum: 15) years.

All the individuals were tested after an overnight fast that lasted 8–12 hours and resting for 20–30 minutes in the supine position. They were examined before breakfast at baseline and at 30, 60, and 90 minutes after breakfast for a total of four times. A standard breakfast including 60 grams of carbohydrates, 30 grams of protein, and 30 grams of lipids was given to each person. Group B was asked to smoke 2 cigarettes in 10 minutes just after the meal was finished. Group C was asked not to smoke after the meal. An independent observer supervised the study subjects.

All the Doppler sonographic examinations were performed with the same US device and 3.5 MHz transducer (Sonoline Elegra Siemens) by the same operator in order to avoid interobserver variance. The operator was blinded to the study groups.

The measurements were done of the superior mesenteric artery (SMA) at the first two centimeters from the origin in the sagittal plane. For the portal vein (PV), the confluence of the superior mesenteric and splenic veins was used for the measurements. The diameter of the SMA was measured in grayscale. Doppler signals from the SMA and PV were detected by maintaining the Doppler angle at 60°; the pulse repetition frequency was adjusted in accordance with the velocity of flow. Sample volumes, which involved the possible lumen without contacting the walls, were adapted. Repeated measurements were done on the same segment and the same position with the same angle. The PSV (peak systolic velocity), EDV (end-diastolic velocity), RI (resistive index), diameter, and flow volume values were measured from the flow spectrum automatically (Figures 1,2). The maximum velocity (MV) of the PV was registered for each

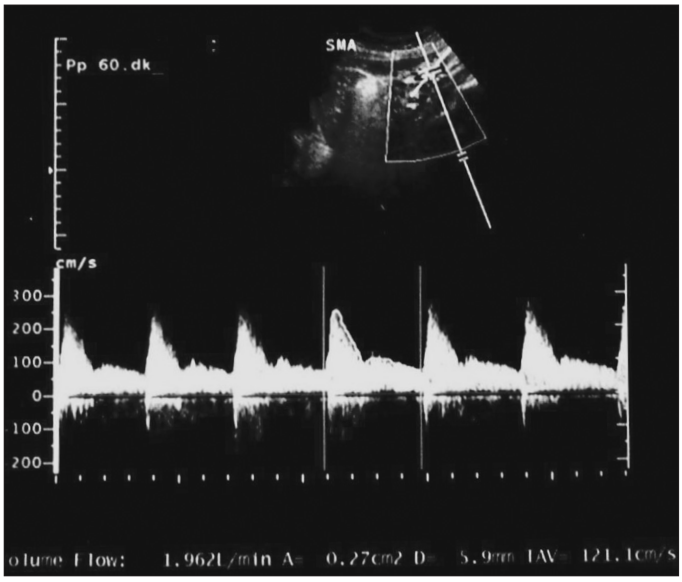

Figure 1. A case from Group A. The flow volume of the SMA at the 60th minute following the meal was measured as 1.962 ml/s. The baseline flow volume of the SMA was 0.696 ml/s (not shown).

period of time together with these processes. The study by Unal et al. (5) was taken as an example for the method of this study but some additional parameters (the flow volume of the SMA and the MV of the PV) were included in order to better clarify the blood-flow changes.

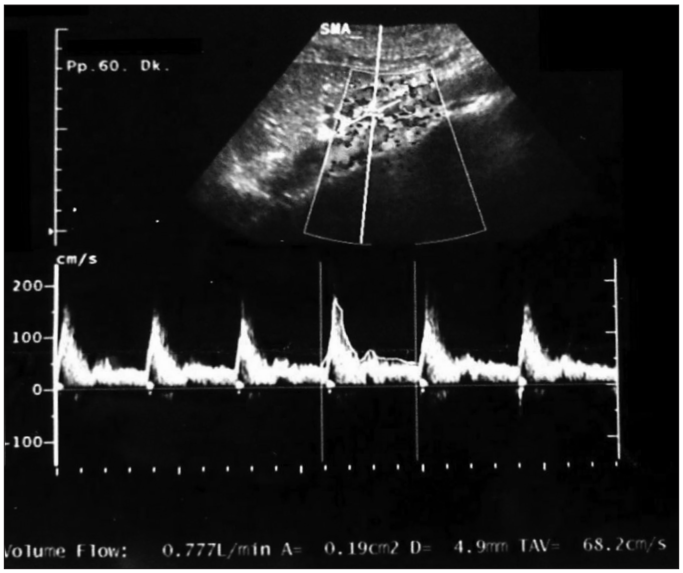

Figure 2. A case from Group B. The baseline flow volume of SMA was 0.393 ml/s (not shown) but it reached 0.777 ml/s at the 60th minute.

The statistical assessment of the study was performed with the STATISTICA AXA 7.1 statistics program. The Kolmogorov-Smirnov Test was used to check normality; repeated measures ANOVA was used for comparison of the groups. F value in ANOVA test is defined as explained variance/unexplained variance. The mean± SD (standard deviation) values are given as descriptive statistics. A p value<0.05 was considered to indicate statistical significance.

Results

The descriptive statistics for baseline and 30, 60, and 90 minute postprandial PSV (mm/s), EDV (mm/s), RI, volume (mL/s), and diameter (mm) of SMA and MV (mm/s) of PV according to the groups are shown in Table II.

The peak systolic velocity of the SMA changed with time to reveal a significant main effect of groups (F=12.058, p<0.001), and the alteration of the PSV of the SMA was in the same direction in each group (F=0.358, p=0.904). Smoking caused a significant difference in the PSVs (F=3.206, p=0.048). The PSV was much lower at baseline and at the 30th, 60th, and 90th minutes in group C than in the other two groups.

Table II. The descriptive statistics of the baseline and postprandial of PSV, EDV, RI, diameter and flow volume of the SMA and maximum velocity (MV) of the portal vein (PV) for all groups.

	Time, min				
	0	30	60	90	p†
PSV (mm/sec)					
Group A	147.63±40.61	181.88±52.79	186.71±46.28	163.93±36.54	<0.001#
Group B	163.37±44.73	190.66±51.02	195.41±58.33	173.52±46.44	0.048‡
Group C	139.41±28.87	157.25±35.00	161.11±33.45	147.62±33.74	
EDV (mm/sec)					
Group A	19.85±7.34	47.47±51.16	35.37±14.15	30.82±12.67	0.515#
Group B	22.15±7.32	42.23±18.14	35.92±13.56	32.65±11.84	0.372‡
Group C	18.80±3.92	30.39±7.56	28.54±8.79	26.48±6.72	
RI					
Group A	0.86±0.03	0.79±0.05	0.81±0.05	0.81±0.06	<0.001#
Group B	0.85±0.04	0.77±0.06	0.81±0.05	0.81±0.04	0.708‡
Group C	0.86±0.02	0.80±0.05	0.82±0.04	0.82±0.04	
Diameter (mm)					
Group A	5.53±0.86	5.76±1.15	5.91±1.06	5.87±1.00	0.007#
Group B	5.41±0.80	5.08±0.85	5.27±0.80	5.38±0.71	0.222‡
Group C	5.68±0.69	5.61±0.78	5.66±0.77	5.73±0.82	
Flow volume (ml/min)					
Group A	598.29±296.99	1116.68±486.98	1199.14±570.96	952.04±353.11	<0.001#
Group B	662.50±283,17	885.67±306.08	1023.00±553.19	867.83±313.12	0.134‡
Group C	588,19±171,80	844.38±275.54	872.19±247.79	800.31±238.10	
PV- MV (mm/sec)					
Group A	26.39±10.72	38.40±16.15	36.41±11.33	32.82±11.67	<0.001#
Group B	26.80±8.90	37.00±14.79	31.85±8.97	30.98±7.82	0.026‡
Group C	20.65±3.85	29.92±6.30	31.05±6.30	26.51±6.57	

†:Repeated measures of ANOVA, p<0.05 is significant

#: Test of within subjects effects, ‡:Test of between subject effects.

The EDV averages did not change with time (F=0.765, p=0.515), and this was similar in all groups (F=0.539, p=0.778). The EDV values of the groups based on smoking did not differ from each other (F=1.005, p=0.372). The baseline and postprandial EDV were close to each other in all three groups. The highest EDV in the SMA occurred at the 30th minute postprandial, and then gradually decreased.

Resistive index values changed with time (F=31.199, p<0.001), in the same direction in each group (F=0.276, p=0.948). The RIs did not differ between the groups based on smoking (F=0.348, p=0.708).

In group A, the flow volume was found to be quite high compared to the other two groups, but the flow volume averages did not show a statistical difference on the basis of smoking (F=2.082, p=0.134).

The diameter of the SMA (mm) changed with time (F=4.153, p=0.007), but the alterations were not all in the same direction (F=6.406, p<0.001). The diameter did not differ between the groups based on smoking (F=1.542, p=0.222). No significant difference among the groups was observed in the baseline and postprandial diameter means at the 30th, 60th, or 90th minutes.

The MV of the portal vein changed with time (F=15.597, p<0.001) in the same way in each group (F=0.536, p=0.780). The maximum velocities differed from each other based on smoking (F=3.888, p=0.026). The maximum velocity baseline values were lower in group C, whereas they were close to each other in groups A and B. The averages were higher in group A than in group B in the postprandial 30th, 60th, and 90th minutes, and they were the lowest in group C.

Discussion

The non-smoker group showed all the physiological hemodynamic alterations in the mesenteric circulation after eating that have been previously described[15-17]. That is, whereas the mesenteric blood flow had moderate to high resistance (characterized by a low diastolic rate and reverse flow component) during starvation, after eating vasodilation and antegrade flow appeared with a low resistance; the mesenteric arterial blood flow eventually reached about 200% of the starvation level. The changes in blood flow started 15 minutes after the intake of food and continued for approximately 90 minutes. However, the hemodynamic alterations were considerably different in smokers (groups B, C). The most relevant difference was observed in alterations of the arterial lumen diameters. The result agrees with Unal's study[5]. In non-smokers, vasodilation progressed from just after the intake of food until the 30th and 60th minutes; then the artery gradually recovered its initial diameter after the 60th minute. On the other hand, in group B, the artery decreased in diameter at the 30th minute and gradually widened to regain its initial diameter. In group C, minimal vasoconstriction was observed at the 30th minute, and then the diameter returned to the basal level gradually. Despite the significant vasoconstriction of the SMA, the resistance in the vascular bed was not increased because of the altered PSV in smokers.

In group B, the SMA diameter and RI decreased and PSV and EDV increased at the 30th minute. This outcome could be explained by a sympathomimetic effect of vasoconstriction and the increase of the velocity in the vessel. The high value of RI indicated that the increase of PSV was higher than the increase of EDV. In group C, postprandial PSV and EDV increased, but RI decreased at the 30th minute; this event was explained by a higher increase of EDV than of PSV.

In group B, EDV (mm/s), RI, flow volume (mL/min) of the SMA and MV of the PV (mm/s) were lower than in the non-smokers. The PSV in group B was significantly higher both before and after eating than in the other groups (*p=0.048*). Since vasoconstriction developed after eating and smoking, the increase in velocity is connected to the sympathomimetic effect caused by smoking. It appears that the Doppler flow parameters at the 30, 60 and 90th minutes after the meal mainly depend on their values at the baseline. For this reason, the presence of chronic damage from nicotine to the vessels could be the cause of the variances of the baseline values. However, only the acute effects of smoking on mesenteric blood flow were investigated in this study. As the number of participants in our study is small and they are all young smokers, it is not possible to draw a conclusion about chronic damage. The basal velocity in group B was unexpectedly high compared to the other smoking group (group C) which is attributed to the small number in the study group. The lack of interobserver variance analysis is also a major weakness of the study, but since the nature of the study is fairly tiring and time-consuming, examination by a second observer was unfortunately impossible in our department. The findings of Unal et al.[5] , that the postprandial PSV and EDV in groups B and C were found to be significantly low (for PSV, *p=0.007* and *0.006*, respectively; for EDV, *p=0.006* and *0.004*, respectively) were unexpected. They also found that the RI was different from our study, growing out of the differences of the PSV and EDV values. The cause of the critical differences between the results of the two studies is thought to result from the usage of different US devices and interobserver variances[18]. The content of the breakfast given to the individuals and the nicotine rate of the cigarettes were chosen identically in our study protocol so that other factors affecting our results could be minimized[4,5]. However, the lack of blood nicotine measurements concurrent with CDUS values of mesenteric circulation is considered a limitation of both studies. In a recent study, the increased vascular resistance was found correlated with higher urinary nicotine and concentration of carbon monoxide in the exhaled air in pregnant women smokers[19].

The quantity of blood flow in the SMA is determined by the current volume in mL/s and is mainly affected by the diameter of the artery and flow velocities. The increase of the flow volume was lower in smokers and, surprisingly, lowest in group C. Mesenteric vascular dilation, which had been expected to develop after eating a meal in the group of smokers, did not occur; instead vasoconstriction developed. As a reason for this, the study raises the possibility that an abstinence syndrome inducing sympathomimetic activity had developed among the individuals who did not smoke in our study although, usually they smoked after breakfast. Unfortunately, we cannot prove this hypothesis because the subjects were not examined clinically for abstinence syndrome. The nicotine withdrawal syndrome is defined as decreases in heart rate and blood pressure during about 3.5 to 18 hours of nicotine abstinence, and an increase in anxious and depressed mood as the length of abstinence increases[17]. We did not find any study investigating the blood flow alterations in

arteries or arterioles after nicotine withdrawal except Sighinolfi et al.[8], who do not support our results; they concluded that after cessation of smoking in patients with erectile dysfunction, penile hemodynamics improved dramatically. Further studies need to be done to reveal the effects of the nicotine withdrawal syndrome on the cardiovascular system.

The impairment of the portal vein's maximum velocity was the other relevant result from group C. The impairment of mesenteric arterial circulation should indirectly decrease portal venous blood flow.

Unal et al.[5] explained the reason for the common belief that 'smoking inhibits getting fat' through prevention of postprandial mesenteric blood flow by the cigarette. Our results agree with this hypothesis and we add that impairment of mesenteric circulation could be the reason for appetite whetting in subjects who are giving up smoking during the acute period, in addition to the effects of nicotine to lessen appetite and prevent excessive eating.

It is known that smoking has a protective effect against chronic inflammatory bowel disease[20-26]. The etiology has not yet been clarified; an experimental study demonstrated that nicotine may correct the imbalance of immune response via neurotransmitter receptors[24-26]. According to our study the vasoconstrictive effect of nicotine on the intestinal mucosa should contribute to reducing the inflammation. However, this hypothesis needs to be proven by future research.

Nevertheless, smoking generally causes harmful hemodynamic effects on the mesenteric circulation. Postprandial smoking could bring about serious problems such as mesenteric infarct in older patients with atherosclerosis and chronic intestinal ischemia whose perfusion had already weakened even in basal conditions.

As a conclusion, both smoking and breaking the habit of postprandial smoking disturbs normal hemodynamic alterations in mesenteric circulation. In the SMA, vasoconstriction occurs instead of vasodilation, and consequently the flow volume decreases. A theory on the protective effect of smoking against celiac disease and ulcerative colitis by the vasoconstrictive effect of nicotine on mesenteric circulation is proposed. However all the smoker patients should be advised, encouraged and assisted to quit.

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