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ORIGINAL ARTICLE

Effects of chronic smoking on the superior mesenteric artery (SMA)

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Abstract

Objective: *Evaluate the effect of chronic smoking on peak systolic velocity (PSV) of the superior mesenteric artery, evaluated with pulsed Doppler, determining whether the longer the snuff consumption, the greater the increase in PSV.* **Material and Methods:** *This is an analytical cross-sectional study, where patients who came to perform abdominal Doppler and had a history of chronic smoking were included. Additional measurements were performed on PSV superior mesenteric artery in 60 patients. Subjects were selected by the non-probability method, taking the total number of patients who presented. Once selected, patients performed pulsed Doppler, and a study and interpretation of these was carried out.* **Results and Discussion:** *We studied 60 patients with a history of chronic smoking between the ages of 25 to 50 years with a median of 38.6 ± 7.1%; chronicity of smoking was measured in years, with a mean of 17.1 ± 8.4 (range 3-37) and PSV was divided into four ranges in cm/sec.* **Conclusions:** *Chronic smoking significantly increases the PSV of the superior mesenteric artery after 21 years of the onset of smoking.* (Gac Med Mex. 2015;151:276-9)

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KEY WORDS: *Chronic smoking. Peak systolic velocity. Superior mesenteric artery.*

Introduction

Tobacco is considered a readily available licit drug at all social levels and strata. When there is dependence on tobacco, smoking is regarded as a disease, which is a public health concern worldwide, since it constitutes a risk factor for six of the eight main causes of death in the world[1]. According to the World Health Organization (WHO), every year, 5.4 million people who have suffered from lung cancer, heart diseases or other tobacco consumption-related diseases die, and if the current situation continues, this figure is estimated to rise to just over 8 millions by the year 2030[2]. In the USA, Canada and Mexico, tobacco consumption causes more deaths than the combination of alcohol, other licit and illicit drugs, suicide, murder and AIDS. Data from developed countries indicate that the consumption of tobacco accounts for one third of heart disease-related deaths and one third of cancer-related deaths; most deaths caused by tobacco result from heart diseases[3]. Smoking is well known to physiologically and pathologically increase the heart rate and sistolic blood pressure, and acute tobacco smoke inhalation has been shown to reduce blood flow to different structures such as the skin, the muscles, the carotid artery, the brain, the liver, the gut and the rectum[4]. According to its effect on smokers, when inhaled by

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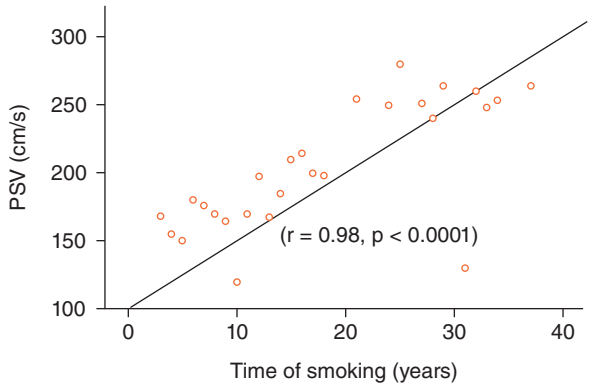

Figure 1. *PSV and time of smoking in 60 chronic smoker patients.*

the mouth, it passes immediately to the lung, and through the pulmonary circulation, to the arterial circulation, thereby reaching the brain very quickly, in 9 or 10 s. Subsequently, it spreads hematogenously to other tissues such as the liver. Ninety percent of the nicotin present in the systemic circulation runs freely in plasma, which facilitates its transportation into the cells and its binding to specific receptors[5]. Immediately after its absorption, nicotin produces an activation of the adrenal glands and, in turn, these produce a bodily stimulation and a sudden glucose discharge, as well as an increase in blood pressure, respiration and heart rate[6]. Furthermore, the habit of cigarrette smoking reduces the diameter and distensibility of medium and large-caliber arteries. The mechanism by which cigarrette smoking exerts this vasoconstriction effect is associated with local or systemic catecholamin release and sympathetic neural stimulation[7]. Patients facing problems associated with damage to the mesenteric artery suffer from chronic abdominal or acute post-prandial pain, abdominal distension, vomiting, abdominal bruit, unspecific weight loss and malabsorption. Therefore, any probable vasoconstrictor effect of smoking on visceral perfusion can produce unwanted results in smoker patients, such as stenotic lesions of these arteries and chronic intestinal ischemia[8].

Color and pulsed Doppler is a screening test for patients with these symptoms that allows, within the diagnostic algorithm, for the physician to be guided to include some future or emergency intervention such as, for example, an angiography to corroborate or exclude some diagnosis, and this way contribute to improve the lives of patients and population suffering from these smoking-related diseases and their effects[9]. Doppler ultrasound is also synonym for non-invasive method for clinical assessment of blood flow velocity, volume and characteristics in different arteries. Splanchnic blood flow has been studied under normal conditions and in different physiological and pathological conditions with Doppler ultrasound. The vasoconstrictor effect of smoking on splanchnic vessels has also been demonstrated[10]. Doppler ultrasonography provides anatomical and functional information on diastolic and systolic activity, as well as on damages at the arterial level. In addition to its demonstrated technical reproducibility, it has the advantage that it allows for diastolic activity to be assessed instantaneously, heartbeat by heartbeat, which in some concrete situations, turns it into a useful method. This technique analyzes blood kinematics, i.e., blood flow speed changes, which are not easy to translate into parameters reflecting pressure or volume changes[11]. SMA blood flow is usually significantly decreased during ingestion. Systolic pressure can be doubled, with a broad systolic peak. In diastole, there is also a significant velocity increase with continuous flow. However, the response to ingestion is variable and can be normal, very increased, and sometimes even reduced. If associated with tobacco consumption, a mean peak systolic velocity (PSV) of 123 ± 43 cm/s at the origin of the SMA is considered to be normal (Fig. 1); slight elevation, if PSV is 141-180 cm/s; moderate elevation in case of 181-220 cm/s, and severe elevation if PSV is > 220 cm/s. A PSV higher than 275 cm/s may indicate stenosis in

more than 70% of the SMA[12]. The purpose of this study was to define if the longer the tobacco consumption, the greater the increase of SMA PSV, as assessed with pulsed Doppler.

Material and methods

This was an analytical, cross-sectional study conducted at the Radiology and Imaging Department of the Regional Specialties Hospital, UMAE Centro Médico Nacional (CMN) No. 25. Patients attending to have an abdominal Doppler performed and who had a history of chronic smoking were included. An additional SMA PSV measurement was performed in 60 patients. The results were measured in cm/s and were collected in the case report form. The patients were selected with a non-probabilistic method, including the total number of patients attending our Department. The participants were explained the research work approved by the Ethics Committee of the UMAE 25 and their signatures were collected in an informed consent form. Once the patients were selected and the pulsed Doppler test was performed, interpretation of the results was carried out. The SPSS program was used for analysis of the results.

Results

Sixty chronic smoker patients, from 25 to 50 years of age, with a median of 38.6 ± 7.1 years, out of which 15 were females (25%) and 45 (75%) males, were included. Smoking chronicity was measured in years, with a median of 17.1 ± 8.4 (range: 3-37 years). PSV was divided in 4 ranges: normal value (100-140 cm/s), which included 15 patients (25%); mild PSV elevation (141-180 cm/s), with 22 patients (36%); moderate PSV elevation (181-220 cm/s), with 10 patients (17%); and severe PSV elevation (> 220 cm/s), with 13 patients (22%). A significant SMA PSV increase was obtained in 27% of the patients ($p = 0.00007$) (Fig. 1).

Discussion

Smoking represents a public health problem, since it is a risk factor for six of the eight main causes of death in the world[1]. According to the WHO, 5.4 million people who suffer from lung cancer, heart diseases and other tobacco consumption-related conditions die each year, but its effect on blood vessels has been scarcely studied. In Mexico and in the rest of the world there are very few articles mentioning the effect of smoking on SMA PSV in chronic smoker patients. In the present study, the SMA PSV of 60 patients with previous tobacco-consumption time ranging from 3 to 37 years was assessed with pulsed Doppler[2].

We know that, immediately after its absorption, nicotin produces an activation of adrenal grands and that these, in turn, produce a body stimulation and a sudden glucose discharge, as well as an increase in blood pressure, respiration and heart rate[6]. Furthermore, the habit of smoking reduces the diameter and distensibility of medium and large-caliber arteries; the mechanism by which cigarrette smoking exerts this vasoconstriction effect is associated with local or systemic release of catecholamines and sympathetic neural stimulation[7]. Therefore, any probable vasoconstrictor effect on SMA perfusion can produce unwanted results in patients who are chronic smokers, such as stenotic lesions in this artery and chronic intestinal ischemia[8]. Patients with SMA lesion refer chronic abdominal or acute post-prandial pain, abdominal distension, vomiting, unspecific weight loss and malabsorption. Pulsed Doppler is a screening test in these patients that allows, within the diagnostic algorithm, for the clinician to be guided to include a future intervention such as, for example, an angiography to corroborate or exclude some diagnosis. Pulsed Doppler has advantages over angiography because it is a non-invasive and reproducible method that serves to screen patients with chronic stomach pain of undetermined origin, which, together with a history of chronic smoking, indicates probable SMA stenosis and/or intestinal ischemia.

Nation-wide, tobacco consumption causes a third of heart disease-related deaths, but the effect of chronic smoking at the abdominal level has been scarcely studied, even though abdominal pain is one of the primary causes motivating patients to seek help at medical outpatient clinics of the IMSS. The results obtained in the present study will help to increase quality and warmth in the care and service of those affiliated to the Institute, which translates into quality of life for the population. In the international panorama, in the Middle East, specifically in Turkey, Babaoglu et al. conducted a research assessing the effects of smoking on the SMA and the portal vein using Doppler ultrasonography in an experimental model with 50 patients, and they found that smoking decreased blood flow in the SMA; the study concluded with a $p < 0.05$ significance[13]. In our study, in addition to corroborating the finding of smoking-associated SMA flow decrease, we were able to determine that chronic smoking significantly increased the SMA PSV 21 years after the start of tobacco consumption. It's very important to mention that the

study protocol calls for an ultrasonographic scan of the patient, with sections of the aorta, including the celiac artery and the SMA. PSV is obtained for the aorta and the SMA. This way, velocity changes at the proximal SMA are inferred to be possibly related to colateral blood flow when the celiac artery is obstructed. Additionally, atherosclerotic changes of the SMA, which may appear as areas of enhanced echogenicity and irregular thickening of the wall, will be looked for[14].

In healthy individuals' Doppler registration, there is high-impedance blood flow in the SMA during the fasting state. Diastolic blood flow can be absent, insignificant and even appear to be inverted. The reading obtained at the SMA origin can show turbulent blood flow, but it is more uniform distally[15]. SMA blood flow usually decreases significantly during ingestion. Systolic velocity can be double with a broad systolic peak. At diastole, there is also significant velocity increase with continuous flow. However, response to ingestion is variable and can be normal, highly increased, and sometimes even decreased. If associated with tobacco consumption, a mean PSV of 123 ± 43 cm/s at the SMA origin is considered to be normal (Fig. 1); 141-180 cm/s values are considered to be mild PSV elevation; values of 181-220 cm/s, moderate elevation, and PSV > 220 cm/s, severe elevation. A PSV higher than 275 cm/s may indicate > 70% SMA stenosis[12].

The present study corroborated that pulsed Doppler has advantages over angiography because it is a non-invasive method thet serves as screening in patients with chronic abdominal pain of unknown causation, which, together with a history of smoking, would indicate probable SMA stenosis and/or intestinal ischemia[16].

Conclusion

Chronic smoking significantly increases the SMA PSV 21 years after the start of tobacco consumption and has a significant relationship with the male gender. Pulsed Doppler should be included as a screening test within the diagnostic algorithm in patients with chronic pain of unknown causation; together with a history of chronic smoking, it can determine whether the patient warrants or not a more invasive study, such as abdominal angiography, since it suggests probable SMA stenosis and/or intestinal ischemia. Pulsed Doppler offeres advantages over angiography because it is a non-invasive, reproducible and readily available method.

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