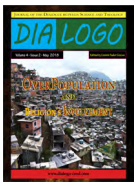
Smoking and the cerebrovascular disease



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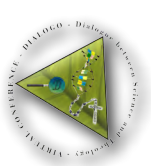

Smoking and the Cerebrovascular Disease

1. Any Docu-Axelerad, PhD
Ovidius University of Constanta,
Department of General Medicine
Constanta,
Romania
docuaxi@yahoo.com

2. Daniel Docu-Axelerad, PhD
Ovidius University of Constanta, FEFS,
Kinethoteraphy Department
Constanta,
Romania

3. Negrea Valentin, PhD
Ovidius University of Constanta, Department of General Medicine
Constanta,
Romania
negreavali@yahoo.com

3. Musat Gheorghe, PhD
Ovidius University of Constanta,
Department of General Medicine
Constanta,
Romania
musat_grg@yahoo.com

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ABSTRACT

Tobacco use has reached epidemic proportions worldwide, about one billion people are smokers globally. Although by comparison there are fewer smokers now than 25 years ago, the increased consumption of tobacco is due to global population growth. A smoking habit is a physical and psychological addiction to tobacco products, with serious health consequences. Smoking significantly increases the risk of developing the cerebrovascular disease due to the harmful effects of chemical substances contained in cigarette smoke in the blood vessels.

I. INTRODUCTION

Smoking was the second leading risk of early death and disability worldwide in 2015.[1] More work is still needed to target tobacco use, despite the progress in reducing the number of smokers. The researchers found that although the percentage of people who smoke has declined, the overall number of smokers has actually increased, due to population growth.[2]

II. MATERIAL AND METHOD:

We analyzed literature data that correlate nicotine abuse with diseases, especially with a cerebrovascular disease

III. DISCUSSION AND RESULTS:

Nicotine is the primary substance in the cigarette and can act as a stimulant or as a depressant, depending on the amount

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consumed and the circumstances, and that's why some people report that smoking gives them energy and stimulates their mental activity, while others note that smoking relieves anxiety and relaxes them. Nicotine deregulates cardiac autonomic function boosts sympathetic activation, raises heart rate, causes coronary and peripheral vasoconstriction, increases myocardial workload, and stimulates adrenal and neuronal catecholamine release. Also, nicotine is associated with insulin resistance, increased serum lipid levels, and intravascular inflammation that contributes to the development of atherosclerosis. [3] Nicotine might alter the function of the blood-brain barrier and disrupt normal endothelial cell function.

Cigarette smoke contains, besides nicotine, other substances like carbon monoxide, formaldehyde, arsenic, and cyanide, chemicals that can cause damage to blood vessels increasing the risk of all types of cardiovascular and cerebrovascular diseases.

Tobacco's smoke ingredients cause endothelial damage and dysfunction, increase oxidative stress and also have a significant effect on lipid metabolism and the regulation of lipid levels in the blood.[4] Smoking is associated with elevated serum concentrations of total cholesterol, low-density lipoprotein(LDL) and very low-density lipoprotein(VLDL) and triglycerides leading to atherosclerosis. On the other hand, smoking lowers serum concentrations of high-density lipoprotein (HDL) cholesterol, a potent protective factor against the development of atherosclerosis. [5] [6]

The atherosclerotic effects of cigarette smoke are due to a substantial degree to thrombosis-related events.[7]

Cigarette smoke also produces many other events such as increased platelet aggregability, polycythemia, and carboxyhemoglobinemia, decreased macrophage activity, increased fibrinogen levels and direct toxic effects of compounds such as 1,3-butadiene, a vapor phase that accelerates atherosclerosis in animal models. All these mechanisms increase the risk of ischemic stroke.

Chronic cigarette smoking affects the normal hemostasis by influencing the coagulation pathways. However, the effect of smoking intensity on the degree of impairment of coagulation cascade remains unclear. Chronic smokers tend to have lower platelet count, shorter APTT, and higher platelet aggregability compared to non-smokers. Therefore, chronic smokers should be investigated for hemostatic dysfunctions.[8]

Usually, erythrocyte masses are larger in smokers compared to non-smokers. Also, the levels of carboxyhemoglobin in cigarette smokers are higher than average, sufficient to cause clinically significant hypoxemia and to account for the increased in red blood cell count.

When the carbon monoxide from de cigarette smoke is inhaled, by either active or passive smokers, blood levels of carboxyhemoglobin are increased while the intake of O_2 to tissues decreases, affecting the vascular permeability. The increase in endothelial permeability, together with the injuries to the intima of the arterial wall associated with exposure to CO, leads to subendothelial edema manifested by early atherosclerotic changes, such as fat deposition in the arterial walls.[9]

Risk factors for stroke are non-modifiable, represented by age, race, genes, ethnicity and modifiable like cigarette smoking, hypertension, diabetes mellitus, hyperlipidemia.

Cigarette smoking remains a significant risk factor for stroke, nearly doubling the risk with a dose-response relationship between pack-years and stroke

risk. [10] [11]

It is estimated that smoking contributes to ~15% of all stroke deaths per year.[12]

Second-hand smoke contains many of the same chemicals that people inhale when they smoke, and it has been identified as an independent risk factor for stroke. Second-hand smoke refers to the smoke exhaled by the smoker in combination with the smoke emitted into the environment from lit cigarettes.

The cerebrovascular effects of passive smoking are nearly the same as for the real smokers, operating through the same biological mechanisms such as vasoconstriction, inflammation and formation of clots.

Smokeless tobacco it is also used around the world. One systematic review consisting of American and European studies has reported an association between smokeless tobacco use and stroke. [13] The risk of fatal stroke associated with smokeless tobacco use was significantly higher.

Cigarette smoking is also associated with higher risk of hemorrhagic stroke, subarachnoid hemorrhage, and intracerebral hemorrhage.

Subarachnoid hemorrhage represents the extravasation of blood into the subarachnoid space, and it can be traumatic or nontraumatic-which usually occurs in the setting of a ruptured cerebral aneurysm or arteriovenous malformation.

Regarding the close link between cigarette smoking and the occurrence of subarachnoid hemorrhage, studies conducted over time, have shown that smoking was found to be a short and long-term risk factor for subarachnoid hemorrhage to all smokers, but especially for aneurysmal subarachnoid hemorrhage and women. Importantly, it has been found that smokers have a greater amount of aneurysms that are also larger than those in non-smokers.[14] [15]

Recent data have implicated a role of inflammation in the development of cerebral aneurysms. Inflammation accompanying cigarette smoke exposure may thus be a critical pathway underlying the development, progression, and rupture of cerebral aneurysms.[16]

Smoking is an established risk factor for subarachnoid hemorrhage, yet the underlying mechanism is largely unknown.

Several hypotheses may explain the effects of smoking in subarachnoid hemorrhage, including enhanced systematic coagulability, inflammation within arterial walls, increased blood pressure, endothelial dysfunction, and the promotion of the degradation of elastin within vessels walls by interfering with alpha1-antitrypsin.[17]

Cigarette smoking can cause an acute increase in blood pressure immediately after smoking, due to stimulation of the sympathetic nervous system, an effect that can last up to three hours. The risk in blood pressure could contribute to the rupture of aneurysms or even small intracerebral arteries.[18]

Connections appear to be dose-dependent, the chance of subarachnoid hemorrhage being directly proportional to the amount of cigarettes smoked, while smoking cessation reduces the risk of this type of bleeding over time.

Intracerebral hemorrhage is the second most common subtype of stroke and is defined as bleeding within the brain parenchyma due to rupture of an intracerebral blood vessel. Regarding intracerebral hemorrhage, the risk factors involved in its occurrence are hypertension, smoking, alcohol, cholesterol, and lipids, diabetes mellitus, cerebral amyloid angiopathy, anticoagulant medication or

genetic risk factors.

Although smoking is not a risk factor as high as hypertension in the development of intracerebral hemorrhagic events, studies have consistently shown tobacco use as a risk factor for intracerebral hemorrhage.[19] [20]

The relationship in this pathology is also dose-response, the risk of intracerebral hemorrhage being lower in non-smokers compared to smokers or even former smokers.

Cigarette smoking is a major risk factor for stroke, where it confers a similar hazard in women as in men. Similarly, the benefits of smoking cessation on the future risk of stroke are the same in both sexes. Tobacco control policies that target both smoking initiation and suspension should be a mainstay of stroke primary prevention programs, particularly in low and middle-income countries, which shoulder the greatest dual burden of tobacco exposure and stroke.[21]

The tobacco epidemic is one of the most significant threats to public health facing humanity, tobacco killing active or passive about half of those who consume it.

Numerous epidemiological studies in various countries (INTERHEART, ISIS) show that smoking is a significant concern in the etiology of cerebrovascular, cardiovascular, peripheral vascular diseases and that there is a dose-effect relationship.

In terms of cerebrovascular disease, smoking itself is one of the most critical risk factors but at the same time, smoking has a potentiating effect on other risk factors (oral contraceptives-for example) and the effect of toxic compounds in tobacco on cerebral vascular system can be both acute and chronic.

Stroke mortality is higher for smokers compared to non-smokers, regardless of sex, and smoking cessation is associated with both lowering the risk of stroke in all its forms and decreasing mortality through this condition.

So, it is essential to achieve early detection and intensive consumption of tobacco, starting with population screening, informing patients about harmful effects of smoking both passively and actively and sustaining them throughout the period through various methods such as psychological therapy [22] or nicotine replacement therapy.

Conclusion:

A smoking habit is a physical and psychological addiction to tobacco products, with serious health consequences. Smoking significantly increases the risk of developing cerebrovascular disease due to the harmful effects of chemical substances contained in cigarette smoke in the blood vessels.

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