Review Article

Effects of Smoking on Cardiovascular System

Abstract

Background: Smoking is a major risk factor for heart disease and death, and is recognized as one of the leading causes of death in the world. Cigarette smoke contains more than 4000 chemicals, including nicotine and carbon monoxide (CO), which can have harmful effects on heart function. These basic components of cigarette smoke cause oxidative stress, endothelial damage and deactivation and are associated with very high serum concentrations of total cholesterol and triglycerides, as well as low levels of cardioprotective high-density lipoprotein. By causing intravascular inflammation, smoking promotes the development of atherosclerosis and heart disease. Exposure to secondhand smoke makes atherosclerosis disappear and increases the risk of MI, stroke, aortic aneurysm and sudden death. Smoking can cause DM and accelerate the occurrence of minor DM and macrovascular complications. Smoking has causal relationships and frequent interactions with other major risk factors for CHD, including hyperlipidemia, hypertension. The effects of smoking are almost the same or almost the same as that of smoking. The purpose of this article is to provide a brief overview of the effects of smoking, and specifically the effects of nicotine and CO on cardiovascular function. Nicotine reduces independent cardiac function, increases sympathetic activity and increases heart rate (HR).

Conclusion: By quitting smoking we have many options like behavioral changes and treatment instead of nicotine and bupropion.

Aim of the study

To understand the effects of smoking on cardiovascular system and some knowledge about associated disorders thus you can manage it to decrease the suffering and prevent catastrophic death for the patient.

Introduction

Smoking is a major risk factor for heart disease and death and is considered one of the most preventable deaths in the world. Globally, 25% of deaths from heart disease are due to smoking. The European Society of Cardiology recently reported that smoking causes 28% of heart deaths in men aged 35 to 69 and 13% in women of the same age. In the European region of the World Health Organization (WHO), smoking is the second highest risk factor for life expectancy due to disability and the highest risk factor for premature death, accounting for 1.6 million deaths annually. happens. It is estimated that 15% of all deaths from tobacco use in the European Union are due to smoking each year, and in Greece, any smoking-related death in people aged 35 and over Also the number of deaths due to old age, accounting for 18.1%. (1).

The WHO estimates that tobacco use kills about 6 million people each year, including more than 600,000 smokers from heart disease, lung cancer and other diseases. If current trends continue, it is estimated that more than 8 million people will die annually by 2030. Smoking is one of the leading causes of heart disease, such as heart disease, ischemic stroke, peripheral arterial disease, and gastrointestinal aneurysm. It is associated with an increased risk of certain types of cancer and is a major cause of chronic obstructive pulmonary disease. Active or passive smoking can cause heart disease through a series of interdependent processes such as increased oxidative stress, hemodynamic changes, and autonomic flexibility. .., Endothelial dysfunction, thrombosis, inflammation, hyperlipidemia, or other side effects. Occasional smoking, less smoking, and even small exposures such as a few cigarettes a day are enough to cause serious side effects. Cigarette smoke contains more than 4,000 chemicals that have a serious effect on heart function (2).

Smoking contributes to other cardiovascular risk factors, such as sugar intolerance and low serum levels of high-density lipoprotein cholesterol (HDLc). However, studies have shown that smoking increases the risk of CVD in addition to the effects of smoking on other risk factors. In other words, if smoking doubles the risk level and at the same time another risk factor, adjustments are made to distinguish between smokers and non-smokers at the level of these other risk factors. The risk is estimated to increase fourfold. Smoking has been linked to

peripheral arterial disease (PAD), aortic aneurysm, CHD and cerebrovascular disease, but the associated risk of disease (RR) depends on the arterial bed (3).

Cardiovascular effects

1-Sudden Death

Most sudden deaths are due to CVD. In a detailed analysis combining data from the Framingham Heart Study and Albany Study, which examined sudden cardiac death in men aged 45-64 years, smoking was a statistically significant risk factor. In a data study of the 1986 National Mortality Follow-up Survey of People without a History of CHD, smoking was the only variable risk factor for sudden cardiac death, increasing the risk of sudden cardiac death in people with heart disease. It was a related factor. .. Known as CHD. In patients with left ventricular dysfunction after MI, there is an association between smoking cessation and reduced mortality from cardiac arrhythmias. Finally, smokers who survived out-of-hospital cardiac arrest had a lower risk of recurrent cardiac arrest in smokers than in nonsmokers (4).

2-Stroke

Smokers have a higher risk of stroke due to cerebral palsy, greater mortality and a clear association with volume response than non-smokers. In addition, following a 20-year prospective mortality study controlled for by other risk factors associated with cardiovascular risk, smoking increases the risk of stroke and increases mortality as the number of smokers increases. In a meta-analysis of data from 32 studies, the data indicated an increased risk of stroke in former smokers younger than 75 years than non-smokers in the same age group. Together for all ages. During the group's 26-year follow-up study on the Framingham Heart Study, smoking was a major risk factor for stroke. However, the risk of smokers who quit smoking for two years was lower than that of smokers who did not smoke throughout their lives after quitting smoking for five years (5).

3-Aortic Aneurysm

Arteriovenous arterial occlusion disease is a different form of vascular degeneration than atherosclerotic and other arterial occlusive diseases. The formation of an aneurysm within the vascular tree has a regional distribution, and the pathological process varies depending on the localization. Abdominal infrarenal aortic aneurysm (AAA) is the most common manifestation of aneurysm disease, and smoking is the most dangerous factor.

Smoking is a much greater risk for AAA than atherosclerosis. Smoking not only plays a role in the etiology of AAA, it also increases the rate of development and the risk of fractures of established AAA. The mechanical relationship between AAA and smoke is established through the use of advanced animals based on smoke or smoke components. The processes involve long-term changes in vascular smooth muscle cell and inflammatory cell function. This review will examine the clinical, epidemiological and technical evidence considering tobacco as a cause of aneurysms, with an emphasis on AAA (6).

Arteriovenous arterial occlusion disease is clinically, historically and functionally distinct from arteriosclerosis and other arterial occlusive diseases. Although atherosclerotic changes can occur in most aneurysms, this is not the cause. Aneurysms are more common in some arteries than others, and their etiology varies by location, eg. Although little evidence suggests that smoking plays a role in some manifestations of aneurysm disease, the role of smoking in the development or progression of other aneurysms has not been considered (7).

4-Hypertension

Smoking is a major risk factor for cardiovascular disease, and smoking cessation is one of the most effective ways to prevent many cardiovascular diseases. Endothelial dysfunction, arteriosclerosis, inflammation, lipid conversion, antithrombotic and thrombosis-promoting changes are the major factors associated with smoking and the acceleration of atherosclerotic processes leading to cardiovascular events. Smoking has a hypertensive effect, especially by stimulating the sympathetic nervous system (Fig. 1) (8).

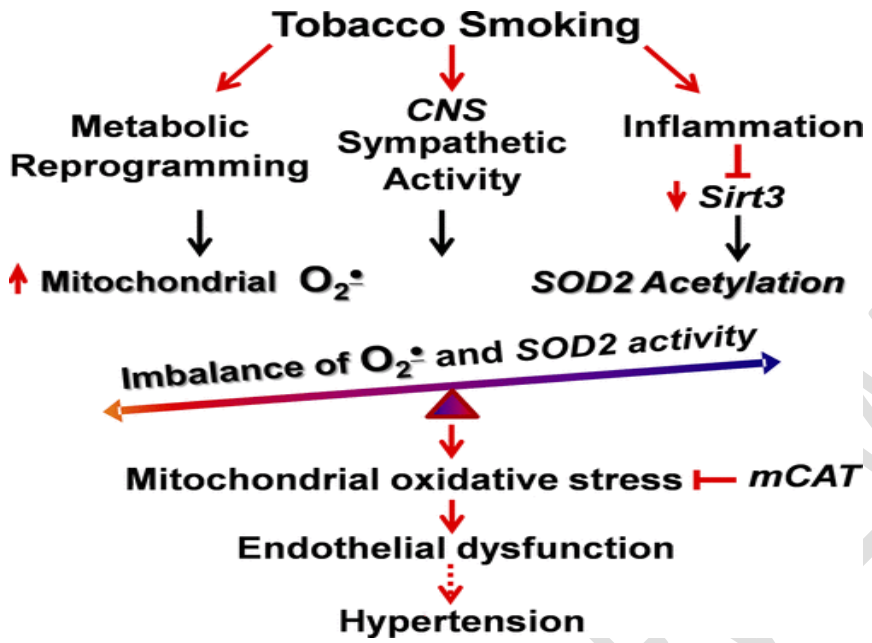

Figure 1 (Hypertension Pathogenesis) (8).

5-lipid metabolism

The effects of smoking include changes in lipid metabolism with increased lipolysis, insulin resistance, and tissue lipotoxicity. Smoking is prothrombotic and atherogenic (Fig. 2) (9).

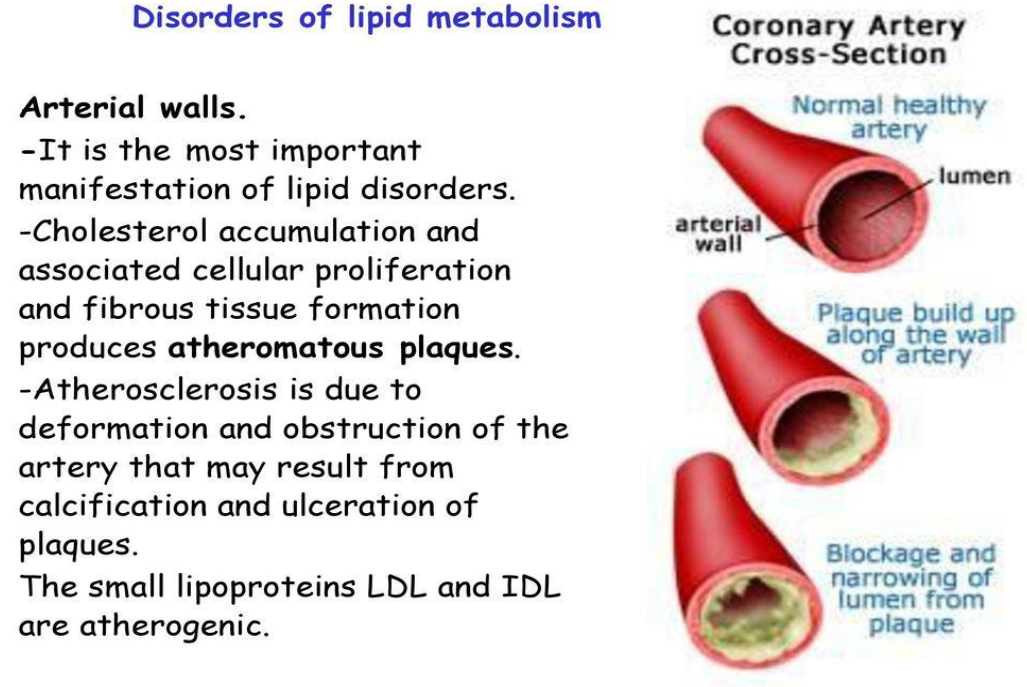

Figure 2 (Disruption of Lipid Metabolism in Smokers) (9).

6- Hemodynamic Effects

6.1-Blood Pressure and Heart Rate

Nicotin stimulates the release of epinephrine and norepinephrine from the adrenal medulla and endothelial nerve endings, resulting in increased heart rate and maximum attenuation by stimulation of myocardial β1 receptors. Peripheral vascular resistance increases with α-receptor-mediated vasoconstriction that raises blood pressure. It also promotes β2 and α2 receptors in the coronary arteries. Stimulation of β2 receptors promotes vasodilation, and stimulation of α2 receptors promotes vasoconstriction (10).

6.2-Coronary Blood Flow

Smoking alters the response of coronary blood flow to increased myocardial oxygen demand; that is, it decreases the flow of the coronary vasodilator blood flow reserve. Therefore, the increase in coronary blood flow based on the level of myocardial function is less than would be expected in the absence of secondhand smoke exposure. There is ample evidence that tobacco use causes coronary artery endothelial dysfunction. Smoking can be associated with narrowing of the coronary vessels. Although smoking increases blood flow to a person without coronary heart disease, it can decrease cardiovascular blood flow in heart disease (10).

7-Thrombogenic Effects

Smoke-induced thrombosis appears to be an important factor in the development of serious cardiovascular events. Epidemiological evidence suggests that smoking increases the risk of dangerous myocardial infarction and sudden death in addition to the risk of angina. Researchers believe that the risk of major myocardial infarction and sudden death is related to thrombosis and that angina pectoris is mainly due to hemodynamic factors. Successful rehabilitation in MI patients after thrombol therapy is more likely in smokers than nonsmokers (10).

8-Arteriosclerosis

The formation of platelets lining the artery wall where there is dynamic blood flow or endothelial damage may be the prodromal stage of atheromatous plaque formation. Nicotine is thought to increase blood viscosity and platelet aggregation as it inhibits the production of prostacyclin which can reduce platelet aggregation. Platelet adhesion increases the production of thrombi, separates the nerves of the coronary arteries, accelerates the process of atheromatous plaque formation and is

associated with an increased risk of cardiac ischemia. In addition, nicotine affects the metabolism of prostaglandins, impairing the defense of vessels against platelet aggregation (Fig. 3) (11).

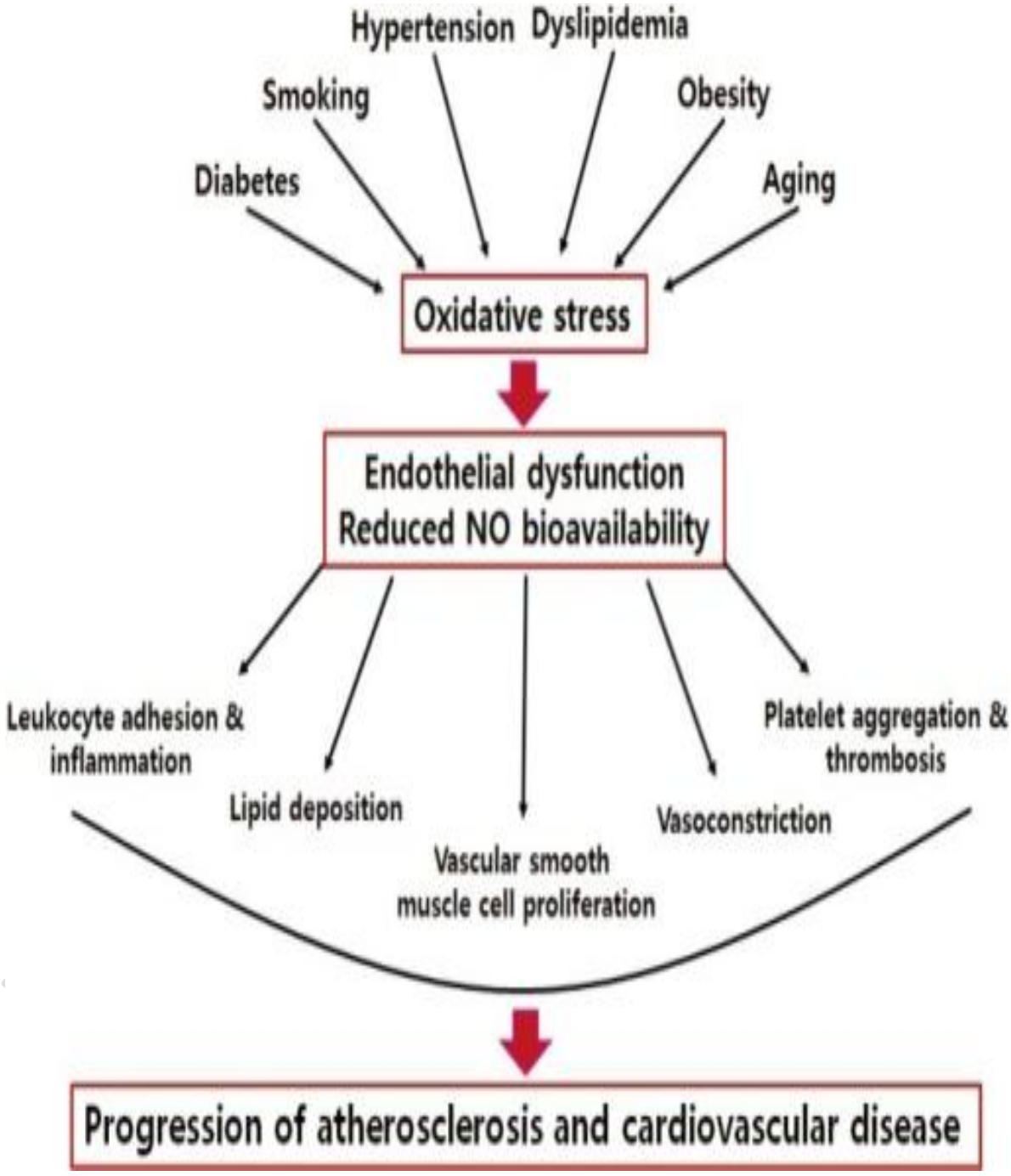

Figure 3 (Atherosclerosis in Smokers) (11).

9-Endothelial Affection :

In response to exposure to smoke, endothelial cells are known to release inflammatory and proatherogenic cytokines. All of these processes lead to endothelial dysfunction. The direct physiological effects of smoke compounds and the production of active forms of oxygen (ROS) lead to endothelial cell loss due to apoptosis or necrosis (Fig. 4) (12).

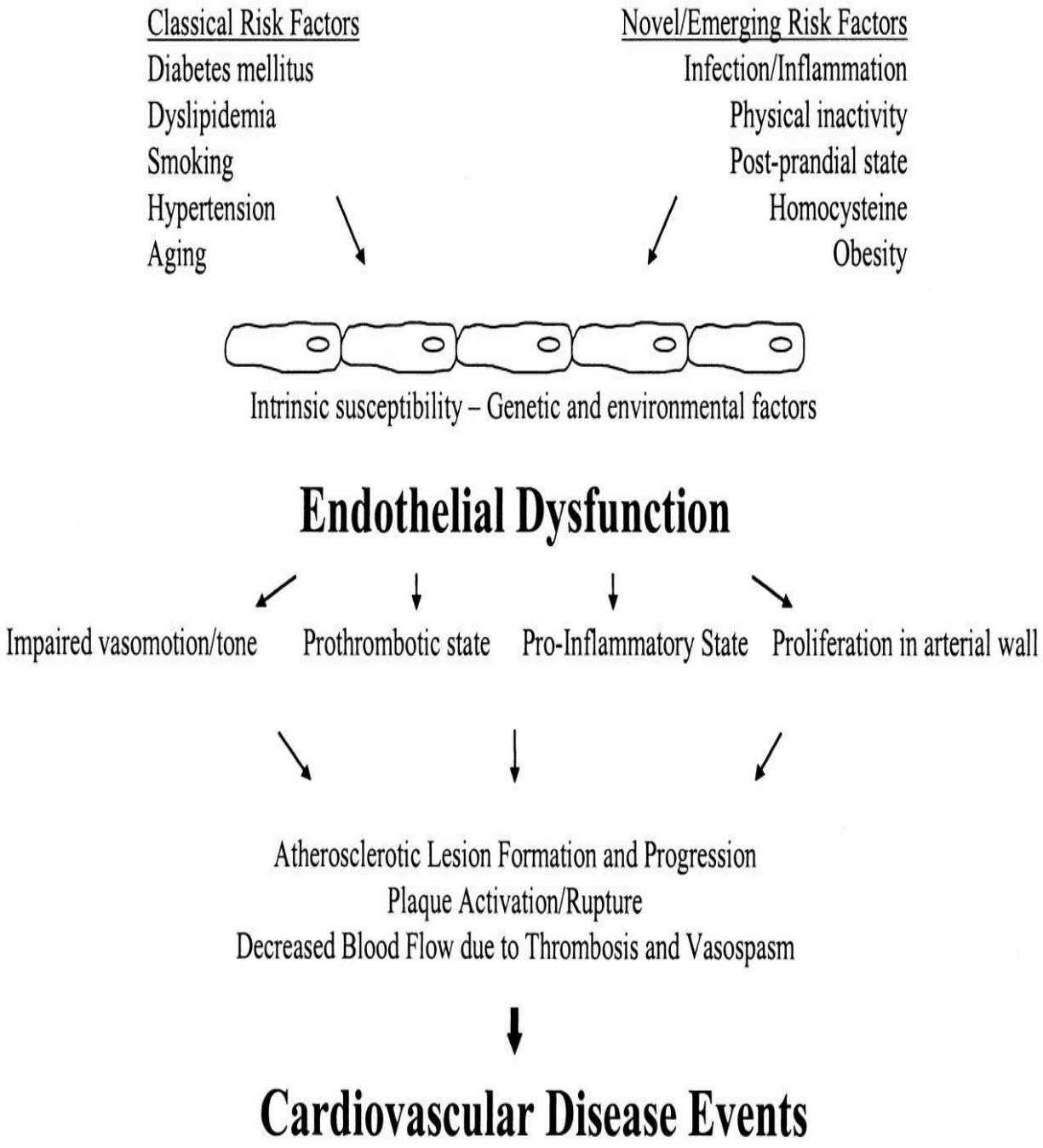

Figure 4 (Endothelial Damage in Smokers) (12).

10-Inflammation

Studies suggest that inflammation affects atherosclerosis as high leukocyte counts and high CRP and fibrinogen levels are potent predictors of future cardiovascular events. However, the mechanism by which tobacco smoking promotes inflammation has not been fully elucidated (Fig. 5) (13).

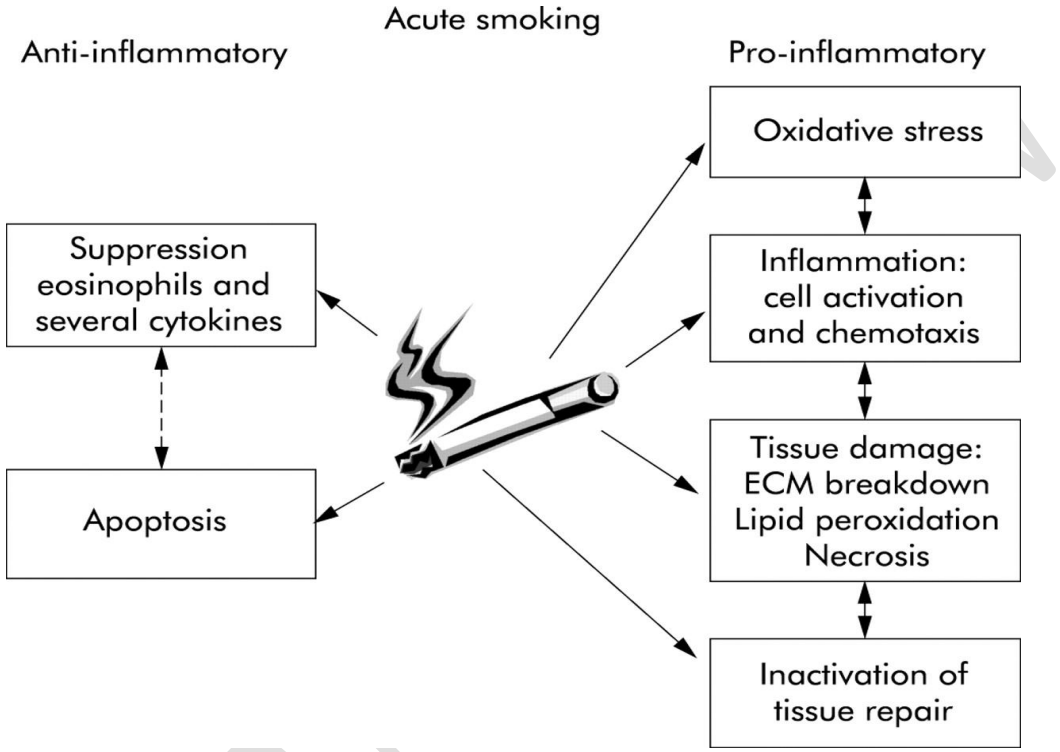

Figure 5 (inflammation pathogenesis in smokers) (13).

11-Smoking and Diabetes

Numerous studies have shown that smoking negatively affects glucose and lipid metabolism in people with or without diabetes. The investigators reported that smoking in diabetic patients was associated with decreased metabolic control and an increased risk of microvascular and macrovascular complications and death (Fig. 6) (14).

Figure 6 (How Smoking causes Diabetes) (14).

Complications:

1-Microvascular Complications

The microvascular disorders of diabetes (retinopathy, nephropathy, neuropathy) are associated with metabolic control in type 1 and type 2 diseases. The mechanisms of development of microvascular diseases are not fully understood, although several pathogenic mechanisms have been proposed. Hyperglycemia plays a key role as a trigger for downstream events such as the conversion of glucose to sorbitol by aldose reductase; non-enzymatic glycosylation of proteins and receptors in affected tissues; increased exposure to oxidative stress; and activation of protein kinase C and mitogen-activated protein kinases (Fig. 7, 8) (15).

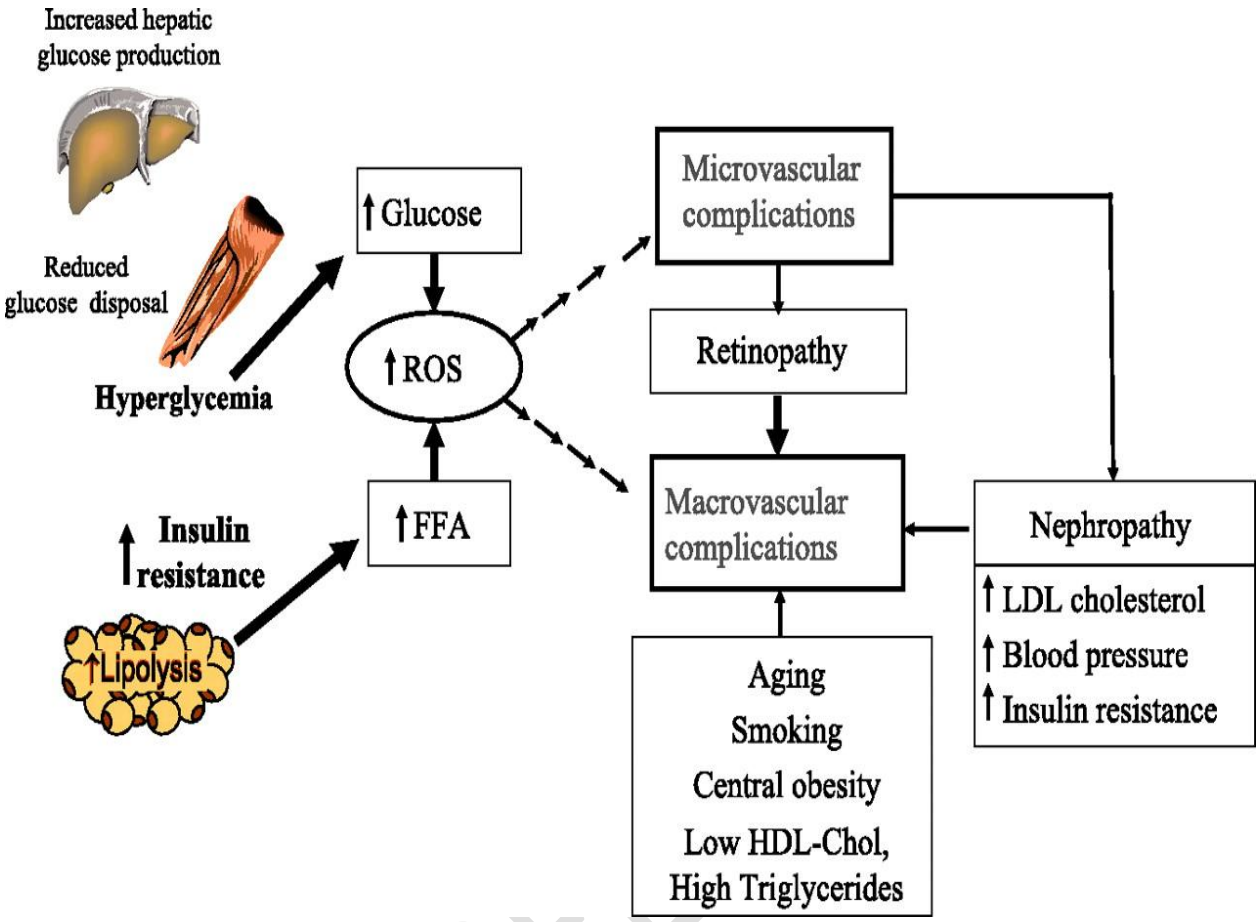

Figure 7 (Microvascular Complications of Smoking) (15).

1.1-Nephropathy:

Smoking and hyperglycemia increase oxidative stress and lipid accumulation, which regulates TGF-β, accumulates AGEs, reduces nitric oxide production, leads to inflammation of the lower glomerular membrane and mesangial proliferation, and further progression of mesangial glomerulosclerosis and interstitial fibrosis, as well as the effects of fibrosis (fig. 8) (15).

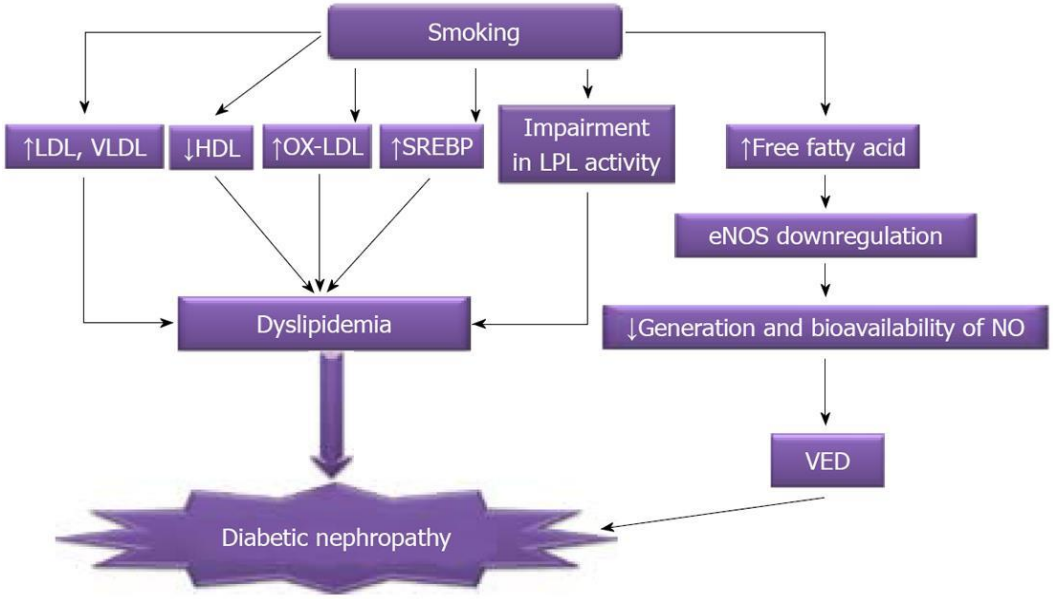

Figure 8 (How Smoking Causes Diabetic Nephropathy) (15).

1.2-Retinopathy

In general, researchers have never considered smoking to be a major risk factor for diabetic retinopathy. The results of a very large multicenter study show no strong support for such associations, except in the elderly with certain medical conditions. However, at least two studies of patients with type 1 diabetes suggest that smoking prioritizes patients with this retinopathy (Fig. 9) (16).

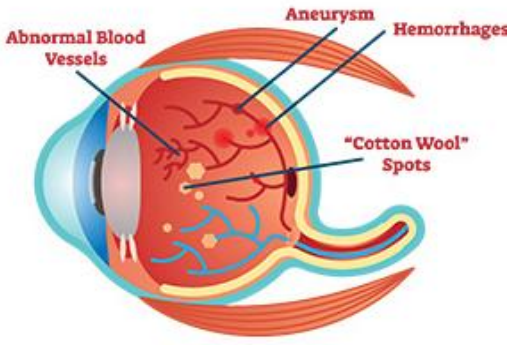

Figure 9 (Effect of Smoking on the Eye) (16).

1.3-Neuropathy

Smoking affects blood circulation and can increase the risk of peripheral neuropathy. Care should be taken in foot care, as peripheral neuropathy can begin slowly with numbness in the feet.

Check your feet daily for cuts and other wounds (Fig. 10) (17).

Figure 10 (Effect of Smoking on Neurons) (17).

2-Macrovascular Complications

Patients with diabetes are at increased risk of smoking-related complications as they are at increased risk of coronary artery disease and death. In a study group called the Multinational Study of Vascular Diseases of Diabetes, sponsored by the World Health Organization, in a study group in London, England, among other things, smokers with type 1 or type 2 diabetes did not significantly increase their risk of CHD. After an eighth year in the Stroke Diabetes and Complication Management Study (New England Journal of Medicine 1993), which examined the role of intensive insulin therapy and glycemic-controlled therapy in type 1 diabetes, smoking was not a major risk factor for macrovascular complications. Because the participants were small, this study was not well designed to investigate the role of tobacco use. Some studies in young participants with type 1 diabetes have reported that smoking increases the risk of CHD (Fig. 11) (18).

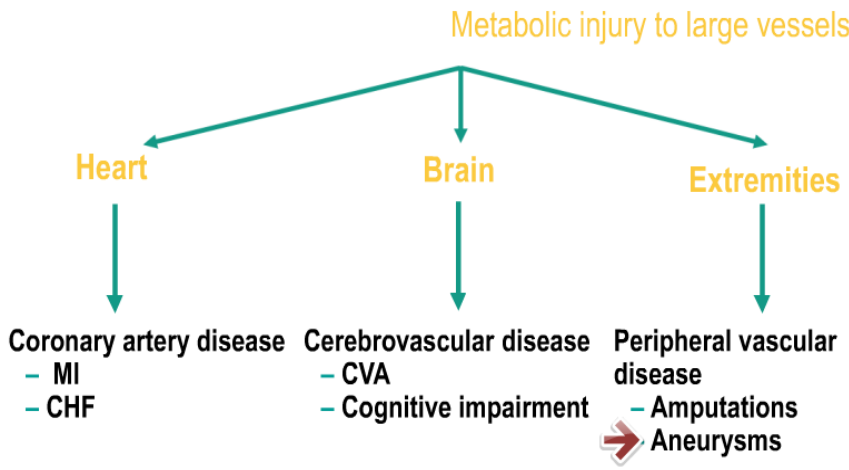

Figure 11 (Macrovascular Complications of Smoking) (18).

Results

Tobacco kills about one person every six seconds, killing 1 in 10 adults. Up to half of current consumers will eventually die of tobacco-related disease. About 80 percent of the world's more than 1 billion smokers live in low- and middle-income countries, where the burden of tobacco-related diseases and deaths is much higher. Every year an estimated 6 million people worldwide die from the effects of smoking. About half of children regularly breathe air polluted with cigarette smoke in public places. More than 40% of children have at least one parent who smokes. Secondhand smoke causes 600,000 premature deaths each year. According to the 2012 Eurobarometer report "European Attitude Tobacco", the prevalence of smoking among Europeans aged 15 years and older is 28%, with the highest prevalence in Greece (40%) and the lowest in Sweden (13%). Italy (like the Netherlands) is in fourth place with a frequency of 24% from last time. The 2012 Italian DOXA survey, which is commissioned every year by the Istituto Superiore di Sanita, found a low rate of 21% (19).

Specifically, in Egypt, 37.7% of men, 0.5% of women and a total of 19.4% (9.6 million adults) currently smoke. In the country, 35.8% of men and 0.5% of women smoke every day. 6.2% of men, 0.3% of women and a total of 3.3% (1.6 million adults) currently use hookah. 6.5 million adults (60.7 percent of adults whose workplace includes home) are exposed to secondhand smoke. Another study of high school students in the suburbs of Egypt found that 19% of people ever smoked hookah. Three studies of older men living in the valleys found that 11-15% smoked in pipe groups. The percentage of smokers is also high among healthcare professionals and medical students. According to a study

published in 2010, the prevalence of smoking among healthcare workers in Italy is 44%, more than double the general population, and this is only due to an increase in the number of nurses (48.2%). Doctors (33, 9%), medical students. (35%) and graduate students (52.9%) (19).

Discussion

Three-quarters of cigarette smoke has received much attention contributing to CVD: nicotine, carbon monoxide (CO) and oxidizing gases, and other cigarette smoke components, including atherogenesis in the pathology of CVD. Nicotine, which is rapidly absorbed in cigarette smoke, is found in blood levels of 40 to 100 ng / ml after each cigarette is consumed. The usual amount of nicotine taken systematically per cigarette is 1 to 2 milligrams (mg). This pattern of deposition is associated with the end of a half-life of nicotine in two hours. In smokers, nicotine levels in venous plasma reach the plateau early in the morning and remain at this level until bedtime. Thus, these results show that the average smoker receives significant nicotine levels 24 hours a day. Nicotine is sympathetic to the release of catecholamines locally from neurons and from the adrenal gland. In studies of the pharmacodynamics of nicotine, the severity of its main effects is more rapid (20).

Summary and Conclusion

Smoking, with its parent ingredients, nicotine and CO, increases oxidative stress, endothelial damage and dysfunction, is associated with higher concentrations of total cholesterol and serum triglycerides, lowers cardioprotective HDL and, by promoting intravascular inflammation development is an important risk factor for Atherosclerosis and heart disease. Nicotine decreases self-regulation cardiac control, increases sympathetic activity, and increases resting HR, while during sustained exercise slows HR levels and reduces elevated HR gains. Overall, smoking is a very important factor in the ever-increasing risk of heart disease, a major risk factor for heart disease and death, and is recognized as the leading cause of preventable death worldwide. Smoking causes endothelial damage and dysfunction of the coronary and peripheral arteries. Exposure to secondhand smoke increases the risk of thrombosis, stroke, which is a major factor in the pathogenesis of cardiovascular events.

Smoking produces a chronic inflammatory condition that contributes to atherogenic disease processes and increases the

level of biological symptoms of inflammation, powerful predictors of cardiovascular events. Smoking produces an atherogenic lipid profile mainly due to an increase in triglycerides and a decrease in high-density lipoprotein cholesterol. The deleterious effects of smoking on the cardiovascular system and other organs must be addressed throughout the medical community. Cardiologists must collaborate with other professionals to promote integrated legal measures, economic measures, and public education as we work to end tobacco use.

Tobacco smoke is made up of more than 4,700 different compounds. At least 250 of them are considered dangerous and more than 50 cause cancer. Specifically, tobacco smoke contains aromatic and non-aromatic hydrocarbons (dioxene and benzopyrene), alpha and beta aldehydes (acrolein), heavy metals (cadmium, zinc and iron), and a wide range of gases. Toxic (nitrogen dioxide, nitric oxide and carbon monoxide) and bacterial compounds (lipopolysaccharide [LPS]) that have important biological effects on the innate and resilient immune system. The effect of these many components is obvious. Most importantly, some of the effects produced by some of these ingredients were unlike those of secondhand smoke. Thus, it can be assumed that the effects of tobacco smoke cannot be predicted by studying the individual components, but are better estimated using tobacco smoke.

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