

Cigarette Smoking and Coagulation-Fibrinolysis Disorders

Aurelio Leone*

Fomer Director, Department of Internal Medicine, City Hospital Massa, Italy

Corresponding author: Aurelio Leone MD, FASH, FRSPH, Fomer Director, Department of Internal Medicine, City Hospital Massa, Via Provinciale 27, 19030 Castelnuovo Magra, Italy, **E-mail:** reliol@libero.it

It has been for nearly half a century epidemiological and clinic-pathological studies have been identified smoking exposure as a detrimental factor for various components of the cardiovascular system, including coagulation-fibrinolysis cascade.

There is evidence that a wide spectrum of factors as blood vessels, platelets and plasma components play a fundamental role in the formation and dissolution of blood clots, the first step of which initially recognizes a mechanism of vasoconstriction to protect an injured vessel.

Two pathways, deeply interacting among them, intrinsic and extrinsic pathways, lead to the formation of fibrin clot that impedes bleeding. Then, clot lysis is activated by plasmatic factors known as fibrinolytic system. Physiologically, the balance between coagulation and fibrinolytic factors usually determine the restoration of vascular alterations.

Cigarette smoking influences all the steps of the coagulation - fibrinolysis cascade primarily by nicotine and carbon monoxide. These two chemical compounds may act at different levels of hemostatic chain leading to vascular thrombosis. Undoubtedly, severe thrombotic alterations have been clearly documented primarily in the coronary arteries of smokers compared to that of non-smokers.

It is worth noting that thrombosis is the results of three components

specifically belonging to the Virchow triad: injured vessel wall, impaired blood flow and changes in the metabolites of hemostasis.

Among the metabolites of the coagulation - fibrinolysis cascade, increased plasma fibrinogen levels with a reduction of the plasminogen activator system are the parameter mostly influenced by cigarette smoking together with enhanced platelet adhesiveness and aggregation, and endothelial dysfunction.

Several clinical studies have clearly shown that increased concentrations of intravascular fibrin are a strong predictor of severe atherosclerosis in both coronary and peripheral arteries. It is well known that intravascular fibrin is related to plasma fibrinogen levels.

Nicotine and carbon monoxide exert their adverse effects on blood coagulation by a double mechanism: a direct mechanism, which involves a biochemical influence of blood components, and effects mediated through adrenergic and sympathetic stimulation.

In conclusion, it is worth noting that cigarette smoking damages various structures of cardiovascular system, causing adverse effects of anatomical type on heart and artery vessels, but also acts on biochemical components of the blood. The latter fact should be emphasized in a proper manner since it is omitted in the large majority of findings.

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