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Perspective

Atherothrombotic Events in Patients with Type 2 Diabetes Mellitus

Antonio Zoccai*

Department of Cardiovascular Science, Campus Bio-Medico University, Rome, Italy

DESCRIPTION

Diabetes mellitus is a significant risk factor for both poorer post-cardiovascular event outcomes and a first cardiovascular event. The prothrombotic condition seen in diabetic individuals may have contributed to this issue, at least in part. Therefore, individuals with diabetes may benefit clinically more from modern antithrombotic approaches, such as stronger drugs or medication combinations, than those without diabetes. Early endothelial failure, oxidative stress, and vascular inflammation are all linked to a slow-moving process that induces monocyte migration, the formation of foam cells and fatty streaks, and the years-long formation of atherosclerotic plaques in diabetes. Atherothrombotic plaques from diabetes patients are more fragile (prone to rupture) than those from individuals without the disease, which increases the risk of superimposed thrombosis. This is because atherosclerotic plaques from patients with diabetes have higher levels of soft extracellular lipids, inflammation, and a prothrombotic milieu.

The primary cause of the inflammatory pathways linked to vascular problems in T2DM patients is endothelial dysfunction. Additionally, it is the first stage of atherogenesis, which includes suppression of the anticlotting systems as well as an imbalance in the closely controlled equilibrium of vasodilators and vasoconstrictors. These modifications further reduce vasorelaxation and boost vascular smooth muscle cell growth. Platelet dysfunction is one of the major causes of the prothrombotic condition in T2DM, along with other factors. Platelet activation and adherence to active endothelium are mediated by enhanced cytokine release and adhesion molecule production from the endothelium. When endothelium is stimulated, more adhesion molecules and platelet agonists are produced by the endothelium, which increases the susceptibility of platelets to activation. Leukocyte recruitment is thus mediated by activated platelets, which are then stimulated by subsequent contact. Leukocytes subsequently transmigrate and present to lipid and cells in the vessel wall following strong adherence to activated endothelium. This enhances atherosclerotic vascular problems and speeds up subsequent inflammatory responses in the vascular wall.

Endothelial dysfunction is often assessed by measuring NO-dependent endothelium relaxation, also known as vasodilation. When agonists (like acetylcholine) are infused intracoronarily, the diameter of the coronary arteries may be measured by quantitative angiography to quantify endothelial function at the macro and microcirculation levels, respectively. Since it is used to evaluate the vascular bed including atherosclerosis and cardiac events, this approach is known as the "gold standard" for assessing endothelial function and has the highest clinical value. Due to the intrusive nature of forearm blood flow, a different technique that makes use of the brachial artery's Flow-Mediated Dilatation (FMD) in response to shear stress has been developed in recent years. In this method, high resolution Doppler ultrasonography is used to measure changes in artery diameter. Shear stress was employed to stimulate blood flow in the brachial artery by inflating and deflating a blood pressure cuff (reactive hyperemia). The same technique is applied when utilizing finger plethysmography to evaluate peripheral endothelial function. Peripheral Artery Tonometry (EndoPAT), a different method, utilizes the same endothelial-induced vasodilatory response to acetylcholine as a basis for recording increased digital pulse amplitude in response to reactive hyperemia.

The primary role of platelets in hemostasis is to start the blood coagulation process. These should ideally remain dormant and only become active when there is a vascular damage. Upon activation, platelets assemble and release various prothrombotic substances from their granules. These elements consist of vWV, fibrinogen, and coagulation factor V. Different surface Glycoproteins' (GP) expression was different in activated platelets. P-selectin, an adhesion molecule contained in the Weibel Palade bodies of endothelial cells and the granules of platelets, is translocated to the surface during this activation phase. Endothelium controls the action of proaggregants (such as thrombin, collagen, ADP, and TxA2) and releases some antiaggregants, such as NO and PGI2, in healthy blood arteries to prevent the formation of thrombus. NO penetrates the membrane and causes the synthesis of guanylate cyclase, in contrast to proaggregants, which manifest their actions after attaching to certain platelet surface receptors. Diabetes,

Correspondence to: Antonio Zoccai, Department of Cardiovascular Science, Campus Bio-Medico University, Rome, Italy, E-mail: antzoccai@unich.it

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atherosclerosis, hypertension, and heart disease all share the risk factor of excessive thrombus formation brought on by elevated platelet activity.

There is a higher risk of thrombotic vascular events due to platelet dysfunction. The use of antiplatelet medications as part of the treatment regimen for lowering cardiovascular risk has brought attention to the relevance of platelet abnormalities in the atherothrombotic process. Although the multiple causes of platelet dysfunction are outside the purview of this analysis, diabetes and the resulting hyperglycemia cannot be disregarded. In addition to causing a hypercoagulable state, hyperglycemia is a factor in both micro- and macrovascular disease. According to studies, the effect can be minimised by maintaining appropriate regulation of fasting and post-prandial glucose levels. The latter's ability to alter the morphological results of the platelet ultrastructure in diabetic individuals is yet unknown. Type 2 diabetes is associated with a range of haemostatic disorders, including the suppression of fibrinolysis and an increase in fibrinogen. By increasing blood viscosity, clot size, tissue deposition, and stimulating atherosclerosis and vascular thickening, elevated fibrinogen increases the burden of cardiovascular risk. Because changes in erythrocyte composition and rheological function are linked to increased oxidative stress, erythrocytes also play a role in diabetes' high prevalence of atherosclerotic illnesses.