Editorial Article

Stress, atherosclerosis and coronary artery disease

Maria Polikandrioti

During last decades the vast majority of studies has turned their interest to the exploration of the relation between anxiety and coronary disease, as well as to the extent that this relation influences the quality of patients' life. It is well documented that coronary disease consists the first leading cause of death and one of the major causes of disability due to the accompanying complications both in women and men in developed countries. [1-4]

In previous years, the care of the disease was limited to the treatment and the prevention of its' complications. On the contrary, nowadays that the impact of the demographic and clinical characteristics on the development of the disease is fully understood, their assessment is included both in daily clinical practice and in primary and secondary prevention. Moreover, recent data research put emphasis on the role of anxiety and other psychosocial parameters in the development, the outcome and the rehabilitation of the disease. [1-4]

Regarding, biological pathways that link stress to coronary disease, the response to acute stress includes excess release of the sympathomimetic "flight or fight" hormones, cateholamines, due to the hyperactivation of the Hypothalamus-Pituitary - Adrenal (HPA) Axis. As a result, heart rate and blood pressure responses are exaggerated and they return to normal only when the stressor factor disappears. It becomes apparent that the more the perceived threat persists the more chronic is the elevation of blood pressure and heart rate, leading to injury of the vessel's endothelium. Additionally, the excess of catecholamines due to chronic stress results in vasoconstriction and increased platelet activation, altering blood clotting thus accelerating the development of atherosclerosis. Furthermore, the release of corticosteroids mobilize free fatty acids, causing endothelial inflammation and excessive clotting, and are associated with hypertension,hypercholesterolemia, and glucose dysregulation. Increased circulating lipids and endothelial shearing stress can lead to vascular damage and plaque formation. However, the high levels of corticosteroids suppress the immune system and as a consequence the inflammatory response is decreased. [1-4]

Considerable advances are made in understanding other behavioural mechanisms indirectly related to the impact of anxiety in coronary disease, such as lifestyle factors and treatment adherence. More in detail, the response to acute stress includes change in dietary habits, loss of appetite and food intake reduction, whereas chronic stress is associated with changes of the amount, the quality and frequency of food intake. The severity of the stressor factor and the physical or psychosocial consequences that implicates in the daily life of the patients is significantly related to the increase of food intake. A possible explanation is that stressed individuals resort to food as a solution to their problems. Another commonly held view is that stressed individuals have the tendency to smoke more frequently, avoid physical activity or even resort to heavy alcohol consumption. [1-4]

Due to massive research efforts, the role anxiety on coronary artery disease is better understood. However, this bidirectional relation, still remains a matter of controversy in the literature, since high prevalence of coronary disease is remarked in patients suffering from anxiety disorders and on the other hand, coronary disease may trigger anxiety that exerts negative influence the outcome of the disease.

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