Psychological Stress and Coronary Heart Disease

Dear Editor,

We have read the "Editorial" written by Hassan and Sheps[1] in the July-August 2008 issue of the journal concerning "Acute psychological stress and coronary artery disease" with great interest. With regard to the pathophysiology underlying mental stress-induced myocardial ischemia, the authors have implicated several factors including vascular mechanism, platelet/coagulation dysfunction and inflammatory changes as the principal pathophysiological mechanisms of the disease. It should be noted, however, that different types of stress-induced cardiac disorders may have different mechanisms of stress response. Viewing a stressful soccer match, for instance, may represent a triggering factor of plaque rupture and subsequent thrombosis,[2] whereas other mental stress situations (e.g. anger and anxiety) may induce endothelial dysfunction and coronary vasospasm.

The Tako-Tsubo cardiomyopathy, also known as apical ballooning syndrome, is a recently described emotional stress-related acute cardiac syndrome which was initially recognized and first reported in the Japanese population.[3] The syndrome is clinically characterized by chest pain, ischemic electrocardiographic changes, echocardiographic ventricular dysfunction and small elevation of cardiac troponin levels. The features described thus mimic acute myocardial infarction (AMI). However, there is no angiographic evidence of obstructive coronary artery disease. Since the ventricular dysfunction observed is not confined to the apical region of the heart, the term "Stress-induced cardiomyopathy" is more appropriate and should be used.[4] Another important feature of the syndrome distinguishable from AMI is the marked predominance of females in the advanced age. This can be attributed to reduced estrogen levels observed in post-menopausal women. In a rat model of emotional stress reported by Ueyama et al.,[5] estradiol supplementation attenuates the stress-induced changes in left ventricular function in ovariectomized female rats. This result may explain the protective effect of estrogen on the cardiovascular system involving the induction of vasodilatation through activation of endothelial nitric oxide synthase (NOS), and inhibition of the response of blood vessels to injury of the cardiomyocytes.[6] The pathophysiological mechanism underlying this type of stress-induced cardiomyopathy may thus include endothelial and microvascular dysfunction. The direct toxic effect of catecholamines on myocardial cells has also been described. Although in general regarded as a rare and benign condition, the syndrome should and must be considered in the differential diagnosis of acute coronary syndromes, since the treatment of stress-induced cardiomyopathy is different from that of atherosclerotic obstructive coronary heart disease.

Coronary atherosclerosis is a multifactorial disorder, with hypercholesterolemia/dyslipidemia, hypertension, smoking and type 2 diabetes mellitus being considered major coronary risk factors. It should, however, be appreciated that psychological stress which represents an integral part of our every day life may contribute substantially to the initiation and progression of atherosclerosis and, more importantly, may trigger acute coronary events and even deaths.[7] Thus, it is imperative that life style and behavior changes avoiding stress circumstances be seriously considered as effective means in the primary and secondary prevention of cardiovascular disease.

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