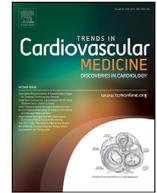




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Trends in Cardiovascular Medicine

journal homepage: www.elsevier.com/locate/tcmEditorial commentary: Cardiosexology – Let's talk about sex[☆]Dariusz Kałka, PhD^{a,b,*}^a Cardiosexology Unit, Department of Pathophysiology, Wrocław Medical University, ul. K. Marcinkowskiego 1; 50-368 Wrocław, Poland^b Centre for Men's Health in Wrocław, Poland

Erectile dysfunction (ED) is the inability of a male to achieve or maintain an erection of the penis sufficient to permit satisfactory sexual intercourse. At the Panel of the National Institutes of Health Consensus, they concluded that most of the disorders associated with ED are related to the arterial system, although, for 40%, ED is linked to vascular disorders rooted in atherosclerosis [1].

Direct relationships between ED and cardiovascular disease (CVD) discussed in this issue of the Journal by Orimoloye et al. exist [2]. Atherosclerosis is a systemic disease with the same pathophysiological model in the impairment of all arteries. ED results from endothelial dysfunction and atherosclerosis of penile arteries. The occurrence of ED in a 40–50-year-old man often indicates endothelial dysfunction and atherosclerosis throughout the entire arterial system exposed to the same destructive factors. It is worth remembering that ED may be the only symptom of CVD and may serve as an early indicator of developing atherosclerosis. This condition is determined by differences in the diameter of vascular vessels. Penile arteries are smaller (with a diameter of 1–2 mm) and more likely to narrow at an early stage of CVD. As a result, impairment in blood flow in penile arteries is more severe than in larger coronary vessels with a diameter of 4–5 mm. Possible causes of ED prevalence as a clinical manifestation of atherosclerosis of the coronary arteries include not only the small size of deep arteries of the corpora cavernosa but also because of their need to dilate up to 80%. Dilation at this level provides adequate blood flow into the corpora cavernosa, immediately increasing their volume approximately 20-fold and activating a veno-occlusive mechanism that leads to a rigid erection. The ability to dilate is much smaller in other parts of the arterial bed, only 10–20%. Thus, even the initial stages of atherosclerosis, in which typical atherosclerotic changes would not be noticed, are associated with significant worsening in vascular dilation and endothelial dysfunction, which may lead to the occurrence of ED. This association was identified based on epidemiological and clinical data showing that ED appears 2–5 years before the onset of CVD [3–5].

The high dependence of effective blood flow through the deep vessels of the corpora cavernosa and the helicine arteries on the function of the vascular endothelium is why interventions aimed

at improving endothelial function improve the quality of erection. One of these interventions is health-promoting physical activity. In patients with coronary artery disease and hypertension or hyperlipidemia who were subjected to regular cardiac training and had the greatest reduction in systolic blood pressure, the largest improvement in erectile function was observed, possibly via the improvement in endothelial function [6].

The assessment of the effectiveness of filling the corpora cavernosa with blood should not be limited to evaluations conducted during sexual activity. Nocturnal penile tumescence (NPT) is also worth consideration because it allows capturing disorders in men who engage in sexual activity occasionally, with breaks longer than 6 months between sexual intercourse. Spontaneous erections, which appear 3–4 times during REM sleep, are nothing but a type of “gymnastics” that keeps the erectile tissue in good shape. A lack of NPT results in long-term hypoxia, which leads to the loss of smooth muscle and an increase in collagen synthesis; as a consequence, fibrosis within the corpora cavernosa appears, along with increased synthesis of strongly vasoconstrictive endothelin ET1, and apoptosis [7].

The assessment of erectile function can help predict the course of disease in patients with already developed pathologies of the cardiovascular system. ED in patients with post-myocardial infarction (MI) damage of the heart muscle and a moderately decreased ejection fraction of the left ventricle is associated with significantly worse tolerance of effort both at baseline and after cardiac rehabilitation. Thus, ED may play a role in predicting worsening in tolerance of effort and optimizing cardiac rehabilitation. What is more, low tolerance of effort in MI patients indicates an increased risk of future cardiac events and all-cause mortality. For this reason, using the IIEF-5 questionnaire should be introduced into the routine clinical assessment of patients with complicated acute MI who have already qualified for cardiac rehabilitation [8,9].

Endothelial dysfunction and severe inflammation underlying ED increase risk of a prothrombotic condition. In 129 patients with CVD in which 58.9% had paroxysmal atrial fibrillation, the presence of ED significantly determined a greater score on the CHADS₂ and CHA₂DS₂-VASc scale used for the assessment of the risk of peripheral embolism occurring in atrial fibrillation. These scales are based on the identification of risk factors associated with ED and are well recognized in the literature. Thus, ED can be considered an addi-

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tional marker of a prothrombotic condition and may be useful in clinical decision making in men aged 65 and over [10].

Another element of the erection is the activity of the autonomic system. During erection, increases are observed in parasympathetic tone, the activity of non-adrenergic/non-cholinergic (NANC) fibres, and the secretion of nitric oxide, which serves as a neurotransmitter for NANC neurons within the corpora cavernosa. The autonomic balance also results in the proper regulation of the cardiovascular system. Balance disturbances play a role in morbidity and mortality due to cardiovascular causes. The autonomic balance, as measured by the variability of heart rate, has a recognized relationship with both the risk of sudden cardiac death and the presence of ED. Another method measures the reduction in heart rate during the first minute of the recovery phase in a stress test. It evaluates the activity of the autonomic system and indicates a significant relationship between improvements in the tone of the vagus nerve and increases in the IIEF-5 score in patients subjected to 6 months of cardiac training [11,12].

Both ED and CVD share common modifiable risk factors. A change in lifestyle is an effective way to fight ED. However, the level of knowledge on this topic among men is low. An analysis of 417 men with a mean age of 61.10 ± 9.15 who suffered from coronary artery disease and ED showed that their knowledge of the contribution of the classic risk factors to the development of CVD was significantly better than of the contribution of the same risk factors to the development of ED. It has been suggested that numerous campaigns to promote a healthy lifestyle and raise awareness of the influence of these factors on the development of ischemic heart disease should also include their impact on the development of ED [13].

It seems today, however, that the basic problem is rooted in not asking patients about their sexual activity. To improve this situation, involvement of the whole medical community is needed, especially physicians of specialties who have direct access to groups of patients particularly at risk of ED. The pathogenetic association between CVD and ED means that a significant percentage of ED patients visit cardiologists. It would be useful then to begin including a sexual function assessment into the tools for estimating CVD

risk, as well as adding ED to recommendations regarding standard assessments of patients with risk factors or with clinically overt CVD. The use of ED assessment in everyday clinical practice can not only improve patients' quality of life but, in many cases, even save their lives.

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