Editorial

Does erectile dysfunction improve the assessment of cardiovascular risk?

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Erectile dysfunction (ED) is defined as the inability to obtain or maintain sufficient penile rigidity for sexual satisfaction (1), which can create a psychological strain and become a distressing condition for men (2). It is estimated that ED affects 150 million men in the world and approximately 30 million in the United States alone (1).

ED has risk factors in common with cardiovascular disease (CVD), such as age, smoking, diabetes mellitus, hypertension, dyslipidemia, depression, obesity and a sedentary lifestyle (1). These factors are strongly associated with impaired vascular flow, endothelial dysfunction (linked to nitric oxide) and atherosclerosis (3,4) and, based on common physiopathological mechanisms of inflammation, create an association between ED and cardiovascular events (4). Endothelial cells are involved in the regulation of vascular tone and the process of atherosclerosis, exerting an influence on ED, CVD and peripheral vascular disease. The dysfunction of these cells results in a reduction in endothelium-dependent vasodilation (4) and leads to an increase in oxidative stress in tissues, which can cause ED (4). Also, endothelial dysfunction leads to vasoconstriction, atherosclerosis and thrombus formation, linking CVD and ED (4). Moreover, atherosclerosis is believed to affect vascular beds in a uniform manner. Thus, smaller caliber arteries, such as those of the penis, may be subject to occlusion prior to arteries with a larger caliber, which is a hypothesis regarding why men develop ED before suffering myocardial infarction (4).

Clinical and epidemiological evidence suggests that ED is linked to an increased risk for the occurrence of cardiovascular events and precedes the occurrence of symptoms by two to five years (1), making it an independent risk marker for future CVD (4). Indeed, a cohort study showed that ED is a predictor of cardiovascular events (5). There is also evidence suggesting that ED is associated with peripheral vascular disease and, considering its significant morbidity and mortality, it has been proposed that ED is a marker for subclinical CVD and atherosclerosis even in patients with no diagnosis of peripheral vascular disease (3). Many patients with ED have intermediate cardiovascular risk and may benefit from additional tests for a better stratification of risk with the aim of identifying subclinical CVD (2).

Systematic reviews have suggested an association between ED and markers of subclinical CVD, such as flow-mediated dilation and the thickness of the intima-media layer of the carotids, but this association is not yet fully clarified (1). A recent meta-analysis demonstrated that ED is associated with subclinical CVD in the majority of studies, suggesting a strong association between ED and both impaired endothelial function and carotid artery disease (3).

Endothelial dysfunction, which is an early marker of atherosclerosis, is also believed to be one of the earliest changes in individuals with ED (3). There is evidence that the detection of subclinical atherosclerosis may precede symptoms of vasculogenic ED (1) and an increase in subclinical atherosclerosis has been documented in patients who subsequently reported ED (5). Such findings may justify more aggressive preventive therapy for CVD in patients with ED (5) due to the increased risk for subclinical CVD (3), even if patients do not have other associated risk factors (as in the case of young individuals).

The evaluation of the temporal relationship between ED and subclinical CVD is of considerable importance in determining whether signs of subclinical CVD, such as a change in the coronary artery calcification score, may predict incident ED (1). The evaluation of risk markers that may precede the occurrence of ED and, therefore, encourage the early modification of risk factors is seen as crucial to the prevention of CVD (1).

Despite divergences of opinion regarding the role of ED in the prediction and assessment of cardiovascular risk, urologists have a critical function in the screening of patients, especially young patients with moderate to severe ED and no history of diabetes or cardiovascular events (2), who may be at high risk and should be sent to a cardiologist for evaluation. Cardiologists should recognize the importance of ED and can recommend the establishment of an assessment protocol (2). The involvement of urologists in the development of guidelines is highly encouraged aiming to improve the quality of care and general health of men with ED (2).

It is necessary to identify early, low-cost, noninvasive markers of CVD that can be used to evaluate endothelial dysfunction and inflammation to effect changes in modifiable risk factors (1,3,4). Even in cases of patient-reported ED with no diagnostic confirmation, ED has significant predictive value for cardiovascular events, which suggests that this condition can be used as a low-cost diagnostic tool for the stratification of risk (1).

The use of ED as a predictor of risk and guide for preventive therapy has encouraged the inclusion of its symptoms in the assessment of cardiovascular risk, such as the calculation of the QRISK-3 algorithm from the United Kingdom (1). Moreover, studies are needed to determine whether subclinical CVD precedes ED, since intervention in this phase could prevent both ED and clinical CVD (1).

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