Erectile dysfunction (ED) is now recognized as a marker of increased cardiovascular risk both acutely and chronically and considered an early manifestation of generalized vascular disease predicting all-cause mortality, cardiovascular mortality, coronary events, stroke, and peripheral artery disease in men with and without known coronary artery disease. Importantly, ED shares with coronary artery disease similar risk factors and is principally vasculogenic, reflecting the common denominator of endothelial dysfunction. ED is defined as the inability to obtain or maintain a penile erection to support satisfactory sexual performance, and when affecting men with and without cardiac symptoms in the age group 30 to 60 years seems to be a significant predictor of subsequent cardiovascular events.

Vlachopoulos et al have over several years studied the independent link between ED and cardiovascular disease (CVD) using biomarkers as a means of identifying the men most at risk of a cardiovascular event. Their latest contribution to the expanding and important literature identifies aortic stiffness as a marker of increased cardiovascular risk in men presenting with ED (this issue). Previously, the same workers have identified the unfavorable effect on the circulating levels of biomarkers when ED is combined with coronary artery disease. Importantly, the clinical implications of their observations (treated ED and treating cardiovascular risk) may, with the findings of others, not only improve life (by treating the ED) but save lives (by reducing the cardiovascular risk). In this study, they build on previous experience with unfavorable endothelial and inflammatory markers in patients with ED with or without coronary artery disease using aortic stiffness as an independent predictor of all-cause and cardiovascular mortality in patients with hypertension. Using aortic pulse wave velocity, they independently predicted major cardiovascular events and on long-term follow up demonstrated the usefulness of pulse wave velocity in identifying low-risk patients. Of particular importance was the identification of cardiovascular risk in the young patient in whom cardiovascular risk reduction has the greatest potential benefit. This observation is supported by 3 reviews, all basically saying the same thing. In the analysis by Dong et al including 12 prospective cohort studies involving 36,744 participants, it was suggested that ED independent of other risk factors significantly increased the risk of CVD, coronary artery disease, stroke, and all-cause mortality.

Similar findings were reported by Vlachopoulos et al. They evaluated 92,757 participants and noted that ED was associated with increased cardiovascular events and all-cause mortality, and importantly the risk was at its highest in the younger age group where the focus of risk reduction should be targeted, a finding in agreement with the overview by Miner et al. The most recent review by Gandaglia et al who produced a systematic review of the association between ED and CVD concluded that ED and CVD should be regarded as 2 different manifestations of the same systemic disorder with ED usually preceding CVD and ED should, therefore, be considered an early marker for CVD of particular importance in the asymptomatic younger men and in those with diabetes mellitus.

In this publication, Vlachopoulos et al have identified a useful role for vascular markers, particularly in younger men, and this includes an assessment of aortic stiffness, as well as various alternative biomarkers. An alternative strategy has been to suggest that the early use of multidetector cardiac computed tomography and action based on coronary anatomy may be a further means of identifying the man with silent ischemia at coronary risk, given that exercise electrocardiography is unreliable in detecting early plaque disease.

In a series of 65 men referred for treatment of ED and who had no cardiac symptoms, multidetector cardiac computed tomography was performed on all of the men as an outpatient procedure and identified normal coronary arteries in only 5 men. Interestingly, of those 65 men, 12 were hypertensive and 60 had elevated low-density lipoprotein cholesterol. The nature and severity of the asymptomatic coronary disease process were such that 5 men received coronary stents and 4 men underwent coronary artery bypass surgery.

There is no doubt that the men with ED aged 30 to 60 years are at the greatest risk of having undetected silent coronary ischemia, and it may well be that a combination of biomarkers, vascular stiffness, and multidetector cardiac computed tomography is the best way to investigate this individual. It remains important because the risk is evaluated to remind the cardiologist in particular that it is within their remit, and the general physician who may have an interest in hypertension who detects a patient with ED (asking routinely) should immediately trigger an evaluation of cardiovascular risk even when symptoms of a cardiovascular nature are not present. We need to incorporate the question of ED in all our cardiovascular screens.

The opinions expressed in this editorial are not necessarily those of the editors or of the American Heart Association.

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By emphasizing the ED/CVD link, the prediction and prevention of cardiovascular events and all-cause mortality may be reduced by their early recognition and treatment. Routinely asking the man at risk about ED can improve the quality of life by treating the ED and lengthening life by treating the risk of coronary artery disease. It is perhaps the most cost-effective screening weapon we have.

Vlachopoulos et al. have once more identified cardiovascular risk attributable to endothelial dysfunction in patients with ED and suggested that this may be easily detected by the use of conventional biomarkers including pulse wave velocity.

In addition to the acceptance of ED being a marker of increased cardiovascular risk, as a means of identifying risk and treating it as a randomized comparative study of using biomarkers as an alternative to multidetector cardiac computed tomography is needed. We are all seeking the same solution that is the reduction of cardiovascular risk. The best way of achieving this in the man with ED needs to be determined.

Disclosures

None.

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Erectile Dysfunction: A Marker of Increased Cardiovascular Risk
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