

Erectile dysfunction: a marker of silent coronary artery disease

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This editorial refers to 'Association between erectile dysfunction and coronary artery disease. Role of coronary clinical presentation and extent of coronary vessels involvement. The Cobra Trial' by P. Montorsi *et al.*, doi:10.1093/eurheartj/ehl142

Erectile dysfunction (ED) is common affecting over 50% of men aged 40–70 years.¹ It increases with age so that men over 70 years (with a prevalence of 70%) have three times the incidence of men in their 40s. It is an important cause of relationships breaking down with the man losing self-esteem, feeling a failure, and the partner feeling rejected so that the man's problem becomes a couple's frustration and concern. With the development of the phosphodiesterase type 5 inhibitors has come a greater understanding of the mechanisms responsible and the important role of endothelial dysfunction, and hence vascular disease, as the major cause of ED.² Though the commonest cause is now recognized to be organic, psychological consequences will result and both aspects need to be addressed.

With the recognition that endothelial dysfunction is the common denominator linking vascular disease to ED came the realization that ED may not just be a consequence of vascular disease, especially coronary disease, but a harbinger of silent coronary disease—a 'sentinel'.³ The smaller penile arteries (1–2 mm in diameter) potentially suffer from plaque burden and/or endothelial dysfunction symptomatically earlier than the larger coronary (3–4 mm), carotid (5–7 mm), and iliofemoral (6–8 mm) arteries and as a consequence, ED may act as a marker for silent CAD and therefore precede a coronary event.^{3,4} As acute coronary syndromes are invariably due to the rupture of a previous subclinical plaque (often in the presence of only single vessel disease) ED could be a predictor of an acute event as well as being a manifestation of more advanced chronic disease. This concept is reinforced by ED and vascular disease sharing the same risk factors—diabetes, smoking, hypertension hyperlipidaemia, obesity depression, and lack of physical activity.⁴

Several studies have established the link between ED and silent vascular disease and the COBRA study compares the

acute with the chronic condition.⁵ Previous work by Montorsi *et al.*⁶ identified ED as a predictor of acute coronary syndromes with an ED prevalence of 49% (147 of 300 patients). Importantly, of those with ED, 99 (67%) developed ED on an average 3 years beforehand, thereby reinforcing the theory that ED might predict subclinical vulnerable plaque in the coronary tree. Montorsi *et al.* now demonstrate age, multivessel CAD, and chronic coronary syndromes to be independent predictors of ED and in those with an acute presentation, the prevalence of ED was linked to the extent of CAD—again ED coming before the CAD presentation in the majority by 2–3 years.

Moderate-to-severe but not mild ED in a health screening project was calculated to increase the 10 year relative risk of developing CAD by 65% and stroke by 43%.⁷ In an angiographic study, 9 of 47 men (19%) with ED had silent CAD.⁸ In type 2 diabetics, a comparison between men with and without ED recorded the presence of ED in 33.8% of 133 men with asymptomatic CAD and only 4.7% without CAD.⁹ In this diabetic study, ED independently predicted CAD and the authors highlighted the potential value of stress testing diabetic men with ED and no cardiac symptoms for risk assessment. The Prostate Cancer Prevention Trial randomized 9457 men to placebo and prospectively assessed them at three monthly intervals for cardiovascular disease and ED from 1994 to 2003.¹⁰ ED significantly predicted cardiac events with a hazard ratio of 1.45 ($P < 0.001$) for those with ED at entry or developing ED during the study.

The Second Princeton Consensus Conference focused on sexual dysfunction and cardiac risk and concluded that ED is a warning sign of vascular disease with the practical recommendation that a man with ED and no cardiac symptoms is a cardiac or vascular patient until proved otherwise.⁵ As men with ED may be at cardiovascular risk, the possibility arises that aggressive risk reduction may reduce the chance of or prevent subsequent vascular events. Given the findings in this latest publication and the average 2–3 year lead time between ED and silent CAD becoming symptomatic, there is potentially time to prevent cardiac events occurring. As family doctors and urologists rather than cardiologists first see these men, they have a pivotal role to play. Cardiologists need to be actively involved not only in detecting and treating ED in their own patients, but in working with their urological colleagues to develop a multidisciplinary management plan. ED is a distressing problem which should

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be asked about routinely, given the successful treatment options available. As it is now widely recognized to be a warning sign for silent coronary and vascular disease, a public awareness education programme is needed to encourage men (preferably with their partners) to seek help early.

All men with ED and no cardiac symptoms need a detailed cardiac assessment, measurement of blood pressure, fasting lipid profile and glucose, as well as lifestyle advice regarding weight and exercise. Those at cardiovascular risk ideally need stress testing and referral for risk reduction therapy, and advice with appropriate follow-up. We need to think of ED as standing for Erectile Dysfunction, Endothelial Dysfunction, and Early Detection. Just as there is more to sex than an erect penis, there is more to ED prevention than simply restoring an erection.

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