Erectile dysfunction: a warning sign of silent vascular disease

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Erectile dysfunction (ED) is the inability to achieve and/or maintain an erection sufficient to permit satisfactory sexual intercourse [1]. ED is nowadays a common condition affecting approximately 5–20% of adult men; ED has a considerable effect on the quality of life not only of the patients themselves, but also of their spouses/family [2–4]. This Editorial discusses ED as a manifestation/warning sign of clinically silent cardiovascular disease. It does not discuss ED due to other causes (for instance drugs, depression, etc).

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A direct association between ED with cardiovascular disease has been supported [2–6]. For example, almost one in two stroke patients report some degree of ED, which is demonstrated as a diminished post-stroke libido and sexual dysfunction compared with before the episode [3, 7]. A recent study showed that stroke patients had a >3-fold increased risk of reporting ED compared with nonstroke patients (odds ratio [OR], 3.30; 95% confidence interval [CI], 1.22–8.88) [8]. ED is thus an important problem in stroke patients with considerable psychosocial consequences [9].

Another study including 690 male patients who had been referred for stress testing, without diagnosed peripheral arterial disease (PAD) showed that ED is a marker of previously undiagnosed PAD [10]. ED was present in 45%, while PAD was present in 23% of the patient group. More men with ED had concomitant PAD compared with patients without ED (32 vs. 16%, respectively; $P < 0.01$). On multivariate logistic regression analysis, ED was an independent predictor of PAD (OR, 1.97; 95% CI, 1.32–2.94; $P = 0.002$). Therefore, ED may identify men who would benefit from screening using the ankle–brachial pressure index [10].

The prevalence of carotid and/or lower limb artery atherosclerosis was investigated in patients with ED [11]. The incidence of simultaneous penile arterial insufficiency and carotid and/or lower limb artery abnormalities was higher compared with that found in patients with ED of nonarterial organic or

psychogenic origin. Penile artery insufficiency-caused ED was associated with carotid and/or lower limb artery ultrasound abnormalities in approximately three out of four patients [11]. Finally, a retrospective cohort study including patients from a large (>17 million patients) patient database, the Integrated Healthcare Information Services National Managed Care Benchmark Database (IHCIS), showed that after adjustment for age at ED diagnosis, smoking, obesity and use of angiotensin converting enzyme inhibitors, beta-blockers and statins, patients with ED ($n = 12,825$) had a 75% increased risk for the concomitant presence of PAD (OR, 1.75; 95% CI, 1.06–2.90) [12]. Patients aged 40–44 years were >2 times more likely to develop PAD compared with men aged 30–39 years (OR, 2.07; 95% CI, 0.89–4.81), whereas patients aged 50–55 years had a 3-fold increased risk of developing PAD (OR, 3.00; 95% CI, 1.40–6.43). The results of this study indicated that ED may serve as a marker for PAD and that the risk becomes more pronounced with increasing age, indicating the need for cardiologists and internists to monitor ED patients who may not necessarily present with cardiovascular symptoms [12].

In another study it was demonstrated that almost 70% of the patients attending a vascular surgery outpatient clinic have moderate or severe ED [13]. Increased age, abdominal aortic aneurysm and PAD were strongly associated with the presence of ED [13]. It was shown that restoration of normal flow in PAD may not only produce local benefit due to improved blood perfusion, but may also correct/improve several systemic conditions that may appear unrelated, yet share a common etiology of autonomic dysfunction, such as sleep disorders, ED, bowel dysfunction and depression [14]. Given the systemic nature of atherosclerosis, all vascular beds may be affected to some extent; yet symptoms rarely become evident at the same time. According to the artery size hypothesis [15], this variability in symptoms may be caused by the different size of arteries supplying the different vascular beds. This allows a larger vessel to better tolerate atherosclerotic stenosis compared with a smaller one. Thus, because penile arteries are smaller in diameter than coronary arteries, patients with ED will seldom have concomitant symptoms of coronary heart disease (CHD), whereas patients with CHD will frequently complain of ED [15]. Furthermore, ED is a frequently missed comorbidity when

these patients undergo coronary artery bypass surgery [16].

Several treatment modalities for the management of ED have been reported [17–20]. The initiation of intensive lifestyle measures should comprise the mainstay of the therapeutic approach; these include risk factor improvement (e.g., use of lipid-lowering [statins] and antihypertensive agents), smoking cessation, adoption of a healthy diet, weight loss and increased physical activity [4]. ED shares common risk factors (diabetes, overweight, hypertension, smoking, high total cholesterol and low high-density lipoprotein cholesterol) with vascular disease. Improving vascular risk factors may decrease the incidence of ED, as well as CHD risk [4, 21]. The recognition of ED as a warning sign of silent vascular disease has led a recent consensus to express the concept that a man with ED and no symptoms of cardiovascular disease is in fact a cardiovascular patient until proven otherwise [22]. The presence of ED in an otherwise “asymptomatic” patient should therefore be considered as a potential warning sign and trigger the initiation of measures for the detection of an underlying cardiovascular disease.

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