

Erectile dysfunction as a marker of CVD



Michael Kirby

Vascular disease is thought to be the most common physical cause of erectile dysfunction (ED), accounting for around 80% of cases (Chiurlia et al, 2005), and increasing evidence suggests that ED may be an early warning sign of more widespread vascular disease.

Endothelial dysfunction is a marker of preclinical vascular disease that may occur many years before the appearance of clinical vascular disease (Faulx et al, 2003; Chiurlia et al, 2005). Studies have shown that in otherwise healthy men, and especially those with type 2 diabetes, ED may accompany early signs of coronary artery disease (CAD) that are not detectable during stress testing (Montorsi et al, 2003; Jackson and Padley, 2008; Ma et al, 2008).

The fact that ED is often the first clinical sign of endothelial dysfunction, preceding other manifestations of systemic atherosclerosis such as CAD, may be largely attributable to the small size of the penile arteries (Montorsi et al, 2005; Lojanapiwat et al, 2009). The penile arteries are typically 1–2 mm in diameter, while the coronary arteries are around 3–4 mm and the carotid arteries are typically 5–7 mm (Inman et al, 2009). Endothelial dysfunction results in cellular proliferation, vasoconstriction and a proinflammatory environment with plaque destabilisation (Rodriguez et al, 2005). Because of their smaller size, atherosclerotic plaques are likely to occlude the penile arteries first (Inman et al, 2009). This is the reason why individuals with recent onset ED will seldom have concomitant symptoms of CAD, but those with CAD will frequently report ED (Montorsi et al, 2005).

ED severity appears to be related to the degree of atherosclerosis (Greenstein et al, 1997; Solomon et al, 2003), and men with multi-vessel CAD are more likely to have severe ED than those with single vessel disease (31% vs 12.5%, respectively, $P<0.001$; Montorsi et al, 2006). Studies have also shown that men with ED tend to develop more severe CAD (Montorsi et al, 2006) and left ventricular dysfunction (ejection fraction $<40\%$ and $<50\%$ in two studies) than

those without ED (Min et al, 2006; Ward et al, 2008). In a study by Min et al (2006) ED was associated with shorter exercise time (8.0 vs 10.1 minutes; $P<0.001$) and lower Duke treadmill scores (4.4 vs 8.4; $P<0.001$).

ED precedes the onset of CAD in about two-thirds of men (Montorsi et al, 2003). Studies have shown that ED symptoms may predate the occurrence of CAD symptoms by 2–3 years (Montorsi et al, 2006), while the interval between onset of ED symptoms and the occurrence of a vascular event may be up to 5 years (Baumhäkel and Böhm, 2007; Hodges et al, 2007) or longer (Chew et al, 2010).

Although ED is more likely to develop in older men, recent research shows that the significance of ED as a predictor of cardiovascular disease (CVD) risk appears to wane as men get older (Inman et al, 2009; Chew et al, 2010). While the adjusted risk of future CVD was strikingly high in men with ED in their 40s, it reduced with age and was negligible for men of 70 years or over (Inman et al, 2009). A similar trend was seen in the study by Chew et al (2010).

The increasing awareness of ED as a barometer for cardiovascular health, represents an opportunity to improve primary prevention of vascular disease and cardiovascular events both in men with and men without diabetes. However, men are notoriously reticent about seeking help for sexual problems, and this was highlighted in a study investigating the relationship between ED and CVD in 372 people from GP practices across the UK. Results showed that in almost 50% of men with ED, there were missed opportunities to perform CVD risk assessment and provide intervention, because the men did not acknowledge or discuss the fact that they had a problem (Hodges et al, 2007). Although things may have improved since then, this highlights the need for healthcare professionals to be proactive in enquiring about erectile function with males aged 40 years and over, when they present for other reasons. It is essential that it is on the diabetes template for routine discussion and a trigger for optimisation of risk factor control. ■

- Baumhäkel M, Böhm M (2007) *Int J Clin Pract* **61**: 361–6
- Chew KK, Finn J, Stuckey B et al (2010) *J Sex Med* **7**: 192–202
- Chiurlia E, D'Amico R, Ratti C et al (2005) *J Am Coll Cardiol* **46**: 1503–6
- Faulx MD, Wright AT, Hoit BD (2003) *Am Heart J* **145**: 943–51
- Greenstein A, Chen J, Miller H et al (1997) *Int J Impot Res* **9**: 123–6
- Hodges LD, Kirby M, Solanki J et al (2007) *Int J Clin Pract* **61**: 2019–25
- Inman BA, Sauver JL, Jacobson DJ et al (2009) *Mayo Clin Proc* **84**: 108–13
- Jackson G, Padley S (2008) *Int J Clin Pract* **62**: 973–6
- Lojanapiwat B, Weerusawin T, Kuanprasert S (2009) *Singapore Med J* **50**: 698–701
- Ma RC, So WY, Yang X et al (2008) *J Am Coll Cardiol* **51**: 2045–50
- Min JK, Williams KA, Okwuosa TM et al (2006) *Arch Intern Med* **166**: 201–6
- Montorsi F, Briganti A, Salonia A et al (2003) *Eur Urol* **44**: 360–4
- Montorsi P, Ravagnani PM, Galli S et al (2005) *Am J Cardiol* **96**: 19M–23M
- Montorsi P, Ravagnani PM, Galli S et al (2006) *Eur Heart J* **27**: 2632–9
- Rodriguez JJ, Al Dashti R, Schwarz ER (2005) *Int J Impot Res* **17**(Suppl 1): S12–8
- Solomon H, Man JW, Wierzbicki AS, Jackson G (2003) *Am J Cardiol* **91**: 230–1
- Ward RP, Weiner J, Taillon LA et al (2008) *Am J Cardiol* **101**: 502–5

Michael Kirby is Visiting Professor to the Faculty of Health and Human Sciences at the University of Hertfordshire, and Visiting Professor to the Prostate Centre, London. He was previously a GP in Letchworth, Hertfordshire.