ADITUM
Enrich your Research

International Journal of Epidemiology and Public Health Research

Review Article

Erectile Dysfunction (ED) As A Link To Heart Disease

Aksam Yassin[1,2,3*], Hatem Kamkoum[1], Raidh Talib Alzubaidi[1,2], Hasan Abdallah[1], Anas Albudairat[1], Ahmad Ramadan[4], Abdelaziz Saleh[1], Abdulla Al-Ansari1[2]

[1]Hamad Medical Corporation, HMC Aisha Al Attiyya Hospital, Andrology & Men's Health Unit, Qatar.

[2]Weill Cornell Medical School NY, Qatar.

[3]Dresden International University, Preventive Medicine Program, Dresden Germany.

[4]Pharmacy Department, HMC Aisha Al Attiyya Hospital, Qatar.

Article Information

Received: November 20, 2025
Accepted: November 28, 2025
Published: December 01, 2025

***Corresponding author:** Prof. Aksam Yassin MD PhD EdD, Hamad Medical Corporation, Aisha Al Attiyya Hospital, Andrology & Men's Health Unit, Qatar.

Citation: Aksam Yassin, Hatem Kamkoum, Raidh Talib Alzubaidi, Hasan Abdallah, Anas Albudairat, Ahmad Ramadan, Abdelaziz Saleh, Abdulla Al-Ansari., (2025) "Erectile Dysfunction (ED) As A Link To Heart Disease". International Journal of Epidemiology and Public Health Research, 7(5); DOI: 10.61148/2836-2810/IJEPHR/183.

Abstract

Introduction:

Erectile dysfunction (ED) and coronary artery disease (CAD) overlap in risk factors, etiology and clinical outcomes. It has become clear that ED is an important marker of vascular disease throughout the arterial tree – including CAD, stroke and diabetes. Epidemiological studies have demonstrated a close association between ED and vascular disease. The shared etiological factor is endothelial dysfunction.

The fact that ED tends to precede the onset of symptoms of other vascular diseases – because blood vessels in the penis are narrower in diameter than elsewhere in the body, so blood flow is restricted sooner by atherosclerosis – means that it can be used as a 'window' on vascular health. There is growing evidence that patients presenting with ED should be investigated for cardiovascular disease (CVD), including diabetes, even if they have no symptoms. Early detection could facilitate prompt intervention and a reduction in long-term complications. Treatments that reduce endothelial dysfunction offer the potential of improving the functioning of the entire vascular system, improving outcomes in CVD and diabetes, as well as providing effective treatment for ED.

Keywords: Cardiovascular disease; endothelial dysfunction, erectile dysfunction (ED)

Introduction

There is growing evidence that ED is an early sign of cardiovascular disease (CVD). ED is an important marker of vascular disease throughout the arterial tree including coronary artery disease (CAD), stroke and diabetes. Epidemiological studies have confirmed that there is a close association between ED and vascular disease. A recent study in 76 men with chronic stable CAD showed that 75% had ED, to some degree (1). The risk factors for ED –hypertension, smoking, abnormal lipid profile and diabetes – are also risk factors for CAD. The relative risk of ED increases 1.5- fold with hypertension and doubles with hyperlipidemia. The Massachusetts Male Ageing Study, a random-sample cohort study of men aged 40–70, investigated the relationship between baseline risk factors for coronary heart disease (CHD) and subsequent ED, on the premise that subclinical arterial insufficiency might be manifested as ED. Results in 513 men with no ED at baseline revealed that cigarette smoking almost doubled the risk of ED over 8–10 years of follow-up (24 vs. 14%, adjusted for age and covariates). Overweight (body mass index (BMI): >28 kg/m2) and a composite coronary risk score also significantly predicted incident ED.

Associations were also seen for hypertension and dietary intake of cholesterol and saturated fat (2). Additionally, looking at CHD risk factors measured in mid-life could predict the incidence of ED an average of 25 years later. A study, which assessed seven classic CHD risk factors in community dwelling men aged 30–69 from 1972 to 1974 and then again in 1998, found that mean age, BMI, cholesterol and triglycerides were each significantly associated with an increased risk of ED (3). Another study in 215 patients with ED showed that the prevalence of hypercholesterolemia was significantly higher than in matched controls without ED. Nearly three-quarters (70.6%) of the men with ED had hypercholesterolemia (total cholesterol >5.17 mmol/l), compared to 52% of those without ED (p50.06). After exclusion of confounding factors, logistic regression analyses showed that HDL cholesterol and total cholesterol/HDL-cholesterol ratio were significant predictors of ED. Increased 10-year CHD risk was found in more than half (56.6%) of patients with ED, compared to 32.6% in the control group ($p<0.05$). The researchers concluded that hyperlipidemia was common in men with ED, and their risk of later developing CHD was higher than in those without ED, so they suggested that ED might serve as a sentinel for CHD (4). A prospective study showed that one in four men with ED (aged 40–69) but without known CAD will develop symptomatic CAD in the subsequent 12 years (5). ED is particularly common in patients with diabetes, which triples the risk of ED. The United Kingdom Prevention of Diabetes Study (UKPDS) revealed that 20% of patients had ED at the time their diabetes was diagnosed (6).

Further studies have indicated that between 20 and 85% of men with diabetes develop ED (7). One study of men presenting with ED showed that 17% had previously diagnosed diabetes, 4.7% were previously undiagnosed and 12% had impaired fasting glucose (8). Glycemic control has been shown to correlate with ED – the poorer the glycemic control, the more severe the ED (7).

AETIOLOGICAL Association Between ED And CVD:

ED and CAD overlap in risk factors, pathological basis of disease and disease progression. The common underlying factor is endothelial dysfunction. Endothelial dysfunction measured as impaired vasodilation is prognostic for cardiovascular events, such as myocardial ischemia and stroke (9,10). The endothelium is the single layer of cells that line the luminal surface of blood vessels. Over the past few years, it has become increasingly apparent that it is far more than just a structural lining, with a range of important physiological functions. It acts as a direct interface between the components of circulating blood and local tissue, and regulates numerous local blood vessel functions, including vascular tone, cell adhesiveness, coagulation, inflammation and permeability (11).

The vascular endothelium plays an obligatory role in vasodilation. The role of the arterial vasculature is to provide adequate tissue perfusion and buffer changes in blood pressure and flow that occur with each cardiac cycle and during normal activities. At the tissue capillary level, blood flow remains relatively constant despite rapid changes in the tone of arterioles and venules. Regulation of flow depends on the ability to change the resistance of the vascular bed. The endothelium produces and responds to several potent, locally active mediators.

The most important of these is nitric oxide (NO) – a simple, highly reactive gas. Endothelial NO possesses potent anti-atherogenic properties, in addition to inhibiting platelet aggregation and regulating vascular tone (12). Bioavailable NO can be increased by enhancing its production or reducing its inactivation. NO is also protected against atherosclerosis by other mechanisms. Leucocyte adhesion, an inflammatory process contributing to plaque instability and rupture, is inhibited by NO. NO also has anti-thrombotic properties mediated through the inhibition of platelet aggregation and adhesion. Impaired endothelial function is associated with impaired platelet activation. NO induces endothelial vasodilation by increasing the cyclic guanosine monophosphate content of vascular smooth muscle cells, resulting in relaxation. Defects in the production or action of endothelial NO may explain the increased atherosclerosis seen in diabetes (13). Endothelial dysfunction plays a key part in pathogenesis and progression of atherosclerosis (14) and has been implicated as an early functional alteration that precedes structural changes of the vasculature. Cardiac risk factors – including dyslipidemia, hypertension and type 2 diabetes – are all associated with impaired endothelial function (13).

Downregulation of endothelial NO synthase (eNOS) with disease results in reduced bioavailability of NO which results in endothelial dysfunction. Hyperglycemia may additionally impair endothelial function by promoting release of free radicals. One potent free radical, superoxide anion, inactivates NO resulting in the production of peroxynities – a potent oxidant that stimulates the production of vasoconstrictor prostanoids. NO production is also reduced in the presence of free radicals. Oxidative stress leads to the upregulation of anti-oxidation enzymes, such as superoxide dismutase, producing hydrogen peroxide. Overall, a reduction in NO results in endothelial dysfunction (13).

ED As A Surrogate Marker For CAD:

There is growing evidence that ED can be considered as a useful surrogate marker for CAD, with studies showing that a large proportion of men develop the problem before cardiac symptoms become evident. One study showed that ED symptoms occurred before symptoms of CAD in 67% of men in a consecutive series presenting with chest pain and angiographically documented CAD. Note, all patients with type 1 diabetes and ED developed sexual dysfunction before CAD onset ($p<0.001$) (15).

The obvious reason why ED may present before other signs of CVD is because penile artery diameter is smaller (1–2mm) than the coronary artery (3–4mm) or carotid artery (5–7mm), so symptoms associated with atherosclerosis occur sooner (16). The degree of plaque sufficient to cause ED – blocking blood flow through the penile artery – causes only 30–40% stenosis in arteries of greater diameter. Detecting atherosclerosis this early stage could potentially prevent life-threatening events by facilitating appropriate intervention. Several studies have demonstrated that cardiovascular assessment of men with ED enables the detection of CVD. One study showed that ED presented well before anginal symptoms of CAD in 67% of men with a mean time interval of almost 3 years (15,16). Another study revealed that 45% of men with ED had previously undiagnosed hyperlipidemia, 5% had undiagnosed diabetes and 7% had undiagnosed hypertension (17).

ED as A Sentinel For Type 2 Diabetes:

ED is a very clear sentinel for type 2 diabetes and patients presenting with ED should be tested for diabetes. One study

estimated that 12% of patients with diabetes were diagnosed during the evaluation of ED (19). Another study of 129 men presenting with ED revealed that 17% had previously diagnosed diabetes, 4.7% had previously undiagnosed diabetes and a further 12% had impaired fasting glucose. Disturbingly, urine dipstick testing would have missed 80% of these (8). The UKPDS clearly illustrated the potential for preventing complications by earlier diagnosis – in addition to 20% having ED, 35% had hypertension, 1% had an MI, 18% had abnormal ECG and 21% had retinopathy (20). Gazzaruso et al. also evaluated the presence of ED in 133 uncomplicated type 2 diabetic men with angiographically verified silent CAD and in 127 type 2 diabetic men without myocardial infarction at exercise ECG, 48-h ambulatory ECG and stress echocardiography. The findings showed a strong, independent association between ED and silent CAD in apparently uncomplicated type 2 diabetic patients (21).

There is a clear rationale for detecting diabetes as early as possible, because this has been shown to offer the potential for preventing long-term complications. If a patient with impaired fasting glucose or undiagnosed diabetes could be picked up earlier by checking glucose levels in men presenting with ED, it would be possible to achieve prompt control of glucose levels and make efforts to correct hypertension, abnormal lipid levels and reduce body weight, where appropriate. Patients could also be encouraged to make lifestyle changes, including reducing weight and increasing exercise levels, to reduce the risk of progressing to type 2 diabetes. Earlier detection would also be cost-effective, as 50% of the total costs of diabetes have been estimated to be the result of complications (22).

Practical Ways Of Detecting CVD And Diabetes In Patients With ED:

The clear evidence for ED as an 'early warning sign' or subclinical marker of CVD, including diabetes, supports improved efforts to find patients with ED and then to investigate them further for CVD. The possibility that patients might have silent CVD should be considered – and investigated – in men who present with ED, particularly when they have other cardiovascular risk factors (23). Primary care practitioners and nurses have a particularly important role in achieving early detection of ED by asking patients about the problem and then requesting further investigations into diabetes and CVD. Models in secondary care – such as male cardiovascular health clinics – have illustrated the feasibility and importance of considering ED and CVD at the same time. Patients referred to a urologist for investigation of ED could also be checked for signs of early cardiac disease.

Can We Treat ED In The Same Way As CVD?

Treatments that reduce damage to the endothelium will provide protection throughout vasculature, with potential benefits for preventing CVD as well as treating ED. A study in 400 hypertensives (mean BP: 154/94mmHg) treated with a range of antihypertensives showed that patients who showed improved flow-mediated dilation – indicating improved endothelial function – suffered fewer events (27). Treatments that can restore endothelial dependent vasodilation include antioxidants. Improvement has also been seen with L-arginine, although results have been variable. In the coronary circulation, statins improve endothelial function. Statins achieve multiple effects, in addition to reducing cholesterol. They increase NO formation in endothelial cells and increase eNOS expression. They also have anti-inflammatory effects (28–30).

Treatments that reduce damage to the endothelium will improve the entire vascular system – including the cardiovascular system and the vascular system of the penis – thus potentially improving both CVD and ED. Research has shown that PDE-5 inhibitors, such as sildenafil citrate, improve endothelial dysfunction significantly (31–33). It would be expected that this would reduce CVD, as well as providing effective treatment for ED (34). In addition, a less explained effect of PDE-5 inhibitors is that, in patients with diabetes, treating with a PDE-5 inhibitor to restore nocturnal erections and controlling other risk factors achieves a better clinical response in those patients achieving improved control of hypertension and lipid profile (35, 36, 37, 38).

References:

1. Kloner RA, Mullin SH, Shook T et al. Erectile dysfunction in the cardiac patient: how common and should we treat? J Urol 2003; 170: S46–50 [Discussion S50].
2. Feldman HA, Johannes CB, Derby CA et al. Erectile dysfunction and coronary risk factors: prospective results from the Massachusetts male aging study. Prev Med 2000; 30: 328–38.
3. Fung MM, Bettencourt R, Barrett-Connor E. The Rancho Bernardo Study. Heart disease risk factors predict erectile dysfunction 25 years later. J Am Coll Cardiol 2004; 43: 1405–11.
4. Roumeguere T, Wespes E, Carpentier Y et al. Erectile dysfunction is associated with a high prevalence of hyperlipidaemia and coronary heart disease risk. Eur Urol 2003; 44: 355–9.
5. Speel TGW, van Langen H, Meuleman EJH. The risk of coronary heart disease in men with erectile dysfunction. Eur Urol 2003; 44: 366–71.
6. Manley SM, Meyer LC, Neil HAW et al. Complications in newly diagnosed type 2 diabetic patients and their association with different clinical and biochemical risk factors. Diabetes Res 1990; 13: 1–11.
7. Romeo JH, Seftel AD, Madhun ZT, Aron DC. Sexual function in men with diabetes type 2: association with glycemic control. J Urol 2000; 163: 788–91.
8. Sairam K, Kulinskaya E, Boustead GB et al. Prevalence of undiagnosed diabetes mellitus in male erectile dysfunction. BJU Int 2001; 88: 68–71.
9. Schachinger V, Britten MB, Zeiher AM. Prognostic impact of coronary vasodilator dysfunction on adverse long-term outcome of coronary heart disease. Circulation 2000; 101: 1899–906.
10. Suwaidi JA, Hamasaki S, Higano ST et al. A long-term followup of patients with mild coronary artery disease and endothelial dysfunction. Circulation 2000; 101: 948–54.
11. Kharbanda RK, Deanfield JE. Functions of the healthy endothelium. Coron Artery Dis 2001; 12: 485–91.
12. Vane JR, Anggard EE, Botting RM. Regulatory functions of the vascular endothelium. N Engl J Med 1990; 323: 27–36.
13. Browne D, Meeking D, Shaw K et al. Endothelial dysfunction and pre-symptomatic atherosclerosis in type 1 diabetes –

pathogenesis and identification. Br J Diabetes Vasc Dis 2003; 3: 27–34.
14. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. Nature 1993; 362: 801–9.
15. Montorsi F, Briganti A, Salonia A et al. Erectile dysfunction prevalence, time of onset and association with risk factors in 300 consecutive patients with acute chest pain and angiographically documented coronary artery disease. Eur Urol 2003; 44: 360–5.
16. Montorsi P, Montorsi F, Schulman CC. Is erectile dysfunction the 'tip of the iceberg' of a systematic vascular disorder? Eur Urol 2003; 44: 352–4.
17. Billups K, Friedrich S. Assessment of fasting lipid profiles and Doppler ultrasound testing in men presenting with ED and no other medical problems. J Urol 2000; 163: 147. Also presented at the 95th annual meeting of American Urology association, Atlanta, USA.
18. Kaiser DR, Billups K, Mason C et al. Impaired brachial artery endothelium-dependent and –independent vasodilation in men with erectile dysfunction and no other clinical cardiovascular disease. J Am Coll Cardiol 2004; 43: 179–84.
19. Whitehead ED, Klyde BJ, Zussman S, Salkin P. Diagnostic evaluation of impotence. Postgrad Med 1990 August; 88: 123–6, 129–36.
20. UKPDS. UK prospective diabetes study 16. Overview of 6 years' therapy of type II diabetes: a progressive disease. Diabetes 1995; 44: 1249–58.
21. Gazzaruso C, Giordarnetti S, De Amiei E et al. Relationship between erectile dysfunction and silent myocardial ischemia in apparently uncomplicated type-2 diabetic patients. Circulation 2004; 110: 22–6.
22. Bottomley JM on behalf of T2 ARDIS Steering Group. Managing care of type 2 diabetes learnings from T2 ARDIS. Br J Diabetes Vasc Dis 2001; 1: 68–72.
23. O'Kane PD, Jackson G. Erectile dysfunction: is there silent obstructive coronary artery disease? Int J Clin Pract 2001; 55: 219–20.
24. Solomon H, Man J, Wierzbicki AS, O'Brien T, Jackson G. Erectile dysfunction: cardiovascular risk and the role of the cardiologist. Int J Clin Pract 2003; 57: 96–9.
25. Kaiser DR, Billups K, Mason C et al. Impaired brachial artery endothelium-dependent and -independent vasodilation in men with erectile dysfunction and no other clinical cardiovascular disease. J Am Coll Cardiol 2004; 43: 179–84.
26. Soloman H, Man J, Wierzbicki AS et al. The value of routine cardiovascular assessment in patients with erectile dysfunction.Circulation 2002; 106: 741–9.
27. Modena MG, Bonetti L, Coppi F et al. Prognostic role of reversible endothelial dysfunction in hypertensive postmenopausal women. J Am Coll Cardiol 2002; 40: 505–10.
28. Vaughan CJ, Murphy MB, Buckley BM. Statins do more than just lower cholesterol. Lancet
29. 1996; 348: 1079–82.
30. Feron O, Dessy C, Desager J-P et al. Hydroxy-methylglytarylcoenzyme A reductase Inhibition promotes endothelial nitric oxide synthase activation through a decrease in caveolin abundance. Circulation 2001; 103: 113–8. 30 Weitz-Schmidt G. Statins as anti-inflammatory agents. Trends Pharmacol Sci 2002; 23: 482–6.
31. Jackson G. PDE-5 inhibition in cardiovascular disease: experimental models and potential clinical applications. Eur Heart J 2002; 4 (Suppl): H19–H23.
32. Kimura M, Higashi Y, Hara K et al. PDE5 inhibitor sildenafil citrate augments endothelium dependent vasodilation in smokers. Hypertension 2003; 41: 1106–10. Epub 2003 April 14.
33. Halcox JP, Nour KR, Zalos G et al. The effect of sildenafil on human vascular function, platelet activation, and myocardial ischemia. J Am Coll Cardiol 2002; 40: 1232–40.
34. DeSouza C, Akers D, Parulkar A et al. Acute and prolonged effects of sildenafil on brachial artery flow-mediated dilation in type 2 diabetes. Diabetes Care 2002; 25: 13369.
35. Guay AT, Perez JB, Jacobson J, Newton RA. Efficacy and safety of sildenafil citrate for treatment of erectile dysfunction in a population with associated organic risk factors. J Androl 2001; 22: 793–7.
36. Yassin A, Alzubaidi RT, Kamkoum H, Alzubaidi RT, Ramadan A (2025) Recent Update on Advancements in Testosterone Therapy (TTh). J Urol Ren Dis 10: 1413.
37. Yassin A, Albaba B, Kamkoum H, Alzubaidi RT, Al-Qudimat AR (2025) Testosterone Prevention Role in Men's Health: Cardiovascular Dieseases. Cardiol Res Cardio vasc Med 10: 280.
38. Yassin A, Kamkoum H, Alzubaidi RT (2025) Is There a Need for Testosterone Therapy in Older Men? J Advances Med Sci 2: 1-3.
39. Yassin A, Kamkoum H, Alzubaidi RT, Ansari AA (2025) Testosterone Prevention Role in Men's Health: Diabetes Mellitus. J Diabetes Treat10: 10141.
40. Yassin A, Albaba B, Kamkoum H, Alzubaidi RT, Al-Qudimat AR (2025) Testosterone Prevention Role in Men's Health: Cardiovascular Dieseases. Cardiol Res Cardio vasc Med 10: 280.
41. Yassin A, Kamkoum H, Alzubaidi RT, Abdallah H, Assad O (2025) How Long Should We Treat with Testosterone: Stopping Testosterone Therapy (TTh) Wat is Next? Effects of Withdrawal and resumption of TTh. Ann Rev Resear 12: 555843.
42. Yassin A, Alzubaidi RT, Kamkoum H, Albudairat A, Abdallah H, et al. (2025) Testosterone and Prevention: Effects on Renal Function. J Urol Ren Dis 09: 1427. DOI: 10.29011/2575-7903.001427.