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REVIEW

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Erectile dysfunction in diabetes: a cardiometabolic marker and clinical opportunity

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Abstract

Erectile dysfunction (ED) is a common complication of diabetes, affecting more than half of men with the condition. Beyond its impact on quality of life, ED is increasingly recognised as an early manifestation of endothelial dysfunction and a marker of underlying cardiometabolic and cardiovascular disease. Despite this, sexual symptoms remain underreported and under-recognised in routine diabetes care. ED in diabetes is multifactorial, arising from vascular, neurological, hormonal and psychological mechanisms. Early identification provides an opportunity for cardiovascular risk assessment and metabolic optimisation. Clinical evaluation includes a focused sexual history, assessment of contributing comorbidities, targeted investigations and appraisal of cardiovascular risk. Management involves optimisation of metabolic health, evidence-based use of phosphodiesterase type 5 inhibitors and consideration of psychological and relational factors, with escalation to second-line therapies or specialist referral indicated for treatment-refractory symptoms or suspected endocrine or cardiovascular disease. Recognising ED in men with diabetes as a clinically meaningful marker of systemic disease supports more integrated approaches to men's health.

Keywords

Erectile dysfunction; Diabetes mellitus; Cardiometabolic risk; Men's health; Phosphodiesterase type 5 inhibitors

1. Introduction

Erectile dysfunction (ED) is a common and frequently under-recognised complication of diabetes, affecting more than 50% of men with the condition [1]. ED is defined as the persistent inability to achieve or maintain an erection sufficient for satisfactory sexual performance. It involves a complex interplay between vascular, neurological, hormonal and psychological systems, requiring intact arterial inflow, cavernosal smooth muscle relaxation, veno-occlusive function and neural signalling [2]. Despite its impact on wellbeing, sexual symptoms often go unaddressed, as many men do not volunteer these concerns, and clinical encounters instead frequently prioritise glycaemic and metabolic targets [3].

ED is increasingly recognised as an early manifestation of endothelial dysfunction and evolving vascular disease. It may precede or accompany other microvascular and macrovascular complications, signalling broader cardiometabolic decline [4].

Early identification enables cardiovascular risk assessment, metabolic optimisation and holistic support. However, ED remains under-screened and undertreated in routine diabetes care [3]. Integrating sexual health discussion into diabetes care facilitates recognition of ED not only as a distressing symptom, but as a clinically meaningful marker warranting timely evaluation [5, 6].

The pathophysiological link between ED and cardiovascular disease is supported by the artery size hypothesis, which proposes that smaller penile arteries are affected earlier by atherosclerotic burden compared with larger coronary vessels, leading to earlier manifestation of symptoms [2, 7]. Epidemiological studies demonstrate that ED frequently precedes clinically overt coronary artery disease by several years and is highly prevalent in men with established cardiovascular disease [4, 8].

This article highlights the importance of early recognition of ED in men with diabetes and provides a practical framework for clinical assessment and management within a broader men's health context.

2. Discussion

2.1 Pathophysiology of erectile dysfunction in diabetes

ED in men with diabetes reflects the combined effects of endothelial dysfunction, autonomic neuropathy, hormonal alterations and psychological factors [9]. Chronic hyperglycaemia promotes oxidative stress and reduces nitric oxide bioavailability, impairing smooth muscle relaxation and cavernosal blood flow [10]. These microvascular changes parallel other diabetic complications and often precede overt cardiovascular disease, supporting the role of ED as an early vascular marker. Clinical

evidence further supports ED as an early manifestation of systemic vascular disease. Studies have demonstrated that ED is present in approximately 50–75% of men with established coronary artery disease and may precede cardiovascular events by two to five years [4, 8]. Autonomic neuropathy further disrupts erectile signalling, resulting in delayed tumescence and reduced rigidity [10]. Functional hypogonadism is more prevalent in men with metabolic syndrome and may contribute to reduced libido, mood disturbance and diminished responsiveness to phosphodiesterase type 5 (PDE5) inhibitors [2].

Medications may also contribute to symptoms, particularly antihypertensives, antidepressants, opioids and some antipsychotics, alongside established risk factors including poor glycaemic control, obesity, hypertension and dyslipidaemia [11]. Psychological factors such as depression, anxiety, performance concerns and relationship stress frequently coexist and may exacerbate organic ED [5]. Most men present with a multifactorial clinical picture [5]. Recognition of this interplay supports interpretation of ED not as an isolated sexual symptom, but as a potential marker of underlying cardiometabolic decline, prompting timely assessment and individualised management. The prognosis of ED in men with diabetes is variable and closely linked to the degree of underlying vascular and metabolic dysfunction. Compared with the general population, ED in diabetes tends to occur earlier, progress more rapidly and respond less favourably to first-line therapies, reflecting more advanced endothelial and neurovascular impairment [2, 12]. While optimisation of glycaemic control and lifestyle factors may improve symptoms, ED often persists or progresses in the presence of ongoing cardiometabolic risk. Importantly, the presence of ED is associated with an increased risk of future cardiovascular events, reinforcing its role as a prognostic marker of systemic vascular disease [4, 8].

2.2 Clinical assessment

A focused history assists in distinguishing vascular, neurological, hormonal and psychological contributors of ED in men with diabetes. Important elements include onset, severity, duration and the degree of functional impairment. Gradual and progressive symptoms are more suggestive of organic aetiology, whereas sudden onset or fluctuating performance may indicate psychological influences or medication effects. Assessment of morning erections provides a useful clinical discriminator, with preserved nocturnal rigidity supporting a psychogenic component and consistently reduced rigidity suggesting an organic basis [13].

Low libido warrants specific exploration, as it may reflect hypogonadism, depression or adverse medication effects rather than primary erectile failure. Associated features such as fatigue, reduced energy and low mood may further support testosterone deficiency. Medication review remains essential, with particular attention to antihypertensives, antidepressants, opioids and antipsychotics. Alcohol intake, smoking and recreational drug use should also be considered.

Symptoms of peripheral or autonomic neuropathy, including altered sensation, postural dizziness or gastrointestinal disturbance, may provide additional context when interpreting erectile symptoms. A brief cardiovascular history can assist in identifying men who may benefit from earlier metabolic optimisation or cardiology assessment.

Psychological and relational factors frequently coexist and should be addressed sensitively. Many men may minimise or fail to disclose sexual distress due to embarrassment or assumptions relating to ageing or chronic illness. Normalising discussion of sexual health within diabetes care may facilitate disclosure and improve recognition of clinically significant symptoms.

2.3 Investigations and cardiovascular risk assessment

A focused clinical work-up assists in identifying contributors to ED in men with diabetes and supports safe initiation of therapy. Investigative findings should be interpreted within a broader cardiometabolic context [4].

Baseline evaluation includes blood pressure, body mass index, waist circumference and review of glycaemic control. Poorly controlled diabetes is associated with more severe ED, and optimisation of Haemoglobin A1c (HbA1c), blood pressure and lipid parameters may improve erectile function [5, 11]. Fasting lipid profile, renal function and electrolyte testing assist with cardiovascular risk stratification and ensure safe prescribing of PDE5 inhibitors.

Morning total testosterone should be measured, with levels below approximately 8–12 nmol/L suggesting biochemical hypogonadism depending on assay and clinical context [2]. Endocrine pathology is uncommon but should be considered where symptoms are disproportionate or accompanied by features such as headache or visual disturbance.

Medication review remains important, as antihypertensives, antidepressants, opioids and antipsychotics may adversely affect sexual function. Alcohol intake and recreational drug use should also be explored. Where neuropathic features are present, peripheral neurological examination may assist interpretation and guide broader metabolic management.

Cardiovascular risk assessment is central to the evaluation of ED in diabetes, given its association with early vascular disease. This may include formal risk stratification using validated tools such as Framingham or QResearch Cardiovascular Risk (QRISK) scores, electrocardiography where indicated, and consideration of exercise stress testing or cardiology referral in men with high-risk profiles. Symptoms such as exertional dyspnoea, reduced exercise tolerance or sudden onset ED should prompt consideration of cardiology review prior to treatment escalation. PDE5 inhibitors are generally safe in men with stable cardiovascular disease, provided nitrate therapy is avoided.

2.4 Management strategies

Management focuses on optimisation of metabolic health alongside appropriate pharmacological therapy. Lifestyle interventions that improve glycaemic control and vascular function may contribute to gradual improvement in erectile performance [14]. Measures such as weight reduction, regular physical activity, smoking cessation and optimisation of sleep are commonly associated with improved sexual function [15].

Assessment and treatment of suspected obstructive sleep apnoea may provide additional benefit. Moderate weight loss of approximately 5–10% body weight and engagement in at least 150 minutes of moderate-intensity exercise per week have been associated with improvements in endothelial function and erectile performance [16].

PDE5 inhibitors, including sildenafil, represent first-line pharmacological therapy. A summary of pharmacological treatment options is provided in Table 1. Adequate dosing and repeated use are often required before treatment efficacy can be assessed, and erectile response depends on sexual stimulation [8, 17]. Prior to initiation, renal function, potential drug interactions and cardiovascular symptoms should be reviewed. PDE5 inhibitors are contraindicated in men receiving nitrate therapy and in those with unstable cardiovascular disease. Men with diabetes frequently demonstrate reduced responsiveness to PDE5 inhibitors due to advanced neurovascular impairment [12]. In such cases, second-line therapies may be considered, including vacuum erection devices, intracavernosal injections and emerging treatments such as low-intensity extracorporeal shockwave therapy, which may promote neovascularisation and improve penile blood flow [18]. Research into regenerative therapies, including stem cell and platelet-rich plasma approaches, is evolving.

Antidiabetic therapies may also influence erectile function. Glucagon-like peptide-1 receptor agonists are associated with weight loss and improved metabolic parameters, which may indirectly improve erectile function [2, 19]. Sodium-glucose cotransporter 2 inhibitors have demonstrated cardiovascular benefits, although their direct effects on erectile function remain controversial [8, 20].

Medication review may assist by identifying agents that negatively affect libido or erectile performance, with substitution considered where clinically appropriate. Psychological and relational contributors should be explored, and targeted psychological support may be beneficial in men experiencing significant distress [5].

In many men, combined metabolic optimisation and PDE5 inhibitor therapy result in satisfactory outcomes. Ongoing review allows reassessment of treatment response, while persistent or refractory symptoms may warrant further investigation or specialist input.

2.5 When to escalate care

Escalation of care should be guided by symptom severity, treatment response and the likelihood of alternative or contributory diagnoses. While many men with diabetes respond to initial management strategies, timely specialist involvement can support more comprehensive assessment and optimise outcomes.

Urological assessment is appropriate when erectile function remains inadequate despite appropriate dosing and repeated trials of PDE5 inhibitors, particularly following optimisation of metabolic factors and concomitant medications [12]. Specialist input is also indicated when structural abnormalities such as Peyronie's disease or painful penile curvature are suspected, or when second-line therapies, including vacuum devices, intracavernosal injections or surgical options require consideration. Persistent genitourinary symptoms or recurrent balanitis unresponsive to conservative measures may also warrant further evaluation.

Endocrinology referral should be considered in men with persistently low testosterone levels accompanied by relevant symptoms, or where underlying endocrine pathology is suspected. Specialist involvement may also assist in cases of

TABLE 1. Pharmacological management of ED.

Agent	Mechanism of action	Indications	Contraindications	Common adverse effects
Sildenafil	PDE5 inhibition → increased nitric oxide-mediated vasodilation	First-line treatment for ED	Nitrate therapy, unstable cardiovascular disease	Headache, flushing, dyspepsia, visual disturbance
Tadalafil	PDE5 inhibition with longer half-life	ED with desire for spontaneity or daily dosing	Nitrate therapy	Headache, back pain, dyspepsia
Vardenafil	PDE5 inhibition	Alternative first-line option	Nitrate therapy, QT prolongation risk	Headache, flushing, rhinitis
Avanafil	Selective PDE5 inhibition with rapid onset	Patients requiring rapid onset of action	Nitrate therapy	Headache, flushing
Intracavernosal alprostadil	Prostaglandin E1 → direct smooth muscle relaxation	Second-line therapy in PDE5 non-responders	Bleeding disorders, penile deformity	Penile pain, priapism
Testosterone therapy	Restores androgen levels	Symptomatic hypogonadism with confirmed low testosterone	Prostate cancer, elevated haematocrit	Acne, fluid retention, erythrocytosis

PDE5: Phosphodiesterase type 5; ED: Erectile dysfunction.

complex metabolic disease with refractory ED.

Cardiovascular assessment is warranted when ED has sudden onset, is rapidly progressive, or is associated with exertional dyspnoea or reduced exercise tolerance. PDE5 inhibitors are generally safe in men with stable cardiovascular disease, provided nitrate therapy is avoided.

Referral for psychological or sex therapy may benefit men with prominent performance anxiety, depressive symptoms or relationship difficulties, with partner involvement often enhancing therapeutic outcomes.

A practical clinical algorithm is outlined in Fig. 1.

3. Conclusion

ED is a common and clinically significant complication of diabetes that extends beyond sexual function alone. Recognition of ED provides an important opportunity to identify underlying cardiometabolic risk, optimise metabolic health and address associated psychological and relational factors. A structured approach to assessment and management supports timely identification of men who may benefit from targeted intervention or specialist input. Viewing ED within a broader men's health framework reinforces its role as a meaningful marker of systemic disease and facilitates more integrated, holistic care.

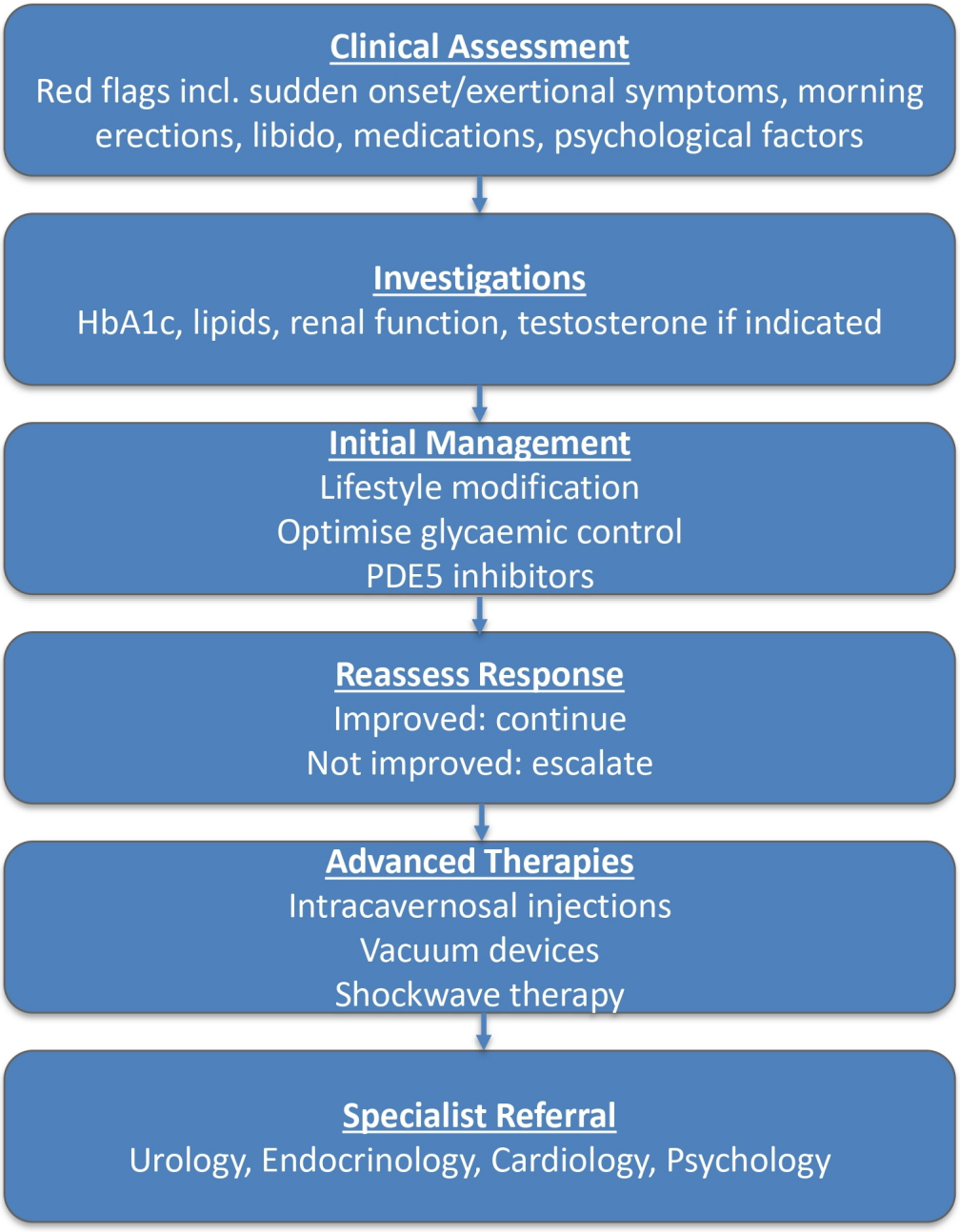

FIGURE 1. Clinical algorithm flowchart. PDE5: Phosphodiesterase type 5; HbA1c: Haemoglobin A1c.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

AUTHOR CONTRIBUTIONS

MK and AR—conceptualised this review. MK—performed the literature review and created the draft manuscript. AR—provided guidance and revisions for the final manuscript. Both authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was not required as this study is a narrative review of existing literature.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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