REVIEW

Erectile dysfunction

A clinical overview with perspectives from African settings

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ABSTRACT

Erectile dysfunction (ED) is a common male sexual disorder with significant implications for quality of life, interpersonal relationships, and overall health. Although its prevalence increases with age and comorbid conditions, ED remains underdiagnosed and undertreated worldwide. This narrative review provides a comprehensive overview of the epidemiology, pathophysiology, diagnostic evaluation, and management of ED, drawing primarily on established global evidence and clinical guidelines. Data and experiences from African settings are incorporated, where available, to illustrate contextual challenges related to healthcare access, cultural perceptions, and resource limitations, as well as emerging trends in presentation and treatment patterns.

INTRODUCTION

Sexual health is a vital aspect of overall well-being, yet sexual dysfunction significantly impacts men's quality of life and relationships worldwide.[1,2] Among the various forms of sexual dysfunction, erectile dysfunction (ED), defined as the persistent inability to achieve or maintain an erection sufficient for satisfactory sexual performance, is the most prevalent, with notable psychologic and social consequences.[3-5]

In many African communities, sexual performance is closely associated with masculine identity, and men may view ED as a personal failure or a disruption of spiritual balance.[6] Beliefs linking ED to curses, witchcraft, or ancestral displeasure may lead individuals to seek traditional or spiritual remedies rather than medical care. These culturally embedded perceptions contribute to stigma, underreporting, and delays in seeking appropriate treatment.[7]

Globally, ED remains underdiagnosed and undertreated, especially in low-resource settings, where healthcare infrastructure, awareness, and research are limited.[5] Overcoming these deeply rooted perceptions is essential to fostering open dialogue and encouraging timely medical consultation.

ED arises from a multifactorial interplay involving physiologic, psychologic, lifestyle, and socioeconomic contributors. Furthermore, ED itself is a significant risk factor for cardiovascular and metabolic diseases, with its onset potentially occurring up to five years before a cardiovascular event.[8,9]

Socioeconomic challenges, such as poverty, limited education, and

poor access to healthcare, impede early diagnosis and management.[10] Cultural and religious norms further shape negative perceptions of sexual health, discouraging men from seeking professional support and reinforcing stigma.[10,11] The lack of trained sexual health specialists and insufficient healthcare resources across many African countries perpetuates this cycle of underdiagnosis and undertreatment.[10]

Despite growing recognition of ED as a significant public health issue, there remains a notable lack of continent-wide data and context-specific research addressing its epidemiology, management strategies, and psychosocial dimensions in Africa. Addressing this gap requires a multidisciplinary, culturally informed approach that takes into account both medical and sociocultural factors influencing care

This review provides a broad overview of ED, synthesizing established global evidence on epidemiology, risk factors, diagnostic evaluation, and therapeutic approaches. Core clinical principles relevant to the assessment and management of ED across diverse practice settings are outlined, with data and experiences from African contexts incorporated to illustrate region-specific challenges, healthcare constraints, and emerging trends. By situating these contextual perspectives within a general clinical framework, the review aims to support clinicians, researchers, and health policymakers in delivering informed, equitable, and effective care for men with ED across varied healthcare environments.

EPIDEMIOLOGY OF ED IN AFRICAN MEN

ED is a highly prevalent condition worldwide, affecting men across diverse age groups, with prevalence increasing significantly with age. Global estimates suggest that approximately 30–60% of men over the age of 40 experience some degree of ED, with variability influenced by differences in study populations, diagnostic criteria, and reporting practices.[11-14] Large, population-based studies, including the Massachusetts Male Aging Study, have demonstrated a steady rise in prevalence from around 40% in men aged 40 years to nearly 70% in those aged 70 years.[12,15] In addition to aging, major global risk factors include cardiovascular disease (CVD), diabetes mellitus, obesity, smoking, and sedentary lifestyle.[9,14-17]

Despite its high prevalence, ED remains underdiagnosed and undertreated worldwide due to stigma, lack of awareness, and limited access to healthcare services, particularly in low- and middle-income countries.[13,18] These global patterns provide an important framework for understanding the burden and unique challenges of ED within African settings.

The prevalence of ED among African men varies significantly, influenced by factors such as lifestyle, comorbidities, healthcare access, and sociocultural attitudes. Studies estimate that ED affects approximately 30–60% of men globally over the age of 40; however, the true burden of ED may be even greater due to underreporting, largely stemming from cultural taboos, stigma, and a lack of awareness.[19]

Epidemiologic studies on ED in Africa have been conducted in both community and hospital settings, although the condition remains less extensively researched than other aspects of sexual health. In community-based studies, the estimated prevalence in Sub-Saharan Africa is around 20%,[18,20] For instance, in Egypt, a population-based study reported that 10% of men experienced moderate ED, while 13% had severe ED.[21] In Ghana, prevalence rates are particularly high, with 66% of men reporting ED. Additional concerns, such as infrequent sexual activity (70%) and premature ejaculation (65%), were also noted.[18,20] In Nigeria, community studies indicate prevalence rates ranging from 44–59%, with similar figures seen in hospital settings, especially among men with chronic conditions like hypertension and diabetes.[22]

In Tanzania, a study conducted in Dar es Salaam found that 24% of men in the community had ED, whereas the prevalence was significantly higher (55%) among those attending diabetic care clinics.[23,24] In Kenya, a striking 95% of hypertensive men attending an ambulatory clinic at Kenyatta National Hospital were affected.[25,26] Similarly, in Ethiopia, 60% of diabetic patients were diagnosed with ED, with an alarming 97.6% remaining untreated or lacking access to medical care[26] (Figure 1).

These findings emphasize the widespread and often overlooked burden of ED across Africa. They also highlight the urgent need for improved public awareness, culturally appropriate education, early diagnosis, and accessible treatment options. Notably, significant data gaps persist in many parts of the continent, particularly in Central, Northern, and parts of Western and Southern Africa, limiting the development of region-specific strategies (Table 1). Future research efforts must prioritize filling these gaps through nationally representative studies that can inform effective and equitable health interventions.

PATHOPHYSIOLOGY AND TYPES OF ED

Erectile function relies on a complex interplay between vascular and neural mechanisms. The internal pudendal artery is the primary source of blood supply to the penis,

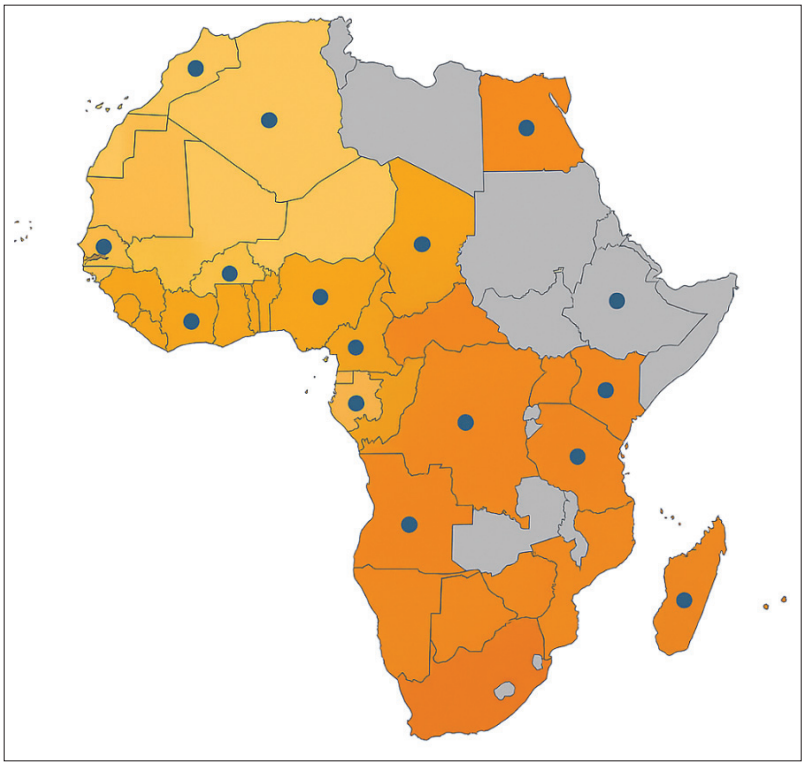

Figure 1. Prevalence of erectile dysfunction across African countries. The map visualizes reported erectile dysfunction (ED) prevalence based on available peer-reviewed studies. Countries in grey indicate lack of published or nationally representative data. Color-coded prevalence ranges: <30% (light orange), 30–60% (orange), >60% (dark orange). Southern African data, such as from South Africa, are primarily drawn from focused clinical populations (e.g., men with HIV or hypertension), limiting direct national extrapolation. This highlights the need for broader epidemiological studies in the region. Note: Data absence does not indicate lack of public health concern but rather a lack of accessible or published data at the time of this review. Data Source and Inclusion Criteria: Prevalence data visualized in this map were obtained from peer-reviewed studies identified through a narrative search of PubMed, Scopus, and the *African Journal of Urology* archives (2000–2024) using the keywords "erectile dysfunction," "prevalence," and "Africa." Only studies reporting prevalence estimates in adult male populations within African countries were included. Where multiple studies were available for a country, the most representative or largest dataset was selected. Grey shading indicates countries with no published prevalence data at the time of this review.

Table 1. Reported prevalence of erectile dysfunction in African countries

Region	Country	Study population	Prevalence (%)	Study source
West Africa	Ghana	General male population	66%	Amidu et al, 2010[18]
	Nigeria	Community-based men	44–59%	Idung et al, 2012; Oyelade et al, 2016[6,7]
North Africa	Egypt	Population-based study	Moderate: 10%, Severe: 13%	Seyam et al, 2003[10]
East Africa	Tanzania	Diabetic patients	55%	Mutagaywa et al, 2014[8]
	Kenya	Hypertensive patients (KNH)	95%	Correia et al, 2022[25]
	Ethiopia	Diabetic patients (tertiary hospital)	60%	Abuhay et al, 2021[19]
Southern Africa	South Africa	Aging men, hypertensive, HIV+	29–47%	Lewis et al, 2004; Shai et al, 2019; Pretorius et al, 2005

distributing blood through the cavernosal branches, while venous drainage occurs via a network of easily compressible venules.[16,27] During sexual arousal, parasympathetic signals from the sacral spinal cord stimulate the release of nitric oxide (NO). This initiates an increase in intracellular cyclic guanosine monophosphate (cGMP), leading to relaxation of vascular smooth muscle. As a result, blood flow into the corpora cavernosa increases.[27] This rapid inflow compresses the subtunical venous plexus, reducing venous outflow and raising intracavernosal pressure, culminating in an erection.

NO, released from both endothelial cells and parasympathetic nerve terminals, is the primary neurotransmitter mediating this process, although other neurotransmitters may also contribute. The resulting cavernosal smooth muscle relaxation leads to compression of small veins in the subtunical region, effectively occluding venous return and maintaining the erection (Figure 2). Penile detumescence, or the process of returning to a flaccid state, begins with the activation of adrenergic receptors on the cavernous arteries and trabecular smooth muscles. This reduces arterial inflow and causes the collapse of the lacunar spaces. As a result, venous drainage from the cavernous bodies is restored, leading to detumescence.

ED occurs when there is any disruption in these vascular or neural pathways.[27] Although aging is an established independent risk factor, it is a misconception that sexual dysfunction is an inevitable part of aging. In reality, comorbid conditions, unhealthy lifestyle habits, and psychologic factors play a major role in the development and progression of ED.[10]

Normal sexual function is a biopsychosocial process that requires coordination among psychologic, endocrine, vascular, and neurologic systems. ED is typically categorized into three types: psychogenic, organic (which may include neurogenic, hormonal, arterial, cavernosal, or drug-induced causes), and mixed, where both psychogenic and organic factors are involved (Table 2). In clinical practice, most cases are of mixed origin.

Psychogenic ED

Psychologic factors play a significant role in the development of ED, either as isolated causes or in conjunction with organic contributors. Among these, performance anxiety or the fear of sexual failure during intercourse is one of the most commonly identified triggers.[28]

Historically, multiple theories have been proposed to explain the role of psychologic factors in ED, suggesting that developmental, cognitive, affective, and interpersonal factors may predispose men to sexual dysfunction.[28]

Today, psychogenic ED is more broadly understood as the result of a complex interplay of predisposing, precipitating, and maintaining factors (Table 3).

Neurogenic ED

ED is associated with various neurologic disorders, including multiple sclerosis, temporal lobe epilepsy, Parkinson's disease, stroke, Alzheimer's disease, and spinal cord injury.[29] In many African settings, the true burden of neurogenic ED remains underrecognized due to diagnostic limitations, low public awareness of neurodegenerative diseases, and the social stigma surrounding sexual health. Patients undergoing radical pelvic surgeries, such as prostatectomy or cystectomy, are at particularly higher risk of cavernous nerve injury, a common cause of neurogenic ED. Although nerve-sparing techniques have reduced this complication in high-income countries, such advancements are not widely accessible across most African healthcare systems, except in countries like South Africa and Egypt. Postoperative sexual rehabilitation and counseling are also not routinely offered, contributing to persistent untreated dysfunction and diminished quality of life.[30]

Endocrinologic ED

Androgens, particularly testosterone play a critical role in male sexual function. They enhance sexual desire, support nocturnal (sleep-related) erections, and contribute to overall erectile capacity; however, their influence on visually induced erections is relatively limited. Testosterone also regulates the expression of nitric oxide synthase (NOS) and phosphodiesterase type 5 (PDE5) in penile tissue.[31] Testosterone deficiency, or hypogonadism, has been increasingly associated with elevated risks of cardiovascular morbidity and mortality.[32] In addition to low testosterone, hyperprolactinemia can contribute to ED by disrupting the hypothalamic-pituitary-gonadal axis. Elevated prolactin levels inhibit the release of gonadotropin-releasing hormone (GnRH), which subsequently suppresses luteinizing hormone (LH) secretion, an essential stimulus for testosterone production.

Vasculogenic ED

Vasculogenic ED is primarily linked to conditions that impair blood flow, including atherosclerosis, hypertension, hyperlipidaemia, smoking, diabetes mellitus, and pelvic irradiation.[33] A central mechanism in this process is endothelial dysfunction, which serves as a common pathologic pathway for many of these risk factors. Studies have demonstrated that the prevalence of ED is significantly higher in patients with hypertension, with rates reaching up to 68%.[34-36] Notably, improvement in erectile function has been observed following reduc-

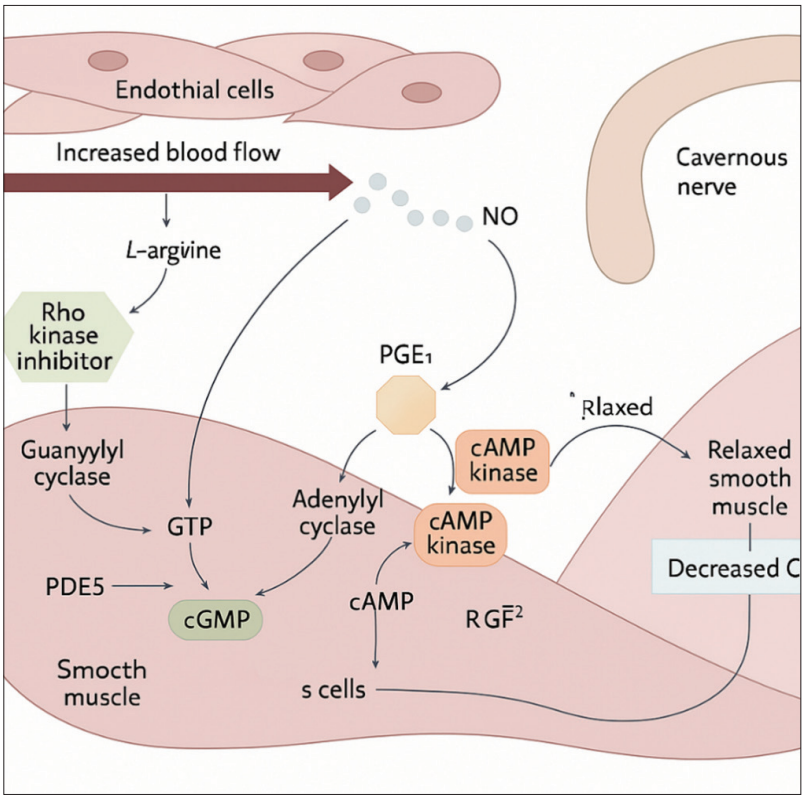

Figure 2. Diagram illustrating the role of nitric oxide (NO) in penile smooth muscle relaxation. NO activates the GTPcGMP pathway, lowering calcium and causing smooth muscle relaxation. PDE5 breaks down cGMP to terminate the erection. A secondary pathway via cAMP also reduces calcium. Erection-enhancing drugs like PDE5 inhibitors and PGE_1 act on these pathways. Key molecular components are labeled (e.g., eNOS, PDE5, GTP, cGMP, GPCR). Concept adapted from Haderer & Muller Biomedical Art, LLC (2009). Created by the authors. ATP: adenosine triphosphate; AMP: adenosine monophosphate; cGMP: cyclic guanosine monophosphate; eNOS: endothelial nitric oxide synthase; GPCR: G-protein-coupled receptor; GTP: guanosine triphosphate; NO: nitric oxide; PDE5: phosphodiesterase type 5; $PGF_{2\alpha}$: prostaglandin $F_{2\alpha}$; PGE_1: prostaglandin.

Table 2. Main organic causes of erectile dysfunction

Category	Examples
Neurogenic	- Central: Cerebral insult, multiple sclerosis, spinal cord injury - Peripheral afferent: Sensory neuropathy (e.g., diabetes mellitus) - Peripheral efferent: Autonomic neuropathy, post-radical pelvic surgery
Endocrinologic	Diabetes mellitus, hypogonadism, hyperprolactinaemia
Vasculogenic	- Arterial: Macro/microangiopathy (e.g., atherosclerosis, trauma) - Venous: Corporal veno-occlusive dysfunction - Sinusoidal: Failure of smooth muscle relaxation (e.g., fibrosis)
Drug-induced and substance use	- Antihypertensives, antidepressants, antiandrogens, major tranquillizers - Cigarette smoking, alcoholism, recreational drugs (e.g., marijuana, heroin)
Systemic diseases/ general health	Liver disease, renal failure, respiratory illness, cardiovascular disease
Local penile (cavernous) factors	Cavernous fibrosis (e.g., post-priapism), Peyronie's disease, penile fracture

Table 3. Factors related to the development of psychogenic erectile dysfunction

Category	Examples
Predisposing factors	- Traumatic past experiences - Strict upbringing - Inadequate sex education - Physical and mental health problems
Precipitating factors	- Acute relationship problems - Family or social pressures - Major life events (e.g., pregnancy, childbirth, job loss)
Maintaining factors	- Ongoing relationship issues - Persistent physical or mental health problems - Lack of awareness about treatment options

tions in total cholesterol and low-density lipoprotein (LDL) levels, either through dietary modification or statin therapy.[32] Risk factors, such as diabetes, dyslipidemia, obesity, and smoking, are not only associated with ED, but also represent well-established contributors to coronary artery disease (CAD).

The Princeton III consensus guidelines recognize ED as an early warning sign and independent predictor of CVD, particularly CAD.[37] This association has been supported by several studies, including a longitudinal investigation by Inman et al, which followed over 1400 community-dwelling men without known CAD for 10 years.[38] The study reported that 11% developed incident CAD, with 15% experiencing myocardial infarction, 79% showing angiographic abnormalities, and 6% suffering sudden cardiac death. Importantly, ED was found to be a stronger predictor of future cardiac events in men under 60 years, compared to older cohorts.

This cardiovascular link may be explained by shared risk factors, with endothelial dysfunction emerging as a critical underlying pathology.[38] The incidence of CAD was influenced by age, with men without ED showing lower incidence densities than those with ED. Notably, ED in men younger than 60 years was associated with a significantly higher risk of future cardiac events compared to those without ED, although this association was less pronounced in older men.

Contributing mechanisms include impaired L-arginine-nitric oxide pathways, increased sympathetic tone, structural vascular changes, and systemic inflammation.[32,35] The "artery size hypothesis," proposed by Montorsi and colleagues, offers a compelling explanation for the early manifestation of ED relative to CAD.[39] Given that the penile artery (1–2 mm) is narrower than the coronary arteries (3–4 mm), atherosclerotic plaques tend to produce symptoms of ED before obstructing coronary blood flow, which typically leads to angina. In addition, inadequate venous occlusion may result from the development of large venous channels draining the corpora cavernosa or from degenerative, functional, or structural alterations of the tunica albuginea, as seen in Peyronie's disease. These changes can impair veno-occlusive function during erection, leading to premature venous outflow and contributing to vasculogenic ED.[40]

Table 4. Summary of drug classes associated with ED

Drug class	Common examples	Risk level	Proposed mechanism of ED
Antidepressants	SSRIs (e.g., fluoxetine, sertraline), venlafaxine	High	Increased serotonin levels inhibit sexual arousal and delay orgasm
Antipsychotics	Risperidone, olanzapine	High	Dopamine blockade, increased prolactin, decreased testosterone
Antihypertensives	Thiazide diuretics, β-blockers (e.g., atenolol)	Moderate to high	Reduced penile blood flow, impaired vascular or hormonal response
Antihypertensives (low-risk)	ACE inhibitors, ARBs, α-blockers	Low	Minimal effect on erectile function; some may improve endothelial health
Lipid-lowering agents	Statins (e.g., atorvastatin, simvastatin)	Low to moderate	Potential endothelial dysfunction; unclear and possibly dose-dependent
Benzodiazepines	Diazepam, alprazolam	Moderate	CNS depression, reduced libido, and impaired sexual response
5-alpha-reductase inhibitors	Finasteride, dutasteride	Moderate to high	Reduced dihydrotestosterone (DHT), impairing libido and erectile function
Hormonal agents	GnRH agonists, anti-androgens	High	Suppression of testosterone synthesis

ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker; ED: erectile dysfunction; GnRH: gonadotropin-releasing hormone SSRI: selective serotonin reuptake inhibitors.

Drug-induced ED

Drug-induced ED is a relatively common but often underrecognized cause of sexual dysfunction. It is primarily associated with psychotropic medications and antihypertensive agents.[41] Among psychotropic drugs, antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs) and venlafaxine, are most frequently linked to ED. These medications increase serotonin levels in the brain, which can negatively affect sexual function (Table 4).

Antipsychotics, including risperidone and olanzapine, are also strongly associated with ED, and rank among the psychotropic drugs with the highest incidence of sexual side effects.[41]

Several antihypertensive medications have been linked to ED as well. Thiazide diuretics are the most commonly associated, followed by β-blockers, both of which can impair erectile function through reductions in penile blood flow or hormonal alterations.[42] In con-

trast, α-blockers, angiotensin-converting enzyme (ACE) inhibitors, and angiotensin receptor blockers (ARBs) have a lower risk profile in this regard compared to thiazides and β-blockers[43] (Table 4).

Although statins are crucial in managing hyperlipidaemia and reducing cardiovascular risk, some studies have reported associations with ED.[44] This may be related to potential effects on endothelial function, which is essential for normal erectile physiology Understanding the potential sexual side effects of commonly prescribed medications is critical for both diagnosis and management. A thorough medication history should be part of the ED assessment, and when appropriate, modifying the pharmacologic regimen may significantly improve sexual outcomes.

IMPACT OF AGING, LIFESTYLE, AND CHRONIC ILLNESS ON ERECTILE FUNCTION

ED is increasingly recognized as a prevalent yet underreported health issue among African men. Consistent with global trends, aging is the most significant independent risk factor, with both prevalence and severity increasing progressively with age. In the Massachusetts Male Aging Study, 39% of men reported some degree of ED by the age of 40, rising to 67% by the age of 70.[15] Similar findings have been observed in regional studies involving over 2400 Spanish men and 1400 Middle Eastern men.[45]

In African the burden of type 2 diabetes mellitus is accelerating due to rapid urbanization and lifestyle transitions, making it a major contributor to ED. ED occurs in approximately 50–75% of diabetic men and may be the first clinical manifestation in up to 12% of patients with undiagnosed diabetes.[15,46] Notably, ED is three times more common in diabetics than in non-diabetics (49.3% vs. 15.6%, respectively), highlighting its potential as a diagnostic marker for metabolic disease.[47]

Lifestyle-related risk factors for ED are increasingly prevalent in African urban centers, driven by changes in diet, physical inactivity, and rising substance use.[46] These include: sedentary behavior, obesity and metabolic syndrome, and poor sleep hygiene, as well as alcohol, tobacco, and recreational drug use (including cannabis and local stimulants).[46]

Additionally, chronic systemic illnesses, such as chronic kidney disease, liver dysfunction, and chronic pulmonary disorders, are strongly associated with ED.[48-50] In many African settings, these conditions are often underdiagnosed or poorly managed due to limited healthcare infrastructure and diagnostic capacity.

Cultural stigma, limited sexual health education, and inequitable access to care further complicate ED management. Addressing these challenges requires an integrated, multidisciplinary approach that combines public health education, routine screening for chronic diseases, and culturally sensitive, accessible sexual health services. ED in Africa should be viewed not only as a quality-of-life issue but also as a clinical indicator of broader vascular and metabolic health.

RISING TRENDS OF ED AMONG YOUNG ADULTS IN AFRICA

Recent reports from clinical centers in countries such as Nigeria, Tanzania, Ethiopia, and Egypt have documented a growing number of ED cases among adolescents and young adult men.[51–53] This emerging pattern appears to be driven by rapid lifestyle transitions associated with urbanization and westernization, including increased consumption of energy-dense diets, alcohol and tobacco use, and a decline in physical activity. These behavioral and metabolic shifts are believed to contribute to early vascular and endocrine changes, thereby increasing the risk of early-onset ED.[52]

The most commonly affected group comprises men aged 25–35 years, many of whom present with performance anxiety, psychologic stress, or features of subclinical metabolic dysfunction. Hormonal profiles in most cases fall within normal ranges, and semen analyses are typically unremarkable. In this cohort, psychogenic factors are often significant contributors to ED. In response to sexual performance concerns, there has been a surge in the unsupervised use of PDE5-inhibitors (PDE5-Is), frequently obtained without prescription; however, treatment outcomes in this group remain suboptimal, likely due to the absence of formal diagnosis and failure to address underlying psychologic and lifestyle factors.[51-53] This evolving trend underscores the need for targeted sexual health education, early identification of modifiable risk factors, and the development of culturally appropriate interventions aimed at young men across the continent.

DIAGNOSIS OF ED

The diagnosis of ED involves a structured assessment that includes a detailed clinical history, physical examination, and targeted laboratory investigations. A goal-directed approach is currently recommended, aiming to confirm the diagnosis, identify the underlying etiology, and evaluate for contributing risk factors.[54] Additionally, it is essential to assess for associated comorbidities, including conditions that may be life-threatening, such as CVD (Figure 3).

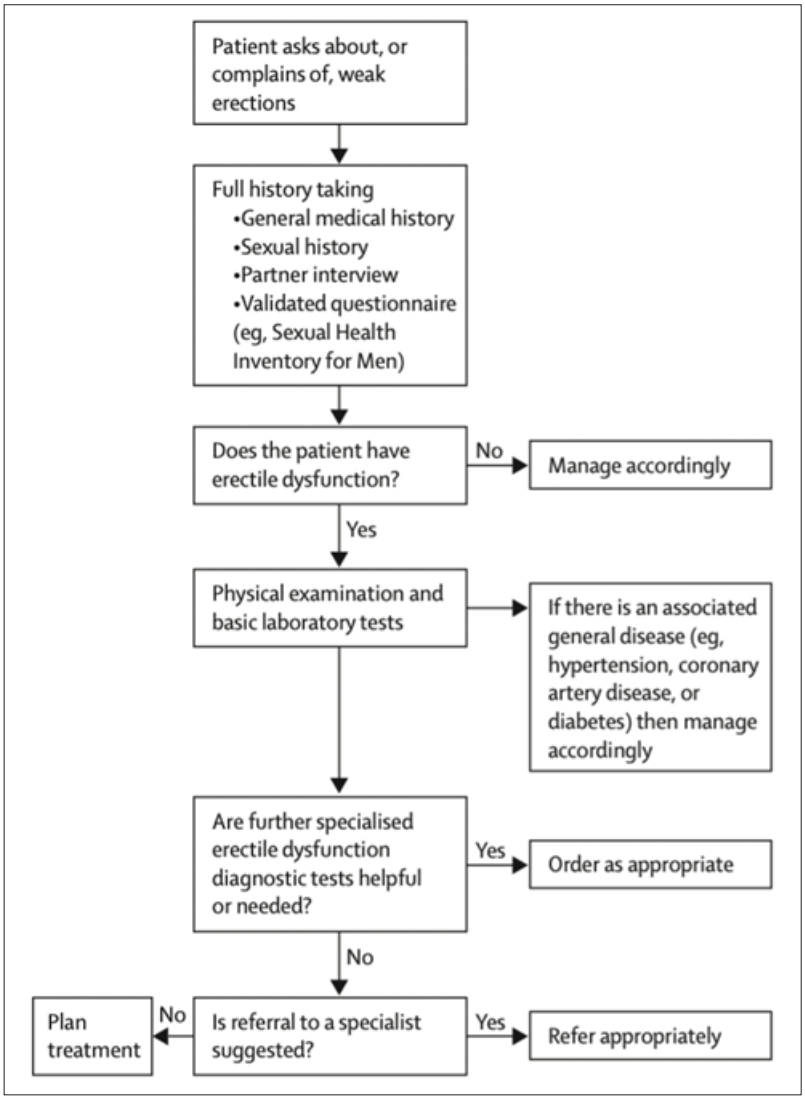

Figure 3. Flowchart outlining the clinical approach to diagnosing erectile dysfunction (ED). The algorithm begins with patient history and symptom assessment, followed by physical examination and basic laboratory tests. Depending on findings, further steps include evaluating psychological factors, assessing hormone levels (e.g., testosterone), and considering penile Doppler ultrasound or nocturnal penile tumescence testing. The chart helps clinicians determine whether ED is psychogenic, organic, or mixed, guiding appropriate treatment strategies.

Common diagnostic tools used in the evaluation of ED include:

- International Index of Erectile Function (IIEF-5): A validated questionnaire to assess the severity and impact of ED.
- Nocturnal penile tumescence (NPT) testing: Used to differentiate psychogenic from organic ED by evaluating erections during sleep.
- Hormonal assays: Including total and free testosterone, prolactin, and LH to detect endocrine abnormalities.
- Psychologic evaluation: To identify anxiety, depression, or other psychogenic contributors.

This comprehensive and systematic evaluation ensures that management strategies are appropriately tailored to the individual's clinical profile.[54]

History taking

A thorough and detailed sexual and medical history is critical in diagnosing ED. During the initial evaluation, the clinician should obtain a comprehensive psychosocial history, including the patient's perception of their sexual performance, level of sexual satisfaction, and attitudes toward sex. When appropriate, interviewing the patient's partner can provide additional insight into relationship dynamics and sexual functioning.

Differentiating between ED and other sexual disorders, such as premature ejaculation, is essential. Patients reporting difficulty maintaining erections may, in fact, be experiencing premature ejaculation. ED is characterized by the inability to achieve or maintain an erection until orgasm, whereas premature ejaculation occurs after erection but before the desired time of climax.

Clinicians should distinguish between organic and psychogenic causes of ED. Psychogenic ED often presents with preserved spontaneous erections (e.g., during sleep or in response to sexual thoughts), sudden onset, intermittent symptoms, or short duration. In contrast, organic ED typically has a gradual onset, a progressive course, and long-standing duration.[54]

A review of the patient's medication use, including antidepressants, antihypertensives, and recreational substances, such as alcohol and tobacco, is vital. Additionally, a complete past medical and surgical history should be obtained, with specific attention to chronic conditions like diabetes, hypertension, and neurologic disorders.

Standardized tools, such as the IIEF and the Sexual Health Inventory for Men (SHIM) are useful for diagnosing ED and grading its severity. They also serve as valuable instruments for tracking treatment response in clinical practice and research.[55,56]

Emerging evidence has established a strong association between ED and CAD. Notably, ED may precede clinical manifestations of CVD by 2-3 years.[57] Consequently, it is now recommended that all men presenting with ED, regardless of cardiac symptoms, be evaluated for cardiovascular risk.[37]

After the initial clinical evaluation, patients should be stratified into low, intermediate, or high cardiovascular risk categories. In intermediate-risk patients, especially those under 60 years, further cardiovascular assessment is warranted. This may include baseline electrocardiogram (ECG), exercise ECG testing, and, if indicated, cardiac imaging or referral to a cardiologist. Additional

screening tools include waist circumference, body mass index (BMI), coronary artery calcium scoring, carotid intima-media thickness, peripheral arterial tonometry, and serum biomarkers of vascular inflammation and endothelial dysfunction.[37]

Physical exam

A comprehensive physical examination is an essential component in the evaluation of ED, encompassing both general systemic assessment and focused genitourinary examination. This evaluation aids in identifying potential organic causes, such as hormonal imbalances, vascular insufficiency, or neurologic deficits, while also providing an opportunity to build rapport and address patient concerns.

The general examination should include:

- Blood pressure measurement (hypertension is a major risk factor)
- Cardiovascular assessment for signs of peripheral vascular disease or heart disease
- Anthropometric measurements, such as BMI and waist circumference, which can suggest metabolic syndrome
- Signs of endocrinopathy, including gynecomastia, decreased body hair, or testicular atrophy, which may indicate hypogonadism

The focused genital examination should assess:

- Penile anatomy for structural abnormalities (e.g., Peyronie's disease)
- Testicular volume and consistency to evaluate for atrophy or masses
- Peripheral pulses and neurologic reflexes (e.g., bulbocavernosus reflex) to assess vascular and nerve function

Importantly, the physical eaxm provides an opportunity for the clinician to reassure the patient about normal anatomical variation, dispel myths regarding penile size and masculinity, and reduce performance anxiety. Addressing these psychosocial factors during the exam is a critical aspect of holistic ED management.

Laboratory assessment

Initial laboratory evaluation for ED should include fasting blood glucose and total testosterone levels, as these can help identify common metabolic and endocrine contributors. Given the established association between ED and CVD, it is also recommended to assess the patient's lipid profile, including total cholesterol, LDL, high-density lipoprotein (HDL), and triglycerides.

If total testosterone levels are found to be low, further hormonal investigations should be performed. These may include measurements of free testosterone, LH, follicle-stimulating hormone (FSH), and prolactin, to evaluate for hypogonadism, pituitary disorders, or hyperprolactinemia.

In cases where the initial findings suggest a complex endocrine disorder or when psychologic comorbidities are suspected, referral to a specialist such as an endocrinologist, urologist, or mental health professional may be warranted for more comprehensive evaluation and management.

Specific investigations

For many patients, particularly younger men and their partners, understanding whether ED is reversible is a critical aspect of the treatment process. Additionally, some forms of ED may be early indicators of serious, potentially life-threatening cardiovascular conditions.[37]

In most African settings, however, routine use of advanced investigative procedures is not recommended due to their high cost, limited availability, and limited impact on standard treatment approaches. Patients in resource-constrained regions may face unnecessary financial burdens without significant changes in management outcomes.

Nonetheless, specialized centers in countries such as South Africa and Egypt are equipped to perform more advanced diagnostic investigations, especially for complex or treatment-resistant cases. These investigations aim to identify underlying vascular dysfunction and may guide tailored therapeutic strategies when initial treatments fail.

Recent research has introduced emerging tools to assess penile endothelial function, such as penile NO release testing, the Endo-PAT2000 device, and the evaluation of circulating biomarkers like endothelin-1, C-reactive protein, and endothelial progenitor cells.[37] The most common diagnostic investigations, along with their clinical utility and limitations, are summarized in Table 5.

TREATMENT MODALITIES OF ED

The management of ED in Africa follows global treatment guidelines but faces unique challenges due to healthcare accessibility, cultural beliefs, and resource limitations. The primary approach remains oral PDE5-Is, which are widely recommended as first-line therapy; however, access to these medications can be limited by cost and availability in many regions (Figure 4).

In parallel, many men continue to rely on traditional remedies, reflecting strong cultural beliefs and widespread use of herbal and spiritual practices in the management

Table 5. Common diagnostic tests for ED and their clinical applications

Test	Purpose	Clinical use	Limitations
IIEF questionnaire	Quantify severity and monitor treatment response	Widely used in all settings	Subjective; relies on patient self-report
Nocturnal penile tumescence (NPT)	Distinguish psychogenic from organic ED	Useful in specialized centers	Requires specialized equipment; not widely available
Penile Doppler ultrasound	Assess arterial inflow and veno-occlusive function	Available in select tertiary facilities	Operator dependent; not widely accessible
Penile NO release test	Evaluate endothelial NO activity	Research tool; available in Egypt, South Africa	Limited to high-resource settings
Endo-PAT2000	Non-invasive assessment of vascular endothelial function	Experimental; cardiometabolic research centers	High cost; requires training and calibration
Serum biomarkers (e.g., CRP, endothelin-1)	Assess vascular inflammation and endothelial health	Research or high-risk cardiovascular cases	Not routinely available; not yet standard of care

These diagnostic tools, while promising, remain largely inaccessible in routine clinical settings across most of Africa. Therefore, their use should be individualized and reserved for referral centers with the necessary infrastructure and trained personnel. CRP: C-reactive protein; ED: erectile dysfunction; NO: nitric oxide.

of ED.[6] This emphasizes both the popularity of these approaches and the urgent need to scientifically evaluate and develop potent African traditional remedies into safe, standardized natural medicines. This perspective highlights an important opportunity to bridge cultural practices with evidence-based medicine, ensuring safer and more acceptable treatment options for African patients.

The role of lifestyle modification in managing ED

Recent clinical and experimental studies suggest that addressing modifiable lifestyle factors, such as smoking, alcohol consumption, obesity, and physical inactivity, can significantly improve erectile function.[58-61] Research by Mannino et al found that former smokers had a lower prevalence of ED compared to current smokers (2.0% vs. 3.7%).[58] Additionally, Guay et al reported that individuals with a history of heavy smoking (over 30 pack-years) experienced rapid improvements in erectile function upon smoking cessation.[62]

The relationship between alcohol consumption and ED remains unclear, as existing studies provide mixed findings.[63-65] In contrast, a landmark study involving 110 obese men with ED demonstrated that those who participated in a structured weight loss program with dietary counseling and exercise guidance showed significant weight reduction, increased physical activity, and improved erectile function scores after two years.[66] These results have been corroborated by subsequent research.[67,68]

A 2011 meta-analysis by Gupta et al, which reviewed six randomized controlled trials involving 740 participants, further supported the idea that lifestyle modification and cardiovascular risk reduction provide incremental benefits for erectile function, independent of PDE5-I use.[69] Possible mechanisms include improvements in endothelial function, reduced insulin resistance, and decreased low-grade inflammation factors commonly linked to diabetes mellitus and metabolic syndrome, both known contributors to ED.[69]

While current evidence suggests that lifestyle modifications can significantly benefit men with ED, definitive conclusions require larger, well-designed, prospective studies. Furthermore, improvements in erectile function through lifestyle changes may take up to two years, which is a considerable timeframe;[66] however, combining lifestyle modifications with PDE5-I therapy can yield noticeable improvements within three months.[70] Importantly, established ED treatments should not be delayed while awaiting the effects of lifestyle changes.

Oral PDE5-I in ED treatment

Oral PDE5-Is are considered the first-line treatment for ED in many parts of the world.[71]

Currently, five oral PDE5-Isare commercially available in the world: sildenafil, tadalafil, vardenafil, udenafil, and mirodenafil. The first three are widely available in Africa, while others, such as avanafil, lodenafil, and SLx-2101, are still under investigation for ED treatment in high-income countries.[72] These PDE5-Is have a success rate of at least 65% and exhibit suitable onset and duration of action.[72-74] Physicians should consider trying multiple PDE5-Is to determine which provides the best efficacy with minimal side effects for each patient. It is recommended to attempt each PDE5-I at least four times before evaluating its effectiveness.[75]

Several studies suggest that chronic or daily PDE5-I use can significantly improve endothelial function, potentially offering a long-term therapeutic benefit for ED.[76-78] Among these, tadalafil 5 mg is the only PDE5-I approved for daily use. The benefits of daily dosing include rescuing on-demand PDE5-I non-responders, modifying disease progression, and promoting a more natural sexual response; however, drawbacks include high costs, limited long-term safety data, and incomplete understanding of its mechanisms of action.[79]

PDE5-Is primarily enhance erectile function rather than libido. In young, healthy men, these drugs can

reduce the refractory period, the temporary post-ejaculation phase of physiologic erectile flaccidity, leading to improved ejaculatory control;[80,81] however, their concurrent use with nitrate medications is strictly contraindicated due to the risk of severe hypotension.[82]

Studies indicate that PDE5-Is do not increase the risk of myocardial infarction or death, nor do they exacerbate ischemia or cardiac function during exercise in patients with CAD or heart failure.[83] Nonetheless, PDE5-Is should be used cautiously in patients with severe CVD such as uncontrolled hypertension or unstable angina, and in those taking α-blockers for blood pressure management. Meanwhile, PDE5-Is are well-tolerated in combination with other antihypertensive agents, such as calcium channel blockers.[82] Vardenafil, however, is contraindicated in patients using type-1A (e.g., quinidine, procainamide) or type-3 (e.g., sotalol, amiodarone) antiarrhythmic drugs or those with congenital long QT syndrome.[83]

The side effects of PDE5-Is are generally mild and well-tolerated, with headache and flushing being the most common. Tadalafil, in particular, has been associated with muscle pain and discomfort in various body regions. Rare cases of PDE5-I-related priapism have been documented.[84] Although a direct association between PDE5-I use and non-arteritis ischemic optic neuropathy has not been confirmed, the possibility remains under investigation.[85] Additionally, patients should be informed of a potential link between sildenafil use and hearing impairment.[86]

Despite their effectiveness, PDE5-Is failed to produce satisfactory results in approximately 35% of ED patients. Common causes of treatment failure include diabetes mellitus and severe neurologic or vascular disorders. While there is no universal definition of PDE5-I failure, an accepted criterion is the inability to achieve or maintain an erection during sexual intercourse on at least four consecutive occasions despite optimal dosing.[75]

Management strategies for PDE5-I failure depend on the underlying cause and may involve patient counseling, switching to another PDE5-I, intracavernosal injection therapy, intraurethral drug administration (e.g., MUSE [Vivus, CA, U.S.]), combination therapy, or referral to a specialist. For patients unresponsive to all medical treatments, penile implant surgery may be considered (Table 6).

The use of testosterone in ED

Testosterone plays a crucial role in maintaining normal erectile function, but its role in ED treatment is limited. Testosterone replacement therapy (TRT) is recommended for men with ED who have confirmed low bioavailable testosterone levels. A meta-analysis of 16 studies found that ED improvement was significantly more common in hypogonadal men treated with testosterone compared to those receiving a placebo (57.0% vs. 16.7%).[87] Additionally, testosterone has been successfully combined with PDE5-Is in elderly men (≥65 years) with low testosterone levels who initially did not respond to PDE5-Is, demonstrating its potential as an adjunct therapy.[88,89]

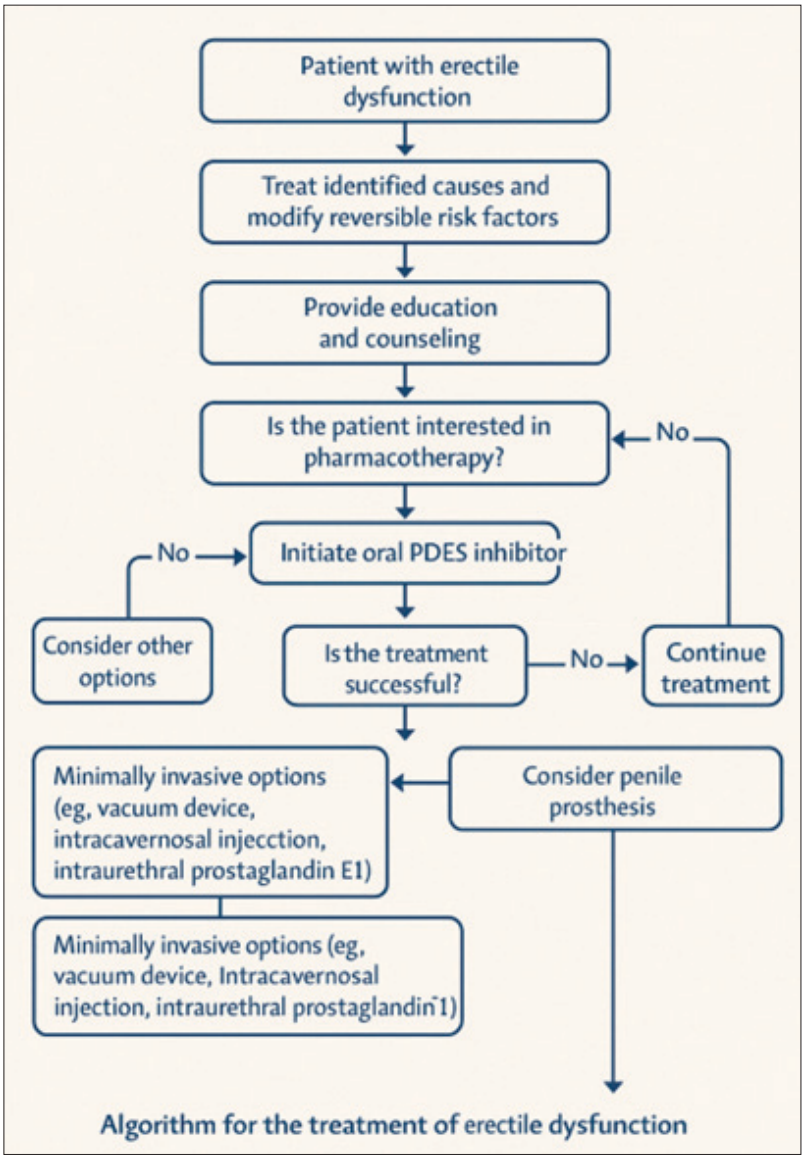

Figure 4. Diagram showing the integrative treatment options for erectile dysfunction (ED). Central pharmacologic therapy (e.g., phosphodiesterase type 5 inhibitors) is supported by adjunct strategies: lifestyle modification (e.g., diet, exercise, smoking cessation), testosterone supplementation for men with low hormone levels, and psychosexual therapy to address psychological or relational issues. The image conveys that ED treatment is most effective when approached holistically.

Table 6. Characteristics of commercially available phosphodiesterase type 5 inhibitors

Drug	Dosage	Onset	Duration	Efficacy	Side effects	Contraindications	Food/alcohol interaction
Sildenafil	25, 50, 100 mg (start 50 mg; max 100 mg)	30–60 min	4–8 h	>65%	Headache, flushing, dyspepsia	Nitrates, recent serious CV events, non-arthritic ION, α-blockers	Food affects absorption; take fasting. No alcohol interaction
Vardenafil	2.5, 5, 10, 20 mg (start 10 mg; max 20 mg)	30 min	4–8 h	>65%	Same as sildenafil	Same as sildenafil + type 1/3 antiarrhythmics, congenital prolonged QT syndrome	Food affects absorption; take fasting. No alcohol interaction
Tadalafil	2.5, 5, 10, 20 mg (start 10 mg; max 20 mg)	45 min	Up to 36 h	>65%	Flushing, back pain, myalgia	Same as sildenafil	No food or alcohol interaction
Udenafil	100 mg (max 200 mg)	30–60 min	12 h	>65%	Facial flushing, nasal congestion, ocular hyperemia, headache	Same as sildenafil	No food or alcohol interaction
Mirodenafil	50 or 100 mg (max 100 mg)	30–60 min	6–12 h	>65%	Facial flushing, headache, nausea, eye redness		

Intracavernosal injection and transurethral therapy

Intracavernosal injection and transurethral therapy are considered second-line treatments for ED, offering the advantage of predictable and rapid erections. With proper training, men or their partners can administer injections using fine 28–30-gauge needles, typically achieving an erection within 10 minutes, independent of sexual stimulation. This therapy is commonly prescribed for men who do not respond to or dislike oral treatments, as well as those with spinal cord injuries or post-radical prostatectomy ED.

Frequently used agents include alprostadil (prostaglandin E1), papaverine, phentolamine, and vasoactive intestinal polypeptide. Combination therapies, such as intracavernosal mixtures of two or more vasoactive drugs, have also shown high efficacy in high income countries. While alprostadil alone achieves success rates of up to 70%, trimix solutions demonstrate even higher efficacy, reaching approximately 90%.[90,91]

Despite their effectiveness, these treatments have potential side effects, including priapism and penile fibrosis, which can be minimized with appropriate patient education and monitoring. Penile pain is a common side effect of alprostadil injections, and high dropout rates (>50%) are primarily due to the inconvenience of administration.[91]

Alprostadil is also available as an intraurethral pellet (MUSE), with reported success rates ranging from 43-69% in developed countries;[90,92] however, its use may be associated with penile pain, urethral discomfort or burning, hypotension, syncope, and priapism.

In Africa, access to intracavernosal injection and transurethral therapies remains limited; however, select specialized centers in countries such as South Africa, Morocco, Egypt, and Nigeria do offer these services, primarily within privately owned urology clinics or tertiary hospitals. Despite their proven efficacy, broader use is constrained by factors such as limited provider training, lack of patient awareness, high out-of-pocket costs, and cultural hesitation toward injectable therapies for sexual health

Vacuum constrictive devices

Vacuum constrictive devices (VCDs) function by creating continuous negative pressure around the shaft of the penis, drawing blood into the corpora cavernosa. An elastic band placed at the base of the penis helps retain the blood, maintaining the erection. These devices are relatively inexpensive and have minimal risks;[14] however, erections achieved through VCDs may feel unnatural, as they are mechanically induced and often associated with a cold penile sensation.[14] As a result, nearly half of patients report dissatisfaction with this method. VCDs are typically recommended for men in stable relationships who have not responded to oral PDE5-Is and prefer to avoid more invasive treatments, such as intracavernosal injections or penile prosthesis implantation.

In many African settings, the availability and awareness of VCDs remain limited. Access is often constrained to private-sector facilities or urban referral hospitals, and cultural perceptions regarding artificial or mechanically induced erections may reduce acceptabil-

ity. Moreover, few sexual health providers across the continent receive formal training in counseling patients on the appropriate use of VCDs. Common side effects, such as petechiae, penile numbness, and delayed ejaculation, may also contribute to poor adherence in settings where followup care is not routinely accessible.[93]

Penile prostheses

Penile prosthesis implantation is considered a third-line treatment for ED, typically reserved for men who have not responded to or are not candidates for medical or less invasive therapies. Once implanted, the prosthesis irreversibly alters the corporal tissue, eliminating the possibility of natural erectile function due to the loss of smooth muscle elasticity.

There are two primary types of prosthetic devices:

- Semi-rigid (malleable) prostheses, which provide constant firmness and require manual positioning
- Inflatable prostheses, particularly the three-piece hydraulic model, which is the most commonly used in high-resource settings; this system includes two inflatable cylinders, a scrotal pump, and a fluid reservoir, allowing the user to achieve an erection on demand and deflate it afterward

Although these devices are most frequently implanted in developed healthcare systems, several African countries, including Tanzania, Kenya, Egypt, Nigeria, and South Africa, have the surgical infrastructure and urologic expertise to offer penile prosthesis implantation, particularly in referral or tertiary-level hospitals. In Egypt and South Africa, in particular, prosthetic surgery is increasingly used for patients with severe ED, including those with post-prostatectomy dysfunction or diabetes-related vascular disease.

Patient and partner satisfaction rates are consistently high, with reports showing up to 70% satisfaction among patients and as high as 90% among partners;[94] however, the procedure is not without risks. Infection remains the most significant complication, occurring in approximately 2–4% of cases.[95] Device malfunction and mechanical failure are other potential concerns, especially in areas where followup and replacement may be logistically difficult. Other surgical options, such as arterial bypass surgery for traumatic penile arterial injuries and venous ligation for congenital venous leak, are rarely performed today due to limited long-term efficacy, high cost, and technical complexity.

In the African setting, the availability of penile prostheses is generally limited to urban centers and private or teaching hospitals, and cost remains a major barrier for many patients. Expanding access and insurance coverage for such interventions may improve quality of life for men with refractory ED, especially those with a strong desire to restore sexual function and intimacy.

PSYCHOSEXUAL THERAPY, COUNSELING, AND THE SOCIAL IMPACT OF ED

Psychosexual therapy is particularly indicated for men with predominantly psychogenic ED and where significant psychologic problems are recognized. Techniques such as sensate focus, sex education, and interpersonal therapy are used to improve sexual confidence and intimacy; however, data regarding the efficacy of these approaches remain largely inconclusive.

Psychotherapy and counseling play a crucial role, especially in African societies, where ED is heavily stigmatized. Addressing mental health concerns such as anxiety, depression, and performance-related stress is essential for improving treatment outcomes. In many African cultures, where masculinity is closely tied to sexual performance, ED can lead to marital discord, low self-esteem, and mental health struggles.[22]

Traditional and herbal remedies are widely used across Africa, often alongside or as alternatives to modern medical treatments.[24] While some traditional medicines may have potential benefits, their efficacy and safety require further scientific validation. Given the psychologic and social consequences of ED, integrating psychosexual therapy and counseling into treatment strategies is essential for holistic patient care.

CONCLUSIONS

Globally, ED remains underdiagnosed and undertreated, especially in low-resource settings such as Africa, where major challenges persist in awareness, diagnosis, treatment access, and data availability. Many men continue to suffer in silence due to stigma, lack of information, and limited healthcare infrastructure. Addressing these issues will require a comprehensive, culturally responsive approach that integrates public education, clinical training, health system strengthening, and research investment.

Closing the data gaps, particularly in Central and Eastern Africa, is essential for developing effective and regionally appropriate interventions. With sustained commitment across clinical, research, and policy domains, Africa can move toward equitable, informed, and compassionate care for men's sexual health.

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RECOMMENDATIONS

1. Promote public awareness and reduce stigma

2. Encourage healthy lifestyle modifications

3. Promote strategies to reduce vascular and metabolic risk factors associated with ED

4. Discourage unregulated use of aphrodisiacs

5. Tailor treatment to comorbidities and clinical profiles

6. provide psychologic support for younger men

7. Train healthcare providers in sexual health

8. Improve access to evidence-based therapies

9. Strengthen data and research capacity

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