Understanding the burden of erectile dysfunction in Africa: A comprehensive review

Charles John Nhungo[1,2], George Elisamehe Mocha[2], Fransia Arda Mushi[3], Charles Anaeli Mkony[2,3]

[1]Department of Surgery, Faculty of Medicine & Dentistry, University of Alberta, Edmonton, AB, Canada; [2]Department of Surgery, Rabininsia Memorial Hospital, Dar es Salaam, Tanzania; [3]Department of Surgery, School of Medicine, Muhimbili University of Health and Allied Sciences, Dar es Salaam, Tanzania

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Corresponding author: Dr. Charles John Nhungo, Department of Surgery, Faculty of Medicine & Dentistry, University of Alberta, Edmonton, AB, Canada; nhungo@ualberta.ca

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ABSTRACT

Erectile dysfunction (ED) is a prevalent yet underreported health concern among African men. Despite increasing recognition, cultural stigma, limited healthcare access, and socioeconomic disparities contribute to persistent underdiagnosis and undertreatment. This review synthesizes available evidence on the epidemiology, risk factors, diagnostic practices, treatment patterns, and psychosocial impact of ED in Africa, while highlighting cultural beliefs, barriers to care, and regional data gaps. Most published studies originate from North and South Africa, leaving Central and Sub-Saharan regions underrepresented. Addressing ED across the continent requires context-specific, multidisciplinary approaches that integrate medical, psychological, and culturally sensitive interventions. Strengthening local research capacity is essential to guide health policy and improve equitable access to care for men's sexual health.

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 psychological and social consequences[3,4,5].

Globally, ED remains underdiagnosed and undertreated, especially in low-resource settings where healthcare infrastructure, awareness, and research are limited [5].

In Africa, the topic of sexual health, particularly male sexual performance, is often surrounded by cultural silence and stigma [6].In many African communities, sexual performance is closely associated with masculine identity, and men may view erectile dysfunction as a personal failure or a disruption of spiritual balance [7]. 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 [6]. Overcoming these deeply rooted perceptions is essential to fostering open dialogue and encouraging timely medical consultation.

ED arises from a multifactorial interplay involving physiological, psychological, lifestyle, and socioeconomic contributors. Physiological conditions such as hypertension, diabetes mellitus, cardiovascular disease, obesity, and hormonal imbalances are prominent causes[4,5]. Age, smoking, and comorbidities like peripheral vascular and coronary artery diseases are also associated with higher ED prevalence[8–12].

Numerous studies suggest that men with these pre-existing non-communicable diseases are more likely to have mild-to-severe ED than those without such conditions[13]. 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 [14,15]. Psychological stressors such as anxiety, depression, and performance-related concerns further compound the problem [16]. Hormonal disorders, including both hypothyroidism, hyperprolactinemia, hypercortisolism, hypotestosteronaemia and hyperthyroidism, can also disrupt erectile function[17].

Lifestyle factors, including tobacco use, excessive alcohol consumption, and physical inactivity, further increase the risk [16]. Additionally, approximately 25% of ED cases are medication-induced, often linked to antihypertensives, antidepressants, and antipsychotics. Drugs such as alpha-blockers, benzodiazepines, beta-blockers, clonidine, digoxin, ketoconazole, and selective serotonin reuptake inhibitors are among those most frequently associated with ED[16,18,19].

ED is frequently comorbid with other sexual and urological health issues, including premature ejaculation, benign prostatic hyperplasia (BPH), and overactive bladder [20,21].Other contributing factors include low testosterone levels, prostate inflammation, prostate cancer, a sedentary lifestyle, obesity, and symptoms of depression [20,21].
Socioeconomic challenges such as poverty, limited education, and poor access to healthcare—impede early diagnosis and management [16]. Cultural and religious norms further shape negative perceptions of sexual health, discouraging men from seeking professional support and reinforcing stigma [16,22]. The lack of trained sexual health specialists and insufficient healthcare resources across many African countries perpetuates this cycle of underdiagnosis and undertreatment [16].

Despite growing recognition of erectile dysfunction (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 synthesizes current evidence on ED across the African continent, outlining regional patterns, risk factors, diagnostic approaches, therapeutic options, and systemic barriers to care. The insights presented aim to support clinicians, researchers, and health policymakers in designing more effective, equitable, and contextually relevant interventions to improve men's sexual health outcomes across Africa.

Epidemiology of erectile dysfunction in African men

The prevalence of erectile dysfunction (ED) among African men varies significantly, influenced by factors such as lifestyle, comorbidities, healthcare access, and socio-cultural attitudes. Studies estimate that ED affects approximately 30–60% of men globally over the age of 40, with a higher prevalence among those with hypertension, diabetes, and cardiovascular disease [23]. However, the true burden of ED may be even greater due to underreporting, largely stemming from cultural taboos, stigma, and a lack of awareness[24]. Epidemiological 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% [20,25]. For instance, in Egypt, a population-based study reported that 10% of men experienced moderate ED, while 13% had severe ED[12]. 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 [20,25]. In Nigeria, community studies indicate prevalence rates ranging from 44% to 59%, with similar figures seen in hospital settings, especially among men with chronic conditions like hypertension and diabetes[9].

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 [10,26]. In Kenya, a striking 95% of hypertensive men attending an ambulatory clinic at Kenyatta National Hospital were affected [21,27], Similarly, in Ethiopia, 60% of diabetic patients were diagnosed with ED, with an alarming 97.6% remaining untreated or lacking access to medical care [21]. (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 & types of erectile dysfunction

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, distributing blood through the cavernosal branches, while venous drainage occurs via a network of easily compressible venules [28,29]. 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 [29]. 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.

Erectile dysfunction (ED) occurs when there is any disruption in these vascular or neural pathways [29]. 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 psychological factors play a major role in the development and progression of ED[16].

Normal sexual function is a biopsychosocial process that requires coordination among psychological, endocrine, vascular, and neurological 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 erectile dysfunction

Psychological factors play a significant role in the development of erectile dysfunction (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[30]. Historically, multiple theories have been proposed to explain the role of psychological factors in erectile dysfunction, suggesting that developmental, cognitive, affective, and interpersonal factors may predispose men to sexual dysfunction[30]. Today, psychogenic erectile dysfunction is more broadly understood as the result of a complex interplay of predisposing, precipitating, and maintaining factors (Table-3).

Neurogenic erectile dysfunction

Erectile dysfunction is associated with various neurological disorders, including multiple sclerosis, temporal lobe epilepsy, Parkinson’s disease, stroke, Alzheimer’s disease, and spinal cord injury[31]. In many African settings, the true burden of neurogenic erectile dysfunction remains under-recognized 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 particular risk of cavernous nerve injury, a common cause of neurogenic erectile dysfunction. 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 counselling are also not routinely offered, contributing to persistent untreated dysfunction and diminished quality of life [32].

Endocrinological erectile dysfunction

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[33]. Testosterone deficiency, or hypogonadism, has been increasingly associated with elevated risks of cardiovascular morbidity and mortality [34]. In addition to low testosterone, hyperprolactinemia can contribute to erectile dysfunction 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 erectile dysfunction

Vasculogenic erectile dysfunction is primarily linked to conditions that impair blood flow, including atherosclerosis, hypertension, hyperlipidemia, smoking, diabetes mellitus, and pelvic irradiation[35]. A central mechanism in this process is endothelial dysfunction, which serves as a common pathological 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% [36–38]. Notably, improvement in erectile function has been observed following reductions in total cholesterol and low-density lipoprotein levels, either through dietary modification or statin therapy[34]. 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 cardiovascular disease (CVD), particularly CAD[39]. This association has been supported by several studies, including a longitudinal investigation by Inman et al. (2009), which followed over 1,400 community-dwelling men without known CAD for ten years [40]. 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[40]. The incidence of CAD was influenced by age, with men without erectile dysfunction showing lower incidence densities than those with erectile dysfunction. Notably, erectile dysfunction in men younger than 60 years was associated with a significantly higher risk of future cardiac events compared to those without erectile dysfunction, 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 [34,37]. The "artery size hypothesis," proposed by Montorsi and colleagues, offers a compelling explanation for the early manifestation of ED relative to CAD [41]. 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 erectile dysfunction[42].

Drug-induced erectile dysfunction

Drug-induced erectile dysfunction (ED) is a relatively common but often underrecognized cause of sexual dysfunction. It is primarily associated with psychotropic medications and antihypertensive agents[43]. Among psychotropic drugs, antidepressants particularly selective serotonin reuptake inhibitors (SSRIs) and venlafaxine are most frequently linked to erectile dysfunction. 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[43].

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 [44]. In contrast, 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[45]. (Table-4)

Although statins are crucial in managing hyperlipidemia and reducing cardiovascular risk, some studies have reported associations with ED [46]. 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 ageing, lifestyle, and chronic illness on erectile function

Erectile dysfunction (ED) is increasingly recognized as a prevalent yet underreported health issue among African men. Consistent with global trends, ageing 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[47]. Similar findings have been observed in regional studies involving over 2,400 Spanish men and 1,400 Middle Eastern men [48].

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. Erectile dysfunction occurs in approximately 50–75% of diabetic menand may be the first clinical manifestation in up to 12% of patients with undiagnosed diabetes[49,47]. 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[50].

Lifestyle-related risk factors for ED are increasingly prevalent in African urban centers, driven by changes in diet, physical inactivity, and rising substance use [49]. These include: Sedentary behavior,Obesity and metabolic syndrome,Poor sleep hygiene,Alcohol, tobacco, and recreational drug use (including cannabis and local stimulants) [49]. Additionally, chronic systemic illnesses such as chronic kidney disease, liver dysfunction, and chronic pulmonary disorders are strongly associated with ED [51–53]. 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 erectile dysfunction among young adults in Africa

Recent reports from clinical centres in countries such as Nigeria, Tanzania, Ethiopia, and Egypt have documented a growing number of erectile dysfunction (ED) cases among adolescents and young adult men [54–56]. This emerging pattern appears to be driven by rapid lifestyle transitions associated with urbanisation and westernisation, including increased consumption of energy-dense diets, alcohol and tobacco use, and a decline in physical activity. These behavioural and metabolic shifts are believed to contribute to early vascular and endocrine changes, thereby increasing the risk of early-onset ED[55].

The most commonly affected group comprises men aged 25–35 years, many of whom present with performance anxiety, psychological 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 phosphodiesterase type 5 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 psychological and lifestyle factors [54–56]. 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 erectile dysfunction

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 [57]. Additionally, it is essential to assess for associated comorbidities, including conditions that may be life-threatening, such as cardiovascular disease.(Figure-3)

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 luteinizing hormone to detect endocrine abnormalities.
- Psychological 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 [57].

History taking

A thorough and detailed sexual and medical history is critical in diagnosing erectile dysfunction (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 erectile dysfunction 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 longstanding duration[57].

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 neurological disorders.

Standardized tools such as the International Index of Erectile Function (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 [58,59].

Emerging evidence has established a strong association between ED and coronary artery disease (CAD). Notably, ED may precede clinical manifestations of cardiovascular disease by two to three years[60]. Consequently, it is now recommended that all men presenting with ED regardless of cardiac symptoms be evaluated for cardiovascular risk[39]. 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 ECG, exercise ECG testing, and, if indicated, cardiac imaging or referral to a cardiologist. Additional screening tools include waist circumference, BMI, coronary artery calcium scoring, carotid intima-media thickness, peripheral arterial tonometry, and serum biomarkers of vascular inflammation and endothelial dysfunction [39].

Physical examination

A comprehensive physical examination is an essential component in the evaluation of erectile dysfunction (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 neurological 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 neurological reflexes (e.g., bulbocavernosus reflex) to assess vascular and nerve function.

Importantly, the physical examination 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 erectile dysfunction (ED) should include fasting blood glucose and total testosteronelevels, as these can help identify common metabolic and endocrine contributors. Given the established association between ED and cardiovascular disease, it is also recommended to assess the patient's lipid profile, including total cholesterol, LDL, 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, luteinizing hormone (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 psychological 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 erectile dysfunction (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[39].

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 centres 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 nitric oxide (NO) release testing, the Endo-PAT2000 device, and the evaluation of circulating biomarkers like endothelin-1, C-reactive protein, and endothelial progenitor cells[39].The most common diagnostic investigations, along with their clinical utility and limitations, are summarized in (Table-5.)

Treatment modalities of erectile dysfunction
The management of erectile dysfunction (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 phosphodiesterase type 5 inhibitors (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 of ED. [7]emphasized 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 erectile dysfunction

Recent clinical and experimental studies suggest that addressing modifiable lifestyle factorssuch as smoking, alcohol consumption, obesity, and physical inactivity can significantly improve erectile function[61–64] . Research by Mannino et al. [61] found that former smokers had a lower prevalence of erectile dysfunction (ED) compared to current smokers (2.0% vs. 3.7%). Additionally, Guay et al. [65] reported that individuals with a history of heavy smoking (over 30 pack-years) experienced rapid improvements in erectile function upon smoking cessation.

The relationship between alcohol consumption and ED remains unclear, as existing studies provide mixed findings[66–68]. In constrast, 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[69]. These results have been corroborated by subsequent research [70,71].

A 2011 meta-analysis by Gupta et al.[72], 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 inhibitor (PDE5-I) use. 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 [72].

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 [69]. However, combining lifestyle modifications with PDE5-I therapy can yield noticeable improvements within three months [73]. Importantly, established ED treatments should not be delayed while awaiting the effects of lifestyle changes.

Oral PDE5 inhibitors in erectile dysfunction treatment

Oral phosphodiesterase type 5 inhibitors (PDE5-Is) are considered the first-line treatment for erectile dysfunction (ED) [74]in many parts of the world. These medications work by inhibiting the PDE5 enzyme, which degrades cyclic guanosine monophosphate (cGMP) in the cavernous smooth muscle. This inhibition prolongs cGMP activity, leading to decreased intracellular calcium levels, sustained smooth muscle relaxation, and ultimately, rigid penile erections.

Currently, five oral (PDE5-Is) are 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 [75]. These PDE5-Is have a success rate of at least 65% and exhibit suitable onset and duration of action[75–77].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[78].

Several studies suggest that chronic or daily PDE5-I use can significantly improve endothelial function, potentially offering a long-term therapeutic benefit for ED[79–81]. 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 [82].
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 physiological erectile flaccidity leading to improved ejaculatory control[83,84]. However, their concurrent use with nitrate medications is strictly contraindicated due to the risk of severe hypotension [85] (70). 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 coronary artery disease (CAD) or heart failure [86]. Nonetheless, PDE5-Is should be used cautiously in patients with severe cardiovascular diseases, 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 [85]. 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 [86].

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 [87]. Although a direct association between PDE5-I use and non-arteritic ischemic optic neuropathy has not been confirmed, the possibility remains under investigation[88]. Additionally, patients should be informed of a potential link between sildenafil use and hearing impairment[89]

Despite their effectiveness, PDE5-Is fail to produce satisfactory results in approximately 35% of ED patients. Common causes of treatment failure include diabetes mellitus and severe neurological 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[78] . 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, USA]), 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 Erectile dysfunction.
Testosterone plays a crucial role in maintaining normal erectile function, but its role in erectile dysfunction (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%)[90]. Additionally, testosterone has been successfully combined with PDE5 inhibitors in elderly men (≥65 years) with low testosterone levels who initially did not respond to PDE5-Is, demonstrating its potential as an adjunct therapy[91,92].

Intracavernosal injection and transurethral therapy

Intracavernosal injection and transurethral therapy are considered second-line treatments for erectile dysfunction (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%[93,94].

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[94].

Alprostadil is also available as an intraurethral pellet (MUSE), with reported success rates ranging from 43% to 69%[93,95] in developed countries. 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 centres 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 utilization 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[23].

However, erections achieved through VCDs may feel unnatural, as they are mechanically induced and often associated with a cold penile sensation[23]. 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 inhibitors and prefer to avoid more invasive treatments such as intracavernosal injections or penile prosthesis implantation.

In many African settings, however, 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 acceptability. 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 follow-up care is not routinely accessible[96].

Penile prostheses

Penile prosthesis implantation is considered a third-line treatment for erectile dysfunction (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 urological 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[97]. However, the procedure is not without risks. Infection remains the most significant complication, occurring in approximately 2–4% of cases [98]. Device malfunction and mechanical failure are other potential concerns, especially in areas where follow-up 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 erectile dysfunction

Psychosexual therapy is particularly indicated for men with predominantly psychogenic erectile dysfunction (ED) and where significant psychological 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[9]

Traditional and herbal remedies are widely used across Africa, often alongside or as alternatives to modern medical treatments[10]. While some traditional medicines may have potential benefits, their efficacy and safety require further scientific validation. Given the psychological and social consequences of ED, integrating psychosexual therapy and counseling into treatment strategies is essential for holistic patient care.

CONCLUSIONS

Erectile dysfunction is an increasingly recognised public health issue across Africa, yet 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.

Recommendations

Addressing erectile dysfunction (ED) in Africa requires a coordinated, culturally informed, and context-specific strategy. The following actions are recommended:

1. Promote Public Awareness and Reduce Stigma: Implement culturally sensitive sexual health education campaigns to reduce stigma, correct misconceptions, and encourage men to seek medical help early for reproductive health concerns. According to many African cultural norms, men may delay seeking care due to perceptions of weakness or embarrassment; this must be addressed through targeted messaging.
2. Encourage Healthy Lifestyle Modifications: Promote a return to traditional African dietary patterns and reduce reliance on Western-style, energy-dense diets. Encourage regular physical activity and fitness as preventive strategies to reduce vascular and metabolic risk factors associated with ED.
3. Discourage Unregulated Use of Aphrodisiacs: Raise awareness about the dangers of unregulated aphrodisiac herbal medications, which are widely used in some

communities. These can cause serious adverse events, including priapism, which may lead to irreversible tissue damage or loss of penile function.

4. Tailor Treatment to Comorbidities and Clinical Profiles: Management should be individualised, particularly for patients with comorbidities such as diabetes mellitus or hypertension. Not all patients are suitable candidates for phosphodiesterase type 5 inhibitors (PDE5-Is), and clinical decision-making should reflect patient-specific needs, risks, and contraindications.
5. Psychological Support for Younger Men: For men aged 25–35, psychological and psychosexual support should be prioritised, especially given the increasing prevalence of psychogenic ED in this age group. In many of these cases, PDE5-Is may be ineffective or unnecessary without addressing underlying emotional or relational stressors.
6. Train Healthcare Providers in Sexual Health: Strengthen clinical capacity by integrating sexual health assessment, counselling, and management into primary and specialist care training. Providers should be equipped to approach these discussions with cultural competence and clinical confidence.
7. Improve Access to Evidence-Based Therapies: Expand the availability of affordable, acceptable, and effective ED treatments—including psychosexual counselling, pharmacological therapies, and second-line interventions—through both public and private healthcare systems.
8. Strengthen Data and Research Capacity: Prioritise nationally representative epidemiological studies and integrate sexual health indicators into routine health surveys. Particular attention should be given to underrepresented regions such as Central and Eastern Africa. Support regional research networks to foster collaboration and innovation in men's sexual health.

REFERENCES

1. Brotto L, Atallah S, Johnson-Agbakwu C, et al. Psychological and interpersonal dimensions of sexual function and dysfunction. *J Sex Med* 2016;13:538-71. https://doi.org/10.1016/j.jsxm.2016.01.019
2. Segraves RT. Considerations for diagnostic criteria for erectile dysfunction in DSM V. *J Sex Med* 2010;7:654-60. https://doi.org/10.1111/j.1743-6109.2009.01684.x
3. Helena C, Abdo N. The impact of ejaculatory dysfunction upon the sufferer and his partner. *Transl Androl Urol* 2016;5:460-9. https://doi.org/10.21037/tau.2016.05.08
4. Rajkumar RP, Kumaran AK. Depression and anxiety in men with sexual dysfunction: A retrospective study. *Compr Psychiatry* 2015;60:114-8. https://doi.org/10.1016/j.comppsych.2015.03.001
5. Liu Q, Zhang Y, Wang J, et al. Erectile dysfunction and depression: A systematic review and meta-analysis. *J Sex Med* 2018;15:1073-82. https://doi.org/10.1016/j.jsxm.2018.05.016
6. Irekpita E, Awe O, Salami T, et al. Clinical, cultural, and psychosocial impediments to self reporting of erectile dysfunction by men in Edo state, Nigeria. *African J Urol* 2017;160-5. https://doi.org/10.1016/j.afju.2016.09.006
7. Ojewole JA. African traditional medicines for erectile dysfunction: Elusive dream or imminent reality? *Cardiovasc J Afr* 2007;18:213-5.
8. Idung AU, Abasiubong F, Ukott IA, et al. Prevalence and risk factors of erectile dysfunction in Niger delta region, Nigeria. *Afr Health Sci* 2012;12:160-5. https://doi.org/10.4314/ahs.v12i2.13
9. Oyelade BO, Jemilohun AC, Aderibigbe SA. Prevalence of erectile dysfunction and possible risk factors among men of South-Western Nigeria: A population-based study. *Pan Afr Med J* 2016;24:124.
10. Mutagaywa RK, Lutale J, Aboud M, et al. Prevalence of erectile dysfunction and associated factors among diabetic men attending diabetic clinic at Muhimbili National Hospital in Dar-es-Salaam, Tanzania. *Pan Afr Med J* 2014;17:227. https://doi.org/10.11604/pamj.2014.17.227.2695
11. Safarinejad MR. Prevalence and risk factors for erectile dysfunction in a population-based study in Iran. *Int J Impot Res* 2003;15:246-52. https://doi.org/10.1038/sj.ijir.3901024
12. Seyam RM, Albakry A, Ghobish A, et al. Prevalence of erectile dysfunction and its correlates in Egypt: a community-based study. *Int J Impot Res* 2003;15:237-45. https://doi.org/10.1038/sj.ijir.3901000
13. Agaba PA, Ocheke AN, Akanbi MO, et al. Sexual functioning and health-related quality of life in men. *Niger Med J* 2017;58:96-100. https://doi.org/10.4103/nmj.NMJ_225_16
14. Saigal CS, Wessells H, Pace J, et al. Predictors and prevalence of erectile dysfunction in a racially diverse population. *Arch Intern Med* 2006;166:207-12. https://doi.org/10.1001/archinte.166.2.207
15. Bacon CG, Mittleman MA, Kawachi I, et al. A prospective study of risk factors for erectile dysfunction. *J Urol* 2006;176:217-21. https://doi.org/10.1016/S0022-5347(06)00589-1
16. Grover SA, Lowensteyn I, Kaouache M, et al. The prevalence of erectile dysfunction in the primary care setting: Importance of risk factors for diabetes and vascular disease. *Arch Intern Med* 2006;166:213-9. https://doi.org/10.1001/archinte.166.2.213

17. Gabrielson AT, Sartor RA, Hellstrom WJG. The impact of thyroid disease on sexual dysfunction in men and women. *Sex Med Rev* 2019;7:57-70. https://doi.org/10.1016/j.sxmr.2018.05.002
18. Selvin E, Burnett AL, Platz EA. Prevalence and risk factors for erectile dysfunction in the US. *Am J Med* 2007;120:151-7. https://doi.org/10.1016/j.amjmed.2006.06.010
19. Seftel AD, Sun P, Swindle R. The Prevalence pf hypertension, hyperlipidemia, diabetes mellitus and depression in men with erectile dysfunction. *J Urol* 2004;171:2341-5. https://doi.org/10.1097/01.ju.0000125198.32936.38
20. Amidu N, Owiredu WKBA, Woode E, et al. Prevalence of male sexual dysfunction among Ghanaian populace: Myth or reality? *Int J Impot Res* 2010;22:337-42. https://doi.org/10.1038/ijir.2010.24
21. Abuhay DA, Gela YY, Getu AA. Prevalence of erectile dysfunction and associated factors among hypertensive patients attending governmental health institutions in Gondar City, Northwest Ethiopia: A cross-sectional study. *Int J Hypertens* 2021;2021. https://doi.org/10.1155/2021/1482500
22. Mathabela B, Madiba S, Modjadji P. Exploring barriers to accessing sexual and reproductive health services among adolescents and young people with physical disabilities in South Africa. *Int J Environ Res Public Health* 2024;21. https://doi.org/10.3390/ijerph21020199
23. Shamloul R, Ghanem H. Erectile dysfunction. *Lance*t *(London, England)* 2013;381:153-65.https://doi.org/10.1016/S0140-6736(12)60520-0
24. Nicolosi A, Glasser DB, Moreira ED, et al. Prevalence of erectile dysfunction and associated factors among men without concomitant diseases: A population study. I*nt J Impot Res* 2003;15:253-7. https://doi.org/10.1038/sj.ijir.3901010
25. Yovwin DG, Imarhiagbe FA, Obazee EM, et al. Erectile dysfunction in a sub-Saharan African population: Profile and correlates in a tertiary care hospital. *Sahel Med J* 2015;18:116-20. https://doi.org/10.4103/1118-8561.169286
26. Nyalile KB, Mushi EHP, Moshi E, et al. Prevalence and factors associated with erectile dysfunction among adult men in Moshi municipal, Tanzania: Community-based study. *Basic Clin Androl* 2020;0:1-7. https://doi.org/10.1186/s12610-020-00118-0
27. Correia MC, Ogola EN, Kayima JK, et al. Erectile dysfunction in hypertensive males in Kenya: A tertiary referral hospital experience. *Afr Health Sci* 2022;22:420-7. https://doi.org/10.4314/ahs.v22i2.48
28. Lue TF. Erectile dysfunction. *N Engl J Med* 2000;342:1802-13. https://doi.org/10.1056/NEJM200006153422407
29. Prieto D. Physiological regulation of penile arteries and veins. *Int J Impot Res* 2008;20:17-29. https://doi.org/10.1038/sj.ijir.3901581
30. Gambescia N, Weeks G. Treatment of erectile dysfunction. In: Fisher JE, O'Donohue WT, editors. Practitioner's guide to evidence-based psychotherapy. Boston, MA: Springer US; 2006. p. 284-90. https://doi.org/10.1007/978-0-387-28370-8_28
31. Siddiqui MA, Peng B, Shanmugam N, et al. Erectile dysfunction in young surgically treated patients with lumbar spine disease: A prospective follow-up study. *Spine* 2012;37:797-801. https://doi.org/10.1097/BRS.0b013e318232601c
32. Mulhall JP. Penile rehabilitation following radical prostatectomy. *Curr Opin Urol* 2008;18:613-20. https://doi.org/10.1097/MOU.0b013e3283136462
33. Traish AM, Munarriz R, O'Connell L, et al. Effects of medical or surgical castration on erectile function in an animal model. *J Androl* 2003;24:381-7. https://doi.org/10.1002/j.1939-4640.2003.tb02686.x

34. Corona G, Rastrelli G, Monami M, et al. Hypogonadism as a risk factor for cardiovascular mortality in men: A meta-analytic study. *Eur J Endocrinol* 2011;165:687-701. https://doi.org/10.1530/EJE-11-0447
35. Jackson G. The importance of risk factor reduction in erectile dysfunction. *Curr Urol Rep* 2007;8:463-6. https://doi.org/10.1007/s11934-007-0049-x
36. Virag R, Bouilly P, Frydman D. Is impotence an arterial disorder? A study of arterial risk factors in 440 impotent men. *Lancet* 1985;1:181-4. https://doi.org/10.1016/S0140-6736(85)92023-9
37. Burchardt M, Burchardt T, Baer L, et al. Hypertension is associated with severe erectile dysfunction. *J Urol* 2000;164:1188-91. https://doi.org/10.1016/S0022-5347(05)67138-8
38. Mittawae B, El-Nashaar AR, Fouda A, et al. Incidence of erectile dysfunction in 800 hypertensive patients: a multicenter Egyptian national study. *Urology* 2006;67:575-8. https://doi.org/10.1016/j.urology.2005.09.040
39. Nehra A, Jackson G, Miner M, et al. The Princeton III Consensus recommendations for the management of erectile dysfunction and cardiovascular disease. *Mayo Clin Proc* 2012;87:766-78. https://doi.org/10.1016/j.mayocp.2012.06.015
40. Inman BA, Sauver JLS, Jacobson DJ, et al. A population-based, longitudinal study of erectile dysfunction and future coronary artery disease. *Mayo Clin Proc* 2009;84:108-13. https://doi.org/10.4065/84.2.108
41. Montorsi P, Ravagnani PM, Galli S, et al. The artery size hypothesis: A macrovascular link between erectile dysfunction and coronary artery disease. *Am J Cardiol* 2005;26;96:19-23. https://doi.org/10.1016/j.amjcard.2005.07.006
42. Wespes E, Schulman C. Venous impotence: Pathophysiology, diagnosis and treatment. *J Urol* 1993;149:1238-45. https://doi.org/10.1016/S0022-5347(17)36358-9
43. Aversa A, Rossi F, Francomano D, et al. Early endothelial dysfunction as a marker of vasculogenic erectile dysfunction in young habitual cannabis users. *Int J Impot Res* 2008;20:566-73. https://doi.org/10.1038/ijir.2008.43
44. Baumhäkel M, Schlimmer N, Kratz M, et al. Cardiovascular risk, drugs and erectile function--a systematic analysis. *Int J Clin Pract* 2011;65:289-98. https://doi.org/10.1111/j.1742-1241.2010.02563.x
45. Thomas A, Woodard C, Rovner ES, et al. Urologic complications of nonurologic medications. *Urol Clin North Am* 2003;30:123-31. https://doi.org/10.1016/S0094-0143(02)00111-8
46. Do C, Huyghe E, Lapeyre-Mestre M, et al. Statins and erectile dysfunction: Results of a case/non-case study using the French Pharmacovigilance System Database. *Drug Saf* 2009;32:591-7. https://doi.org/10.2165/00002018-200932070-00005
47. Lewis RW, Fugl-Meyer KS, Corona G, et al. Definitions/epidemiology/risk factors for sexual dysfunction. *J Sex Med* 2010;7:1598-607. https://doi.org/10.1111/j.1743-6109.2010.01778.x
48. Martin-Morales A, Sanchez-Cruz JJ, Saenz de Tejada I, et al. Prevalence and independent risk factors for erectile dysfunction in Spain: Results of the Epidemiologia de la Disfuncion Erectil Masculina Study. *J Urol* 2001;166:565-9. https://doi.org/10.1016/S0022-5347(05)65986-1
49. Gatti A, Mandosi E, Fallarino M, et al. Metabolic syndrome and erectile dysfunction among obese non-diabetic subjects. *J Endocrinol Invest* 2009;32:542-5. https://doi.org/10.1007/BF03346504

50. Ponholzer A, Temml C, Mock K, et al. Prevalence and risk factors for erectile dysfunction in 2869 men using a validated questionnaire. *Eur Urol* 2005;47:80-6. https://doi.org/10.1016/j.eururo.2004.08.017
51. Köseoğlu N, Köseoğlu H, Ceylan E, et al. Erectile dysfunction prevalence and sexual function status in patients with chronic obstructive pulmonary disease. *J Urol* 2005;174:249-52; discussion 252. https://doi.org/10.1097/01.ju.0000163259.33846.74
52. Bellinghieri G, Santoro D, Mallamace A, et al. Sexual dysfunction in chronic renal failure. *J Nephrol* 2008;21 Suppl 1:S113-7.
53. Huyghe E, Kamar N, Wagner F, et al. Erectile dysfunction in end-stage liver disease men. *J Sex Med* 2009;6:1395-401. https://doi.org/10.1111/j.1743-6109.2008.01169.x
54. Attia AAA, Abdel-Hameed AKS, Amer MAEM, et al. Study of the prevalence and patterns of phosphodiesterase type 5 inhibitor use among sexually active Egyptian males: A National Cross-sectional Survey. *Andrologia* 2019;51:e13364. https://doi.org/10.1111/and.13364
55. Sangeda RZ, Kadinde AW, Masatu CF, et al. Utilization trends of phosphodiesterase type-5 inhibitors for erectile dysfunction between 2019 and 2023 in Tanzania. *Cureus* 2024;16:e58419. https://doi.org/10.7759/cureus.58419
56. Gebreyohannes EA, Bhagavathula AS, Gebresillassie, et al. Recreational use of phosphodiesterase 5 inhibitors and its associated factors among undergraduate male students in an Ethiopian University: A cross-sectional study. *Value Heal* 2016;19:A394. https://doi.org/10.1016/j.jval.2016.09.272
57. The process of care model for evaluation and treatment of erectile dysfunction. The Process of Care Consensus Panel. *Int J Impot Res* 1999;11:54-9. https://doi.org/10.1038/sj.ijir.3900411
58. Rosen RC, Riley A, Wagner G, et al. The international index of erectile function (IIEF): A multidimensional scale for assessment of erectile dysfunction. *Urology* 1997;49:822-30. https://doi.org/10.1016/S0090-4295(97)00238-0
59. Rosen RC, Cappelleri JC, Smith MD, et al. Development and evaluation of an abridged, 5-item version of the international index of erectile function (IIEF-5) as a diagnostic tool for erectile dysfunction. *Int J Impot Res* 1999;11:319-26. https://doi.org/10.1038/sj.ijir.3900472
60. Montorsi P, Ravagnani PM, Galli S, et al. Association between erectile dysfunction and coronary artery disease: Matching the right target with the right test in the right patient. *Eur Urol* 2006;50:721-31. https://doi.org/10.1016/j.eururo.2006.07.015
61. Mannino DM, Klevens RM, Flanders WD. Cigarette smoking: An independent risk factor for impotence? *Am J Epidemiol* 1994;140:1003-8. https://doi.org/10.1093/oxfordjournals.aje.a117189
62. Derby CA, Mohr BA, Goldstein I, et al. Modifiable risk factors and erectile dysfunction: Can lifestyle changes modify risk? *Urology* 2000;56:302-6. https://doi.org/10.1016/S0090-4295(00)00614-2
63. Hannan JL, Heaton JPW, Adams MA. Recovery of erectile function in aging hypertensive and normotensive rats using exercise and caloric restriction. *J Sex Med* 2007;4:886-97. https://doi.org/10.1111/j.1743-6109.2007.00517.x
64. Hannan JL, Maio MT, Komolova M, et al. Beneficial impact of exercise and obesity interventions on erectile function and its risk factors. *J Sex Med* 2009;6:254-61. https://doi.org/10.1111/j.1743-6109.2008.01143.x
65. Guay AT, Perez JB, Heatley GJ. Cessation of smoking rapidly decreases erectile dysfunction. *Endocr Pract* 1998;4:23-6. https://doi.org/10.4158/EP.4.1.23

66. Miller NS, Gold MS. The human sexual response and alcohol and drugs. *J Subst Abuse Treat* 1988;5:171-7. https://doi.org/10.1016/0740-5472(88)90006-2
67. Horasanli K, Boylu U, Kendirci M, et al. Do lifestyle changes work for improving erectile dysfunction? *Asian J Androl* 2008;10:28-35. https://doi.org/10.1111/j.1745-7262.2008.00363.x
68. Chew KK, Bremner A, Stuckey B, et al. Alcohol consumption and male erectile dysfunction: an unfounded reputation for risk? *J Sex Med* 2009;6:1386-94. https://doi.org/10.1111/j.1743-6109.2008.01115.x
69. Esposito K, Giugliano F, Di Palo C, et al. Effect of lifestyle changes on erectile dysfunction in obese men: A randomized controlled trial. *JAMA* 2004;291:2978-84. https://doi.org/10.1001/jama.291.24.2978
70. Esposito K, Ciotola M, Giugliano F, et al. Effects of intensive lifestyle changes on erectile dysfunction in men. *J Sex Med* 2009;6:243-50. https://doi.org/10.1111/j.1743-6109.2008.01030.x
71. Esposito K, Giugliano D. Lifestyle/dietary recommendations for erectile dysfunction and female sexual dysfunction. *Urol Clin North Am* 2011;38:293-301. https://doi.org/10.1016/j.ucl.2011.04.006
72. Gupta BP, Murad MH, Clifton MM, et al. The effect of lifestyle modification and cardiovascular risk factor reduction on erectile dysfunction: A systematic review and meta-analysis. *Arch Intern Med* 2011;171:1797-803. https://doi.org/10.1001/archinternmed.2011.440
73. Maio G, Saraeb S, Marchiori A. Physical activity and PDE5 inhibitors in the treatment of erectile dysfunction: Results of a randomized controlled study. J *Sex Med* 2010;7:2201-8. https://doi.org/10.1111/j.1743-6109.2010.01783.x
74. Konstantinos G, Petros P. Phosphodiesterase-5 inhibitors: Future perspectives. *Curr Pharm Des* 2009;15:3540-51. https://doi.org/10.2174/138161209789206953
75. Andersson KE. Mechanisms of penile erection and basis for pharmacological treatment of erectile dysfunction. *Pharmacol Rev* 2011;63:811-59. https://doi.org/10.1124/pr.111.004515
76. Carson CC 3rd. Phosphodiesterase type 5 inhibitors: State of the therapeutic class. *Urol Clin North Am* 2007;34:507-15, vi. https://doi.org/10.1016/j.ucl.2007.08.013
77. Setter SM, Iltz JL, Fincham JE, et al. Phosphodiesterase 5 inhibitors for erectile dysfunction. *Ann Pharmacother* 2005;39:1286-95. https://doi.org/10.1345/aph.1E487
78. Kendirci M, Tanriverdi O, Trost L, et al. Management of sildenafil treatment failures. *Curr Opin Urol* 2006;16:449-59. https://doi.org/10.1097/01.mou.0000250286.60237.a6
79. Ferrini MG, Kovanecz I, Sanchez S, et al. Long-term continuous treatment with sildenafil ameliorates aging-related erectile dysfunction and the underlying corporal fibrosis in the rat. *Biol Reprod* 2007;76:915-23. https://doi.org/10.1095/biolreprod.106.059642
80. McMahon CG. Treatment of erectile dysfunction with chronic dosing of tadalafil. *Eur Urol* 2006;50:215-7. https://doi.org/10.1016/j.eururo.2006.03.018
81. Behr-Roussel D, Gorny D, Mevel K, et al. Chronic sildenafil improves erectile function and endothelium-dependent cavernosal relaxations in rats: Lack of tachyphylaxis. *Eur Urol* 2005;47:87-91. https://doi.org/10.1016/j.eururo.2004.09.005
82. Bella AJ, Deyoung LX, Al-Numi M, et al. Daily administration of phosphodiesterase type 5 inhibitors for urological and nonurological indications. *Eur Urol* 2007;52:990-1005. https://doi.org/10.1016/j.eururo.2007.06.048

83. Gruenwald I, Leiba R, Vardi Y. Effect of sildenafil on middle-aged sexually active males with no erectile complaints: A randomized placebo-controlled double-blind study. *Eur Urol* 2009;55:969-76. https://doi.org/10.1016/j.eururo.2008.04.048
84. Ekmekçioğlu O, Inci M, Demirci D, et al. Effects of sildenafil citrate on ejaculation latency, detumescence time, and refractory period: Placebo-controlled, double-blind, crossover laboratory setting study. *Urology* 2005;65:347-52. https://doi.org/10.1016/j.urology.2004.09.012
85. Eardley I, Donatucci C, Corbin J, et al. Pharmacotherapy for erectile dysfunction. *J Sex Med* 2010;7:524-40. https://doi.org/10.1111/j.1743-6109.2009.01627.x
86. Morganroth J, Ilson BE, Shaddinger BC, et al. Evaluation of vardenafil and sildenafil on cardiac repolarization. *Am J Cardiol* 2004;93:1378-83, A6. https://doi.org/10.1016/j.amjcard.2004.02.034
87. Aoyagi T, Hayakawa K, Miyaji K, et al. Sildenafil induced priapism. *Bull Tokyo Dent Coll* 1999;40:215-7. https://doi.org/10.2209/tdcpublication.40.215
88. Tomsak R. PDE5 inhibitors and permanent visual loss. *Int J Impot Res* 2005;17:547-9. https://doi.org/10.1038/sj.ijir.3901396
89. McGwin GJ. Phosphodiesterase type 5 inhibitor use and hearing impairment. *Arch Otolaryngol Head Neck Surg* 2010;136:488-92. https://doi.org/10.1001/archoto.2010.51
90. Jain P, Rademaker AW, McVary KT. Testosterone supplementation for erectile dysfunction: Results of a meta-analysis. *J Urol* 2000;164:371-5. https://doi.org/10.1016/S0022-5347(05)67363-6
91. Shabsigh R, Kaufman JM, Steidle C, et al. Randomized study of testosterone gel as adjunctive therapy to sildenafil in hypogonadal men with erectile dysfunction who do not respond to sildenafil alone. *J Urol* 2004;172:658-63. https://doi.org/10.1097/01.ju.0000132389.97804.d7
92. Shamloul R, Ghanem H, Fahmy I, et al. Testosterone therapy can enhance erectile function response to sildenafil in patients with PADAM: A pilot study. *J Sex Med* 2005;2:559-64. https://doi.org/10.1111/j.1743-6109.2005.00071.x
93. Hatzimouratidis K, Hatzichristou DG. A comparative review of the options for treatment of erectile dysfunction: Which treatment for which patient? *Drugs* 2005;65:1621-50. https://doi.org/10.2165/00003495-200565120-00003
94. Perimenis P, Konstantinopoulos A, Perimeni PP, et al. Long-term treatment with intracavernosal injections in diabetic men with erectile dysfunction. *Asian J Androl* 2006;8:219-24. https://doi.org/10.1111/j.1745-7262.2006.00095.x
95. Williams G, Abbou CC, Amar ET, et al. Efficacy and safety of transurethral alprostadil therapy in men with erectile dysfunction. MUSE Study Group. *Br J Urol* 1998;81:889-94. https://doi.org/10.1046/j.1464-410x.1998.00703.x
96. Pajovic B, Dimitrovski A, Fatic N, et al. Vacuum erection device in treatment of organic erectile dysfunction and penile vascular differences between patients with DM type I and DM type II. *Aging Male* 2017;20:49-53. https://doi.org/10.1080/13685538.2016.1230601
97. Bettocchi C, Palumbo F, Spilotros M, et al. Patient and partner satisfaction after AMS inflatable penile prosthesis implant. *J Sex Med* 2010;7:304-9. https://doi.org/10.1111/j.1743-6109.2009.01499.x
98. Selph JP, Carson CC 3rd. Penile prosthesis infection: Approaches to prevention and treatment. *Urol Clin North Am* 2011;38:227-35. https://doi.org/10.1016/j.ucl.2011.02.007

FIGURES AND TABLES

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.

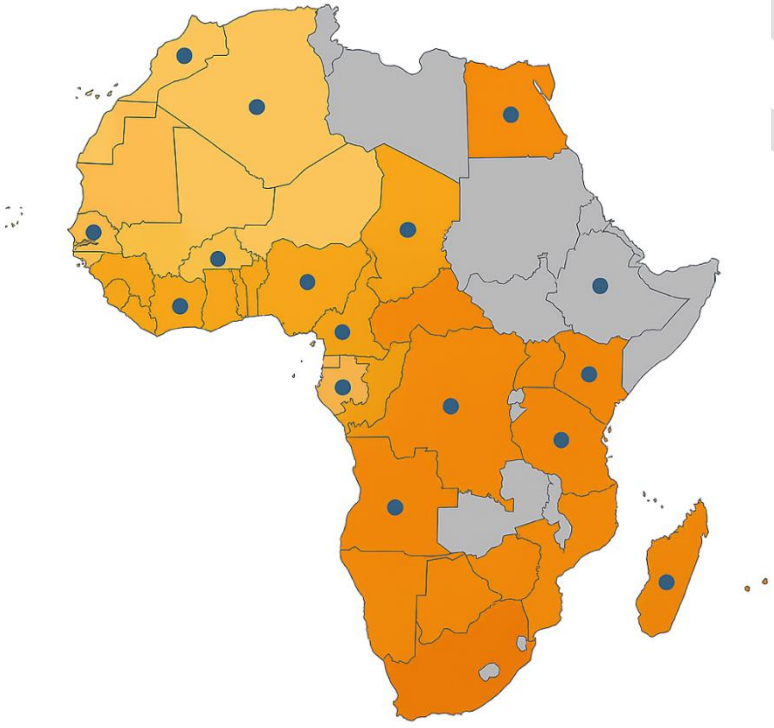

Figure 2. Diagram illustrating the role of nitric oxide (NO) in penile smooth muscle relaxation. NO activates the GTP–cGMP pathway, lowering calcium and causing smooth muscle relaxation. PDE5 breaks down cGMP to terminate the erection. A secondary pathway via cAMP also reduces calcium. Erection-enhancing 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.

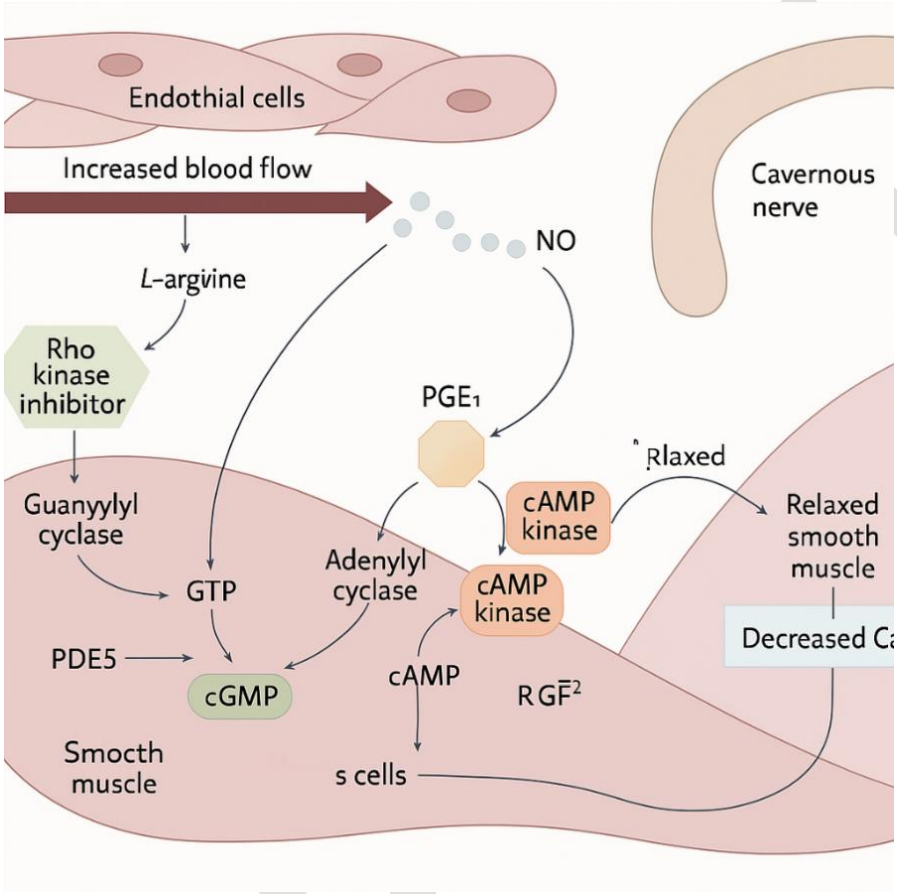

Figure 3. Flowchart outlining the clinical approach to diagnosing erectile dysfunction (ED). The algorithm begins with patient history and symptom assessment, followed by physical examination and basic laboratory tests. Depending on findings, further steps include evaluating psychological factors, assessing 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.

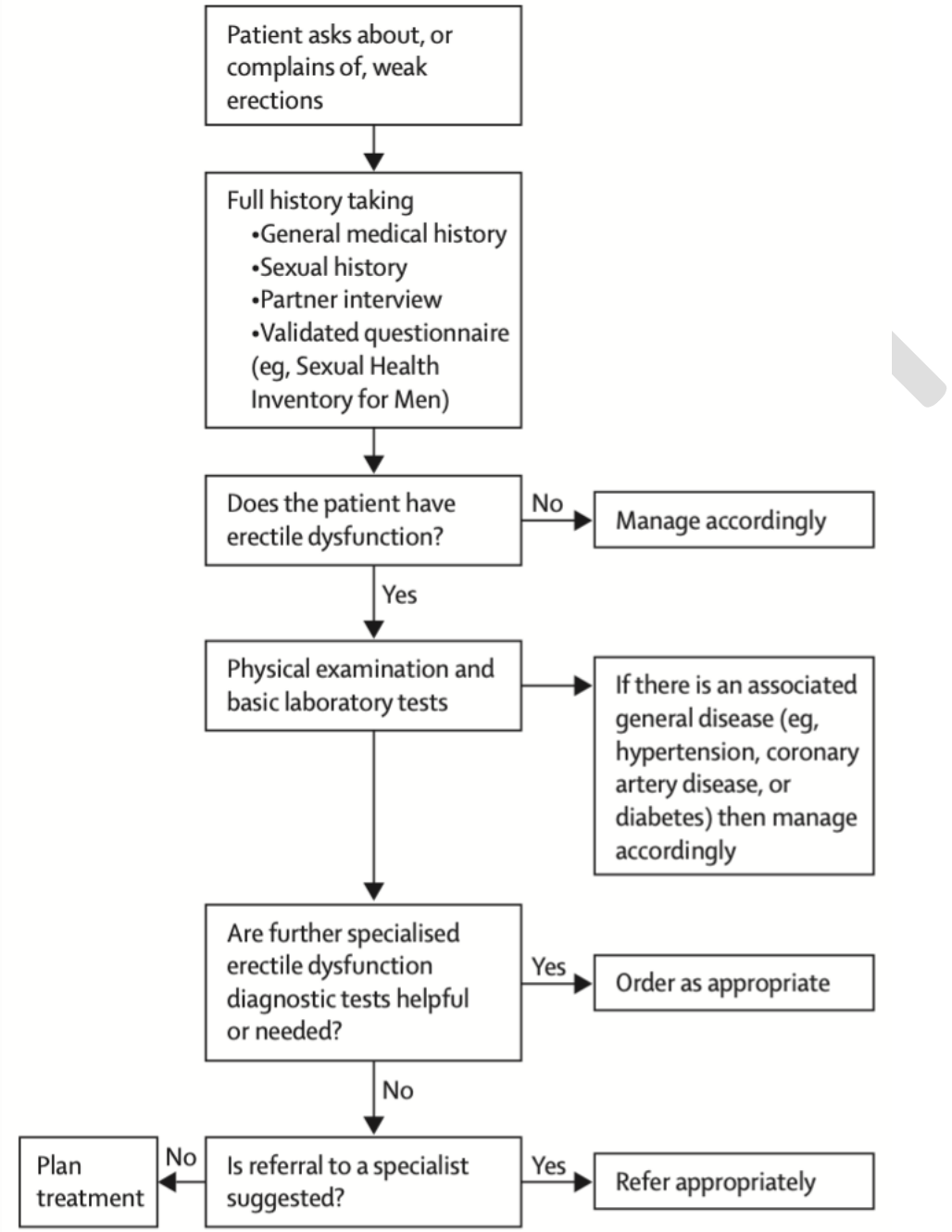

Figure 4. Diagram showing the integrative treatment options for erectile dysfunction. Central pharmacologic therapy (e.g., PDE5 inhibitors) is supported by adjunct strategies: lifestyle modification (e.g., diet, exercise, smoking cessation), testosterone supplementation for men with low hormone levels, and psychosexual therapy to address psychological or relational issues. The image conveys that ED treatment is most effective when approached holistically.

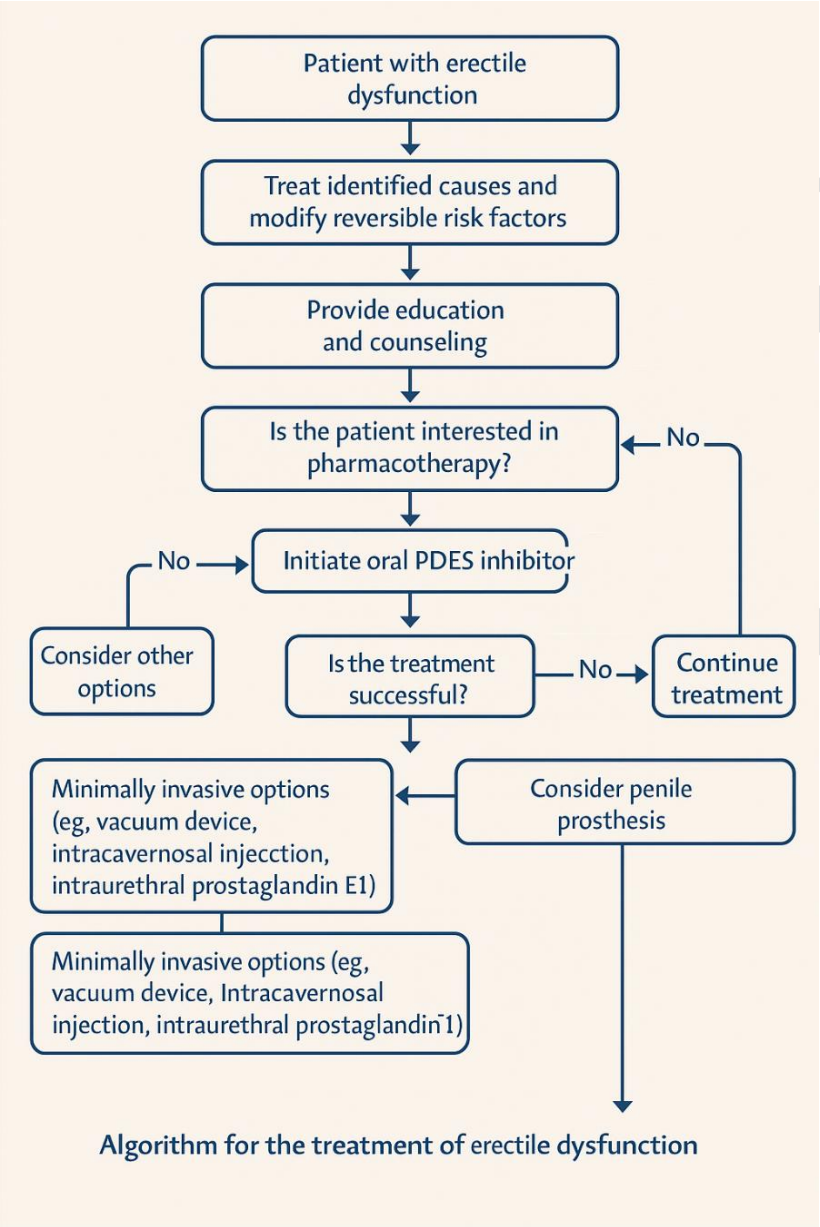

Table 1. Reported prevalence of erectile dysfunction in African countries				
Region	**Country**	**Study population**	**Prevalence (%)**	**Study source**
West Africa	Ghana	General male population	66%	Amidu et al, 2010 (18)
	Nigeria	Community-based men	44–59%	Idung et al, 2012; Oyelade et al, 2016 (6,7)
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

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
Endocrinological	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

Table 4. Summary of drug classes associated with erectile dysfunction			
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

Table 5. Common diagnostic tests for erectile dysfunction 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.

Table 6. Characteristics of commercially available phosphodiesterase type 5 (PDE5) 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-arteritic 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		