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Advances in the Causality, Diagnosis and Management of Erectile Dysfunction

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Abstract. Penile erection demonstrates how psychological influences and hormonal balance modulate microcirculation. It results from a highly coordinated neurovascular process involving the integration and synchronisation of vascular endothelium, smooth muscle, and psychological, neurological, and endocrine systems. Therefore, the proper functioning of these processes is essential for maintaining penile flaccidity or achieving an erection, whereas disturbances in any component can result in erectile dysfunction (ED). ED is a common health condition with a substantial impact on men's quality of life worldwide. Currently, this disease is considered a multifactorial condition involving a complex interplay of social, psychological, and physiological factors, necessitating the adoption of multiple treatment strategies. This paper offers an overview of the mechanisms of erections, etiological factors contributing to ED—including psychological, neurological, endocrinological, and vasculogenic aspects—diagnostic approaches and treatments.

Keywords: Erectile dysfunction, Etiology, Diagnosis, Treatment, Sexual health

1. Introduction

Erectile dysfunction (ED), previously termed impotence, refers to the persistent inability to attain or sustain an erection adequate for satisfactory sexual activity [1]. Historical records, including Egyptian papyri from around 2000 BC, provide the earliest descriptions of ED. This condition can profoundly affect a man's self-confidence, overall quality of life, and capacity to sustain close relationships. It is estimated that around 40% of men in their 40s and up to 70% of those in their 70s experience some degree of ED, with millions affected globally [2]. There are considerable limitations when conducting epidemiological studies. All such research must address the fact that this is a sexual response dysfunction (rather than a disease), so people do not necessarily look for help. Consequently, prevalence rates are likely underestimated, as cultural taboos often prevent men from openly discussing sexual difficulties. Given the age-related increase in ED incidence, projections suggest that by 2025, approximately 322 million men globally will be affected.

2. Physiology of erections

A normal penile erection is a neurovascular event influenced by psychological factors and regulated through the coordinated actions of the endocrine, vascular, and nervous systems. Sexual arousal

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activates parasympathetic nerve pathways, leading to the release of acetylcholine. Within the endothelial cells of penile arteries, nitric oxide synthase (NOS) converts L-arginine into nitric oxide (NO) and L-citrulline through an oxidation reaction. NO stimulates guanylyl cyclase within the corpus cavernosum, elevating levels of cyclic guanosine monophosphate (cGMP). This promotes relaxation of vascular smooth muscle, vasodilation, and enhanced penile blood flow. Rapid filling and expansion of the sinusoidal venous system leads to obstructive occlusion of the venous plexus and white membrane, resulting in almost complete obstruction of venous return. Contraction of the ischiocavernosus muscles compresses the blood-filled corpora cavernosa, while perineal muscle activity can further elevate pressure to several hundred mmHg. After ejaculation, sympathetic activation halts neurotransmitter release, and phosphodiesterase enzymes degrade cGMP, initiating detumescence and smooth muscle relaxation.

3. Cause of ED

Erectile dysfunction can be classified as psychological, organic (i.e., neurological, endocrine, arterial, cavernosal, or drug-induced), or mixed psychological and organic, while the vast majority of erectile dysfunction cases typically exhibit mixed psychological and organic characteristics.

3.1. Psychological factors

An important psychological factor associated with ED is performance anxiety (fear of failure during sexual intercourse). Men are more vulnerable to performance anxiety because men's sexual function can be observed directly and by both the man and his partner. Performance anxiety could be the only factor causing ED, while it may accompany cases of ED even if the cause is primarily medical. In addition to performance anxiety, several other psychosocial factors may contribute to ED. Mental health conditions associated with ED include depression, general anxiety disorder, obsessive-compulsive disorder, and paraphilic disorder. General stress in men's lives, whether related to financial risks, relationship conflicts, or family issues, could also lead to general problems and ED. Lack of proper sex education may also contribute to ED, while men fail to cope with their sexual response.

3.2. Neurogenic factors

Various neurological disorders—such as multiple sclerosis, temporal lobe epilepsy, Parkinson's disease, stroke, Alzheimer's disease, and spinal cord injury—are linked to the onset and progression of ED. Men post-stroke often experience reduced libido, erectile capacity, and ejaculatory function, with reported ED prevalence ranging from 17% to 48% [3]. Following spinal cord injury, ED occurs in as many as 80% of affected men, primarily due to disruption of neural pathways essential for erectile function [4]. The severity of ED varies according to the level of spinal cord injury (LOI), the extent of tissue damage, and the time elapsed since the injury.

3.3. Endocrinological erectile dysfunction

Testosterone regulates erectile physiology through multiple regulatory pathways. It supports the structural integrity and functional capacity of nerves, especially the cavernous nerves. The nitric oxide synthase/cyclic guanosine monophosphate (NOS/cGMP) pathway is central to erectile function, and evidence indicates that testosterone modulates this system by influencing the expression and activity of NOS isoforms within the corpus cavernosum [5]. Furthermore, animal

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studies demonstrate that castration reduces PDE5 expression and activity in rabbits and rats. In contrast, androgen replacement increases PDE5 activity and expression [6]. These findings suggest that androgens are essential for both NOS regulation and PDE5 modulation.

Thyroid hormone abnormalities have been found to disrupt the male reproductive axis, leading to several sexual dysfunctions. Research demonstrates that erectile dysfunction, premature ejaculation, and delayed ejaculation are common manifestations in men experiencing thyroid hypofunction and hyperfunction. Importantly, these symptoms often resolve promptly upon the restoration of euthyroidism, emphasising the need for effective management of thyroid hormone disorders to maintain optimal reproductive health [7].

Hyperprolactinemia is an uncommon endocrine disorder that can lead to androgen deficiency and ED [5]. Prolactin (PRL) is a pituitary-derived peptide hormone secreted primarily from the anterior lobe. PRL contributes to regulating male sexual drive and modulates testosterone utilisation within target tissues. When excessively elevated, PRL alters the balance of neurotransmitters involved in sexual response, particularly reducing dopaminergic tone relative to serotonergic inhibition, which diminishes libido and erectile capacity. At the hypothalamic–pituitary level, hyperprolactinemia suppresses GnRH and gonadotropin release, inhibiting androgen metabolism, and disrupts central dopaminergic signalling [8].

3.4. Vasculogenic erectile dysfunction

Penile erection depends on arterial dilation and enhanced blood inflow; thus, any disturbance of vascular integrity can impair erectile function. Unsurprisingly, vasculogenic ED represents the majority of organic cases and typically results from defects in the penile arterial supply or venous drainage [9].

Penile arteries, owing to their smaller diameter, are particularly vulnerable to atherosclerotic obstruction. In men over 50, nearly half of ED cases are attributed to vascular disease. Common cardiovascular risk factors—such as diabetes, smoking, dyslipidemia, and hypertension—aggravate endothelial injury and are observed more frequently in men with ED than in the general population [10]. The coexistence of multiple risk factors further worsens penile hemodynamics, as reflected in reduced penile blood pressure indices.

Vasculogenic ED is closely associated with both existing cardiovascular risk factors and the subsequent development of major CVD outcomes, including myocardial infarction, stroke, and cardiovascular mortality [11]. Increasing evidence indicates that ED may serve as an early clinical marker of cardiovascular events, providing an opportunity for timely prevention and intervention [12]. Because of its simplicity and low cost, assessing ED has been proposed as a practical prognostic tool, potentially outperforming some conventional risk factors, particularly in men at intermediate cardiovascular risk. Reflecting this evidence, recent CVD prevention guidelines now recognise ED as part of cardiovascular risk assessment and highlight the need for greater awareness in clinical practice [13].

3.5. Medication-induced erectile dysfunction

Frequently prescribed medications—such as antihypertensives, antidepressants, proton pump inhibitors and muscle relaxants—are considered to be associated with the onset of ED. First-generation antihypertensive drugs are linked to adverse effects on erectile function. In contrast, more modern drugs such as CCB and ACEI appear largely neutral. For example, non-selective beta-blockers carry a high potential to cause ED because alpha-adrenergic activity induces

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vasoconstriction in the corpora cavernosa [14]. Aldosterone inhibitors, like spironolactone, bind to mineralocorticoid receptors throughout the body and can cause gynecomastia, mastodynia and ED [15].

Among psychotropic medications, antidepressants—particularly selective serotonin reuptake inhibitors (SSRIs) and venlafaxine—are a common cause of medication-induced ED. Mechanistically, serotonergic signalling disrupts dopaminergic pathways through serotonin receptors in the mesolimbic region, an area central to sexual desire and orgasm [16]. Additional mechanisms of antidepressant-induced sexual dysfunction include reduced nitric oxide synthase activity and the anticholinergic effects of certain agents. Mirtazapine, bupropion and agomelatine are generally less likely to cause ED than SSRIs, while in some cases they have even been reported to improve sexual performance. Consequently, these drugs are sometimes considered for use as augmentation ("antidote") or substitution strategies in patients who develop sexual dysfunction during antidepressant therapy [17].

4. Diagnosis of erectile dysfunction

The standard diagnostic approach for erectile dysfunction comprises three principal steps: (1) medical history taking and physical examination, (2) laboratory investigations, and (3) imaging studies and relevant ancillary tests.

The initial evaluation of ED begins with a detailed history and physical examination. Men with ED should have an extensive medical history given the potential causes for ED. It is critical to assess the potential role of medical conditions in a patient's ED and to differentiate the history of organic from psychogenic causes of ED [18]. A detailed sexual history should also be taken to determine the severity, onset, and duration of ED. The presence and severity of ED should be determined by careful memory investigations (onset of erectile dysfunction, time of onset, association with a specific partner, couple conflicts, erection/stiffness, absence of erection with ejaculation, spontaneous nocturnal erections) and specific questionnaires to evaluate.

Physical examination should involve inspection of the genitalia for lesions, scarring, plaques, and evaluation of the meatal location. Testicular examination is required to assess both size and consistency. Further assessment should cover secondary sexual characteristics, peripheral vascular status and sensation, abdominal masses, and possible gynecomastia.

Standard laboratory evaluation for ED usually includes fasting glucose, lipid profile, cholesterol levels, and hormonal assays. Similar to physical findings, laboratory studies aim to confirm specific etiologies (such as hypogonadism) and to evaluate potential comorbidities or concurrent conditions (e.g., diabetes, dyslipidemia) [19].

Specific diagnostic modalities for assessing penile endothelial dysfunction include intracavernosal injection, Doppler ultrasound, pharmacological arteriography, cavernosography, neurological evaluation, and nocturnal penile tumescence testing. These tests help distinguish organic ED from psychogenic causes and guide surgical planning for patients with arterial insufficiency or veno-occlusive dysfunction. Among all the tests, nocturnal penile tumescence testing is regarded as the closest one to a gold standard in identifying between psychogenic and organic erectile dysfunction.

5. Treatments for erectile dysfunction

Management of ED often begins with lifestyle change, including weight loss, quitting alcohol and cigarettes [20]. Current therapies to treat ED mainly include oral treatment, intracavernosal

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injection, vacuum erection device, penile prosthesis, low-intensity extracorporeal shock wave (Li-ESW), and stem cell injection therapy [21].

5.1. Oral treatment

Phosphodiesterase 5 inhibitors (PDE5I) are the most common treatment for ED. First studied as a potential treatment for heart diseases, Sildenafil has occasionally been found to have an erection-promoting effect during clinical trials. Consequently, Sildenafil was approved by the FDA to be the first oral treatment for ED.

Phosphodiesterase 5 (PDE5) is highly expressed in vascular smooth muscle and is the most common PDE isoform in penile smooth muscle. Penile erection primarily relies upon a nitric oxide(NO)-cyclic guanosine monophosphate(cGMP) signalling cascade, wherein PDE5 functions to degrade cGMP [22]. NO released from neurons and endothelial cells diffuses into the corpus cavernosum, elevating cGMP levels. This triggers smooth muscle relaxation, penile vasodilation, and vascular filling, ultimately resulting in an erection [23]. Inhibition of PDE5 prolongs cGMP activity, lowering intracellular calcium and sustaining smooth muscle relaxation, thereby facilitating rigid erections. Thus, PDE5Is improve erectile response by amplifying NO-mediated cGMP signalling, making them an effective therapy for ED.

At present, four PDE5Is—sildenafil, tadalafil, vardenafil, and avanafil—have FDA approval. Other drugs such as udenafil, lodenafil, and mironafil remain unapproved in the United States but are allowed for sale in certain other regions. Despite pharmacokinetic differences, these PDE5Is share comparable efficacy, tolerability, and safety profiles. Choice of PDE5I depends on patient and physician preference, guided by factors such as cost, tolerability, onset of action, and duration of effect.

5.2. Intracavernosal injection

Intracavernosal injection of vasodilators, such as alprostadil, is an alternative option for ED, especially for men who can not achieve a satisfying outcome from oral drugs. In the 1980s, intracavernous injections of papaverine and phentolamine were observed to have pro-erectile activity. These two substances are then replaced by alprostadil (PGE1). 72.6 per cent of men with ED achieved a firm erection after intracavernosal injection of alprostadil [24]. However, compared with PDE5Is, side effects, including priapism, ecchymosis, hematoma, and penile fibrosis, caused a decrease in the clinical application of intracavernosal injection [25]. At present, intracavernosal injections are primarily used in conjunction with Doppler ultrasound for diagnostic purposes and evaluation of penile hemodynamics [26].

The more recent drug for intracavernosal injections is botulinum toxin-A (BTX). BTX inhibits acetylcholine and norepinephrine release from sympathetic pathways and upregulates vascular endothelial growth factor (VEGF) and CD31 expression [27], promoting vasodilation and endothelial proliferation, thereby influencing ED pathophysiology. A study reported that intracavernosal injection of BTX either alone or combined with PDE5Is successfully induced erections in ED patients unresponsive to other treatments and initially considered for penile prosthesis implantation [28].

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5.3. Vacuum erection device

A vacuum erection device (VED) is a machine that induces penile engorgement by generating negative pressure, thereby enhancing blood inflow into the corpora cavernosa. A constriction ring at the penile base is used to sustain the erection. VED is a major treatment for patients with organic ED, while it has a high success rate and few side effects [28]. However, the erections caused by VED were often described as artificial and associated with penile coldness, leading to dissatisfaction in nearly half of users. VEDs are generally reserved for patients who are in a stable relationship, not sensitive to PDE5I, and who refuse other more invasive options such as intracavernous injections or penile prosthesis implantation.

5.4. Penile prosthesis implantation

Penile prosthesis implantation (PPI) is regarded as a third-line treatment for ED. The modern three-piece inflatable implants can mimic natural erection, allowing activation for erection and deactivation for flaccidity when not required. It is reported that PPI achieve among the highest satisfaction rates (85–90%) of all implantable medical devices, with low revision rates; more than 90% of recipients acquire regular sexual activity with partners [29,30]. Several studies demonstrated that PPI is particularly suitable for patients with ED due to Peyronie's disease or radical prostatectomy [31]. Despite these advantages, PPI remains costly and invasive—irreversibly altering cavernosal tissue during surgery—and carries rare but serious risks such as infection, pump migration, or unintended inflation, which may necessitate reoperation or further interventions. Thus, PPI is often the last choice when oral and injection treatment are ineffective.

5.5. Low-intensity extracorporeal shock wave therapy

Low-intensity extracorporeal shock wave therapy (LI-ESWT) delivers low-intensity acoustic waves (<0.2 mJ/mm²) to targeted tissues in a manner conceptually similar to lithotripsy used for urinary stone management. It can enhance expression of eNOS, VEGF, and other angiogenic factors in the corpora cavernosa, promoting vasodilation, neovascularisation, improved penile blood flow, and better erectile function. In a 2010 trial, 20 middle-aged men with vasculogenic ED treated with LI-ESWT showed marked improvements in erection duration, penile rigidity, and endothelial function. At six months of follow-up, ten participants no longer required PDE5I therapy. The procedure was well tolerated, with no pain or adverse effects reported during follow-up [32]. However, other studies reported that only a very limited impact of LI-ESWT was observed on patients with ED, while its efficacy depends on the degree of severity of the ED [33,34]. Because of inconsistent results and the absence of standardised treatment protocols for intensity, further clinical trials and methodological standardisation are needed before routine use [31].

6. Conclusion

ED remains a prevalent health concern with multifactorial origins, necessitating a comprehensive approach to diagnosis and treatment. While PDE5 inhibitors continue to serve as the cornerstone of ED therapy due to their efficacy and safety profile, there is a growing interest in alternative and emerging treatments that offer personalised solutions for patients. Advances in medical technology and telehealth present promising avenues for improving accessibility to care and facilitating early intervention. Furthermore, the association between ED and cardiovascular disease underscores the importance of integrated care and underscores the need for holistic approaches that address not only

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the symptoms of ED but also its underlying causes and associated comorbidities. Continued research, innovation, and awareness efforts are crucial in advancing ED care and enhancing the overall well-being and quality of life for affected individuals.

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